Textbook of
Forensic Medicine and Toxicology

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Textbook of
Forensic Medicine and Toxicology
Principles and Practice

Fifth Edition

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Table 3.12 (Continued)

Features	Male	Female
	SCAPULA	
Height	More than 157 mm	Less than 114 mm
Glenoid cavity	Height greater (about 3.92 cm)	Height lesser (about 3.26 cm)
	VERTEBRAL COLUMN	
General appearance	General architecture and impressions for muscular markings are prominent	Less prominent
Total length of spinal column	Approximately 70 cm	Approximately 60 cm
Breadth of atlas vertebra	7.4–9.9 cm	6.5–7.6 cm

Table 3.13 Accuracy of Determining Sex from Various Bones as Reported by Krogman

Skeletal remains	Percentage accuracy
Entire skeleton	100
Skull + pelvis	98
Pelvis alone	95
Skull alone	92
Long bones	80
Long bones + pelvis	98

STATURE

The third primary character for the identification of a person is the stature as outlined earlier. Stature or height of a person increases progressively and becomes maximum at the age between 21 and 25 years. Later, for every 25 years, it is shortened by 2.5 cm due to thinning of intervertebral discs and some stooping posture as a result of decreased tone of muscles. Further, the height varies in the various hours of the day, being maximum in the morning and less by 1.5 to 2.00 cm in the evening due to reduction of elasticity of intervertebral discs. In a dead body, soon after death due to primary relaxation of the muscles, the body length may be more by 2–2.5 cm. Later, when rigor mortis develops, it may be shortened. With the passage of rigor mortis and onset of putrefaction, the length may change due to secondary relaxation.

Stature from a Dismembered Body Part

- When both side arms are outstretched in a straight line, the distance between the tips of the two middle fingers of the hands is approximately equal to the stature of the person.
- Stature is approximately equal to twice the length from vertex to symphysis pubis or equal to twice the length from symphysis pubis to one side heel, with the hip and knee extended and ankle dorsiflexed.
- Stature is about 3.3 times the length from the sternal notch to the symphysis pubis.
- Stature is about 3.7 times the distance between the tip of olecranon and tip of middle finger of the same side.

Stature from Bones

Stature is 2.5–4 cm more than the length of the whole skeleton (total thickness of the soft tissues in between the bones at different joints from heel to vertex is about 2.5–4 cm). When the whole skeleton is not available but one or the other long bones are available, anyone of the following formulae can be used to get the stature of a person (due consideration may be given to the racial or geographical origin, sex and condition of the bone).

- **Karl Pearson's formula (1899):** This formula was in use worldwide for a long period. It gives different calculating factors for bones from males and females of European subjects, depending on whether the bones are wet with intact cartilages or are dried and devoid of cartilage. For each long bone, there is a separate multiplying factor. A constant factor (different for each bone) is to be added to the product of the length of the bone with the multiplying factor 2.5–4 cm is to be added for the soft parts (Table 3.14).
- **Trotter and Glesser's formulae (1952, 1958):** They succeeded in finding out more dependable formulae for determination of stature for males and females of White and Negro origins. They considered the lengths of the long bones separately and in combinations. The subjects of their study were between 28 and 30 years of age. Therefore, due consideration should be given while using their formulae for elderly subjects in whom there occurs some decrease in the stature.

  The formulae discovered by Trotter and Glesser were suitable for the people on whom and for whom these were worked out. As such these cannot be satisfactorily used for Indian subjects. Further, people from different regions of India bear different morphological features depending on

Table 3.14 Karl Pearson Formula for Adult Long Bones in Dry State for Europeans used in Estimating the Stature in Centimetres (Constant × Length of the bone)

Bone	Male	Female
Femur	81.306 + 1.880 × Length of femur	72.884 + 1.945 × Length of femur
Tibia	78.664 + 2.376 × Length of tibia	74.774 + 2.352 × Length of tibia
Humerus	70.641 + 2.891 × Length of humerus	71.475 + 2.754 × Length of humerus
Radius	89.925 + 2.271 × Length of radius	81.224 + 3.343 × Length of radius

their geographical distribution and primary racial characters. For this reason, a single formula cannot suit all parts of the country. Different formulae available for different parts of India are given in Table 3.15.

In taking measurements of the bones, their maximum lengths are to be considered. Use of **Hepburn type osteometric board gives most accurate measurements**. To get the maximum length of the bone, it is placed lengthwise in between the two vertical planes of the board. The maximum length of the bone is the distance between these two planes. For femur, overall maximum length from the head to the medial condyle is measured. For tibia, the maximum length from the lateral condyle to the tip of the medial malleolus is measured. For fibula, the length between tip of the head and tip of the lateral malleolus is measured. For radius, the greatest length from the medial margin of the head to the tip of the styloid process and for ulna, the length from the top of the head to the tip of the styloid process is measured. Wet or humid bones are slightly longer than the dry ones. While measuring humid bones, some authors have recommended deduction of 7 mm in case of femur, 5 mm in case of tibia, 5 mm in case of humerus and 3 mm in case of radius to bring the lengths of the bones at par with the lengths at their dried state. To maintain a uniform standard, some authors recommend use of right side bones only. However, the right side bones may not always be available. Therefore, to reach some authentic results, one should examine as many of these long bones as available and the average of all should be recorded as the estimated stature of the individual.

SECONDARY CHARACTERISTICS IN IDENTIFICATION

A number of secondary characteristics can be considered while identifying a person. These are as follows:

Facial Appearance

During life, the general expression of face can readily be altered and, therefore, mistakes can easily be made. After death, such alteration is, of course, impossible; but death so speedily alters facial appearance that too much reliance need not be made upon this mode of identification. Nearly every person might have seen another who is more or less like him, and occasionally this resemblance is so startling that it may become impossible to recognise the difference between the two people when seen apart. A remarkable example reported in literature is of two men, quite unrelated to each other, were in the same prison at the same time, bore the same name and had practically the same Bertillon measurements. All this serves to observe caution by sight recognition in criminal matters.

Table 3.15 Multiplication Factors for Different Bones for Calculation of Stature of Persons of Different Parts of India, as Gathered from the Literature

	For Bengal, Bihar and Orissa		For UP	For Punjab
	Male	Female	Male	Male
Femur	3.82	3.8	3.7	3.6
Tibia	4.49	4.46	4.48	4.2
Fibula	4.46	4.43	4.48	4.4
Humerus	5.31	5.31	5.3	5.0
Radius	6.78	6.7	6.9	6.3
Ulna	6.0	6.0	6.3	6.0

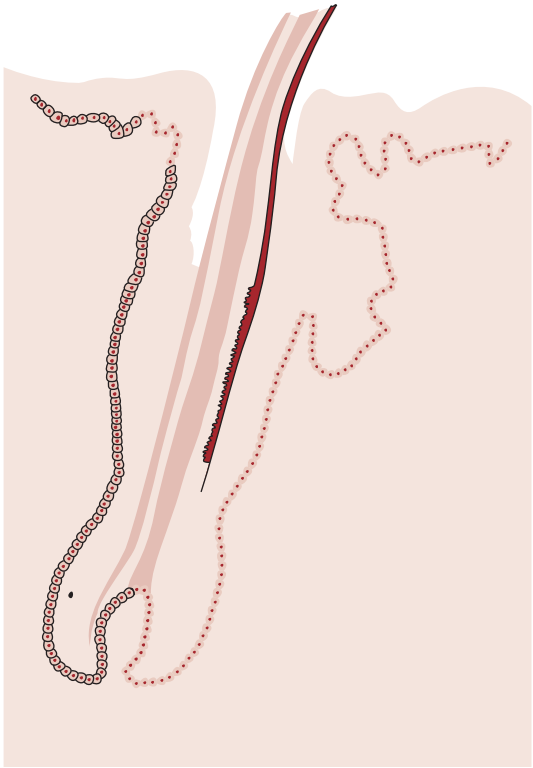
Personal Effects

It includes clothing, pocket contents, laundry marks, peculiar stitching/repair and jewellery, etc.

Clothing may be distinctive, and a detailed description may be obtained from the relative or someone who had last seen the deceased. Moreover, there may be clues in the clothing itself such as laundry mark, maker's tag or dyer's mark(s), etc. The clothes should better be examined under ultraviolet light so as to appreciate apparently invisible ink-marks.

Clothing may also provide a clue to the social status and occupation of the individual. Bullet holes/defects (with or without deposits of soot, powder grains, etc.), cuts or tire-impressions may yield information regarding the cause and manner of death. Disarranged clothing, missing button(s), stains due to poison, vomit, faecal matter, blood, semen, saliva, etc. suggest nature of assault that can be helpful in further identification.

Pocket contents like papers, letters, keys, license/identity card, diary, passport, etc. often provide the initial evidence that usually leads to positive identification. In a series of exhumations of allied war crime victims following the Second World War, over one-third of the bodies were identified by laundry marks and personal effects; although in several cases efforts had been made to conceal the identity of the deceased.



However, caution may be exercised in accepting laundry marks as evidence of identity on small articles like handkerchiefs, etc., as these are easily transferred or misplaced.

Cigarette packages bear code symbols that indicate the date and place of manufacture. Watches frequently have private marks inside the cases made by the watch makers who have carried out repairs.

However, a person might have been clothed in borrowed garments or second-hand clothes and rarely, a body may be deliberately clothed in another's attire and documents/articles, etc. placed in the pockets in order to mislead the investigative agencies. All the pocket contents must be carefully recorded, preserved and handed over to the police in a sealed parcel.

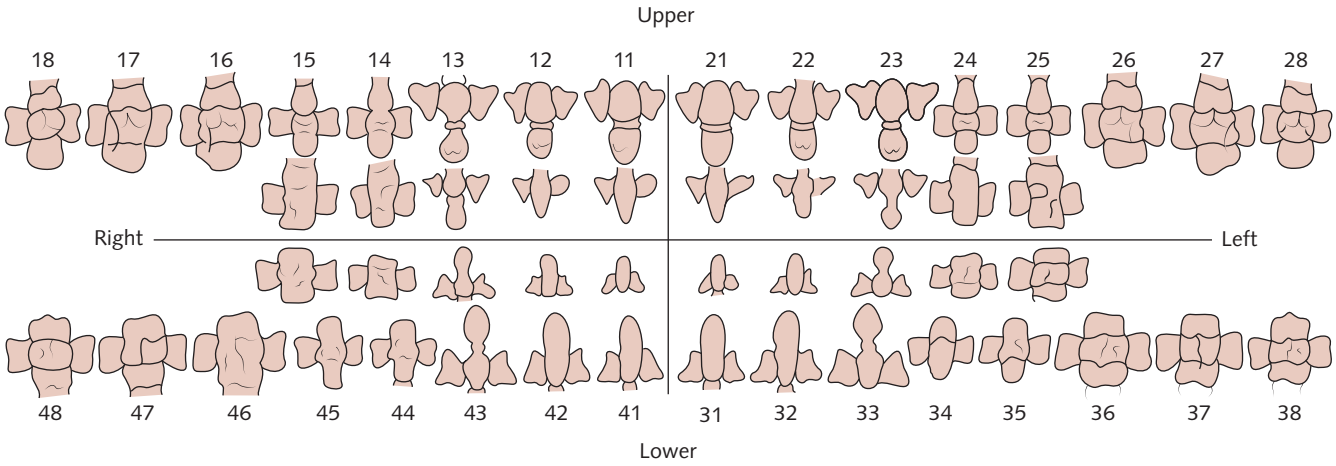
Hair

Examination of hair is of considerable help in crime investigation, as it is one of the most decay-resistant identifying features, sometimes lasting for years in favourable circumstances. Hair is frequently found at the scene of crime or upon the victim and/or suspect as contact/trace evidence. It may also be found in the hand(s) of the victim and/or suspect in an assault; in cases of rape, pubic hair may be transferred from the assailant to the victim and vice versa. In hit-and-run accidents, some of the victim's hair may be found upon the vehicle involved in the accident. In chronic poisoning by metals, examination of hair provides essential data. However, it must be clear that examination of the hair is the province of the forensic biologist. In this context, the following basic information may be considered:

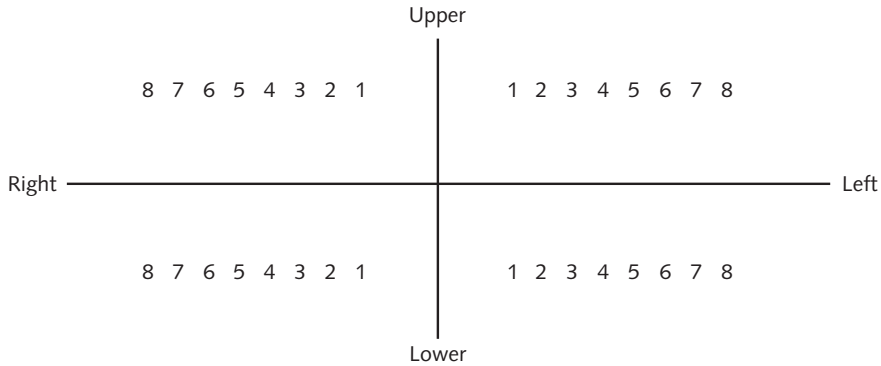
Nature (whether material is hair or some other fibre): Hair is an appendage of skin that grows from hair follicle. It has **a root (bulb or knob), a shaft and a tip**. The portion of the hair that lies in the follicle is known as the root. It is surrounded by the loose connective tissue known as root sheath. The root lies in the dermis. The shaft grows and projects out of the skin. Distal end of the shaft is known as tip. Hair consist of three zones: **(*i*) cuticle**, **(*ii*) cortex** and **(*iii*) medulla** (Fig. 3.5).

The cuticle is outer covering of the shaft of the hair consisting of scales of keratin and forming a certain pattern. The scale pattern of different animals is characteristic and differs from that of human hair. In human hair, the scales are flattened. (Moritz described seven types of scale-pattern. Type I is the elongated variety of imbricate type. Type II is the serrated or dentate variety of the coronal type. Types III, IV and V are not much elongated and there is gradual decrease in vertical length in them. Type VI is the flattened variety with smooth free margin and Type VII is the flattened variety with serrated margins. Type VII is the human type.)

The cortex is the intermediate zone of varying thickness and forms the bulk of the thickness of the hair. The cortex consists of longitudinally placed non-nucleated elongated cells. Diffuse and/or granular pigmentation occurs in the cortex, which may be distributed all around the cortex or may be limited near the central or the peripheral zone, depending upon the species. In human beings, the pigment is usually distributed



'**Odontogram**' designed for Interpol. The chart displays each surface of each tooth, including the deciduous teeth.



Palmer's notation: This system utilises numbering of teeth as 8 to 1, starting with the third molars and moving to the middle (for example: 37 designates the lower right canine).

Right								
Permanent	18	17	16	15	14	13	12	11
Temporary				55	54	53	52	51
Temporary				75	74	73	72	71
Permanent	38	37	36	35	34	33	32	31
Right								

Left								
21	22	23	24	25	26	27	28	Permanent
61	62	63	64	65				Temporary
81	82	83	84	85				Temporary
41	42	43	44	45	46	47	48	Permanent
Left								

Modified FDI System (Federation Dentaire Internationale) or Modified International System of numbering teeth.

Fig. 3.7 Dental charts.

into any system, when required at a later stage. The following features need to be recorded on the chart:

- Extractions (recent or old)
- Fillings—their number, composition and position
- Artificial teeth—metal to be mentioned, if discernible
- Other prosthetic work such as bridge-work or braces
- Broken teeth
- Crowned teeth
- Pathological conditions, if any
- Congenital defects like enamel pearls, Carabelli cusps, etc.
- Malpositioned teeth
- Racial pointers like shovel-shaped upper central incisors or multicusped molars
- General condition of care and hygiene.

Dactylography (Dermatoglyphics/ Galton System/Fingerprint Study, etc.)

History

Dactylography is the process of taking impressions of the pulp of fingers and thumbs on an unglazed white paper and examining them with a magnifying lens. The system was discovered

by Sir William J Herschel, ICS, who introduced it in Hoogly District of Bengal in 1877. It was systematised in 1892 by Sir Francis Galton, an English anthropologist, whose name it bears. It was further elaborated and improved by Sir Edward Henry of Scotland Yard. It is, therefore, also known as the Henry–Galton system of identification. The first recorded instance of a fingerprint having been used to prove the identity of a murderer was in Argentine in 1892 (the case of Francesca Rojas). In Great Britain, the first case was R vs. Stratton.

Principle

It is based on the principle that skin of the balls of the fingers and thumbs is covered with characteristic ridges, the arrangement and distribution of which remains constant and persists throughout life and that the patterns of no two hands resemble each other. Even the fingerprints of the twins are not similar. It has been estimated that the chances of two persons having identical fingerprints is about one in sixty four thousand millions. (Quetelet's rule that every nature-made object shows infinite variations of forms, and in the world's crime records no two identical fingerprint patterns have been reported.)

Classification

Primarily, there are four types of ridge patterns. According to the percentage of their distribution, these are as follows (Fig. 3.8):

Loop	–	about 65%
Whorl	–	about 25%
Arch	–	about 7%
Composite	–	about 2–3%

A considerable amount of finer details of branching and sub-branching and coalescence of ridges, island, core and delta arrangements permitting sub-grouping and an unlimited quantity of extremely fine pore details makes the system absolutely peculiar towards identification.

Recording of Fingerprints

Dactylography is a progressing science and new methods for the recording, lifting and developing of prints under different field conditions, including those from the decomposed body, appear regularly. For recording fingerprints, hands are washed, cleaned and dried to ensure clear prints. The print is taken by using printer's ink on an unglazed white paper. A **plain impression** is obtained by gently pressing the inked surface of the tip of the finger or thumb on the paper while a **rolled impression** is taken by rolling the inked finger or thumb from side to side. A rolled impression recording the complete pattern of the whole ball of the finger or thumb is much more complete and desirable than a plain one, which is only partial. In case of criminals, impressions of all the digits of both the hands are taken and preserved by the police for future identification. [Under the Identification of Prisoners Act, the police are legally authorised to take the measurements, finger impressions, footprints and photographs of persons convicted or arrested due to some crime. Also, Section 73 of the IEA empowers a court to direct any person including an accused, to allow his finger impressions to be taken and the Supreme Court has held that being compelled to give finger or thumbprints does not violate the constitutional safeguards under Article 20(3).] However, it is customary to take usually the left thumb impression of an illiterate person in lieu of signature on legal and other documents. In practice, 16–20 points of fine comparison are accepted as proof of identity.

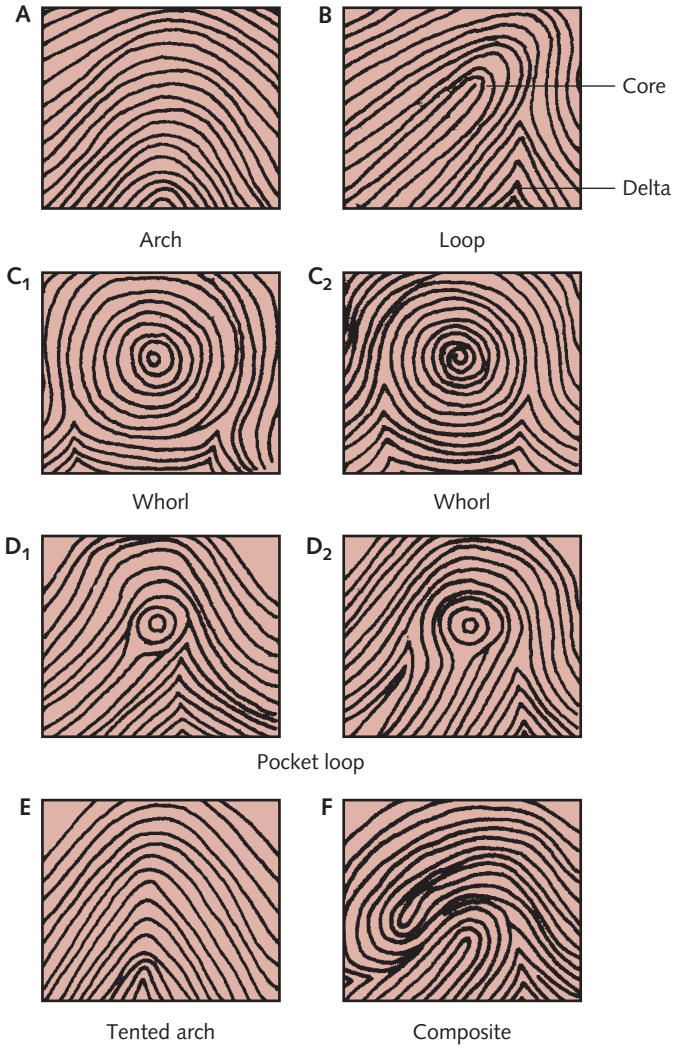


Fig. 3.8 Primary fingerprint types.

In a dead body, if the fingertips are dried up, the prints can be taken after soaking the fingers in an alkaline solution for sometime. If the skin has peeled off as a result of putrefaction, burns or drowning, etc., the prints can still be recorded either from the dermis or from the peeled-off skin hardened by formalin.

Advantages

The practical applications of this method include the following:

- Recognition of chance impression left at a scene of crime.

- Criminals often leave their fingerprints at the site of crime, unknowingly or unconsciously, which, though not apparently visible, can subsequently be developed and studied successfully.
- As stated earlier, they can be taken even from a decomposed body, either from the peeled-off epidermis of the fingers or from the dermis when the epidermis is lost.
- To apprehend internationally operating criminals, details of fingerprints can be sent from one country to another by telecommunication.
- Prevention of impersonation.
- As an extra precaution on cheques, bank notes and other legal documents that may bear a fingerprint in addition to manual signature.
- In case of unknown dead bodies, skin from finger tips should be removed and preserved in 10% formalin and handed over to police in a sealed packet for onward transmission for evaluation.
- As with fingerprints, the skin pattern of palms, soles and even lips, are said to be unique and have been used in identification, but again, this is the job of the concerned expert.

Removal or Alteration of Fingerprints

Criminals sometimes attempt to mutilate the fingerprint pattern by inflicting injuries such as wounds or burns on the bulb of their fingers or thumbs, but there still may exist definite delineation unless the true skin is destroyed. However, it has been reported that by derm-abrasion, identification by fingerprints can be circumvented. According to TJ David et al. in coeliac disease, the fingerprints may be temporarily modified or obliterated. Ridge alteration usually occurs in eczema, acanthosis nigricans, scleroderma and dry or atrophic skin. Permanent impairment of fingerprint pattern can occur in leprosy, electrical injury and after exposure to radiation. In rickets and acromegaly, though the pattern is not altered, distances between the ridges can be changed.

Superimposition Technique for Identification

Where potential candidate(s) for the identity is/are in the knowledge of the investigating agencies and the photographs taken during life are available, photo superimposition technique may be carried out for identification. In this method, photographs of the skull are taken in exactly the same orientation as the available life-time photograph of the missing person. Transparency (negative) of the skull photograph and the transparency of the life-time photograph are focused on the same sensitive printing paper. The focussing is so adjusted that different anatomical landmarks of the face (from both the transparencies) should have maximum alignment, after giving due consideration to the thickness of the soft tissues in the transparency of the life-time photograph. (It is presumed that interpupillary distance is always the same in all directions.) Then, from the positive print thus obtained that now possesses shadows of the outer surface of the face (from the transparency of life-time photograph) and skull bone (from the transparency of skull), attempt is made to study the matching, non-matching of major anatomical landmarks, which include (*i*) eyes within the orbit with two pairs of canthuses properly aligned; (*ii*) the nasion; (*iii*) the prosthion in the central line; (*iv*) the nasal spine in the centre that is little above the tip of the nose; (*v*) the lower border of the nose; (*vi*) the lower border of upper jaw; (*vii*) the zygoma below the eyes; (*viii*) supra-orbital ridges; (*ix*) angle of the jaw; (*x*) external auditory meatus; and (*xi*) the teeth.

The test mainly carries exclusory value in that, if the matching cannot be made then the skull is not that of the person in the photograph. If the match is good (or even excellent), then it can at best be said that the skull could be that of the person in the photograph.

A more modern variant of the photo superimposition technique is with the use of video cameras, where two images—one of photograph and other of skull—are mixed on one video display unit. By altering the camera angles and the degree of magnification of the images, superimposition can be tested quickly without the need for laborious photographic processing. This method was used in 1994 to identify a number of the 12 victims of the notorious 'House of Horror' in Gloucester.

Reconstruction of Facial Contour from the Skull

The method depends upon a pre-knowledge of the usual tissue thickness at various points on the normal skull, which now has quite a large database. Modelling clay is laid on to the unknown skull in layers corresponding to these standard thicknesses and some more imaginative modelling added through the information obtained from the acquaintances. The obvious defects include lack of knowledge about the eyes, lips, nose, ears, head hair, etc., all of which contribute to the individualistic characteristics. Such information may be gathered from the persons who knew the suspect and then, facial curves, creases, shape of the chin, eyebrow, shape of the lips may be mapped out.

Similar methods have been used by the graphic artists who use their portraiture talents to create a face on the two-dimensional base provided by the skull profile plus knowledge of the tissue thickness at many anatomical points. The method was used with success in 1988 in the investigation of murder of Karen Price in Cardiff, skeletonised after being buried for 8 years in a carpet beneath a garden. A medical artist rebuilt her face on a skull with sufficient accuracy for its display on public television to be recognised by her parents.

Neutron Activation Analysis

This method is very helpful in identifying minute traces of elements present in the hair, nail, drugs, soil, glass particles, gunshot residues, paints, etc. This will be helpful when comparison samples are available. It has been found that levels of trace elements in

the body hair remain uniform in an individual, and this differs from person to person. This method is also helpful for detecting abnormal amounts of metallic elements in the hair even in a highly decomposed or in an exhumed body.

In this method, hair is bombarded with neutrons in a cyclotron and the emission of spectrum therefrom is analysed. The spectrum depends upon the mineral content of the hair, which varies from person to person and hence considered to be individualistic. However, the mineral constituents of the hair may vary depending upon the dietetic and environmental conditions. Moreover, there are not many institutions in India that can undertake this precision work.

Anthropometry (Bertillon System)

'Anthrops' means man and 'metron' means measure, i.e. it deals with the measurements of various parts of the human body. It is also called the Bertillon System or Bertillonage after the name of Alphoso Bertillon, a French Criminologist who introduced it in 1882. It is based on the principle that measurements of various parts of the body do not alter after adult age (21 years) and that no two persons show the same measurements in all respects. The system is, therefore, applicable to adults only.

It comprises of registration of characteristics of the individual under three heads: **Descriptive Data** such as colour of the hair, eyes, complexion, shape of nose, ears, chin, etc.; **Body Marks** such as birth marks, tattoo marks, scars; and **Body Measurements**, namely, the standing and sitting heights, length and breadth of the head, breadth of the face, length of right ear, the span of the outstretched arms, length of the left foot, length of the left middle finger, length of the left little finger, and the length of the left forearm and hand. The photographs of the full face and side profile are also taken.

Drawbacks of the system are following:

- Applicable to adults only.
- Personal factor in measurements introduces many errors.
- Requires delicate instruments and well-trained operators.
- Photographs in themselves not being reliable means of identification.

Hence, it has been replaced by fingerprint system and the only measurements still made as a routine are the height and weight.

Other Fortuitous Comparisons

Sometimes, identification can be effected by the presence of some previous disease or any surgical condition, and comparing the same with the findings available at autopsy or in the skeletal remains. Examples may include finding of gall stone/kidney stone, horse-shoe shape kidney, uterine fibroids, etc. Surgical devices like peculiar wire-sutures; pacemakers (each pacemaker bears not only the manufacturer's name but also a model and serial number); bullet recovered from the body and compared with the bullet fired from the alleged weapon; protheses like implanted heart valves, hip or knee joints, plates in the skull and other devices may be used as a means of effecting identification.

Trace Evidence Comparisons

Edmond Locard, Head of the Institute of Criminalistics in the University of Lyon in France, coined in *Manual of Police Techniques* his 'theory of interchange' at the scene of crime (Locard, 1923, 1928, 1930). His postulation was proved when on his advice, three suspected persons linked with dealing with counterfeit coins were examined and found to carry in their garments the particles of the metals used in the counterfeit coins. The suspects were arrested and all confessed commission of the offence. Similarly, a foreign body (may be a portion of hair or piece of glass or traces of paint or of vegetation, etc.) may be found in/around the wound and/or upon the body/clothing of the victim that may provide important clue towards the circumstances. Such evidence is termed as 'trace evidence' (also see in the Chapter "Transportation Injuries").

Blood as Trace Evidence

Blood itself is an extremely important entity in the medicolegal practice, which alone or along with other trace evidences can play a clinching role to unfold different criminal problems. It is the task of the police to collect such evidence from the scene and of the autopsy surgeon to collect such evidence from the victim's body/clothing, etc., which may help the police in locating matching materials from the suspect(s) in order to provide objective evidence of their presence at the scene. Following are the steps in the examination of blood/blood stains:

- Whether the stain is of blood or not.
- If of blood, whether human or of animal origin.
- If human, then one should try to find out
  - Age of the stain
  - Arterial or venous origin
  - Whether of antemortem/postmortem origin
  - Source of blood
  - Distribution pattern of the blood (Figs. 3.9A–C)
  - Sexing of the stain
  - Blood group of the stain

Whether the Stain is of Blood or Not Benzidine test and phenolphthalein test (Kastle–Meyer test) are used to find out if a stain is of blood or not. These chemical tests are based upon the presence of enzyme peroxidase in the red blood cells. The action of the peroxidase is demonstrated with change in colour of the reagents used in the tests. Hydrogen peroxide is used in tests. This, when acted upon by the peroxidase, liberates oxygen that acts on the reagent that changes in colour (blue colour changes in case of benzidine test and pink colour changes in case of phenolphthalein test). Benzidine test is very sensitive (positive with 1 in 1000,000 dilution). But benzidine

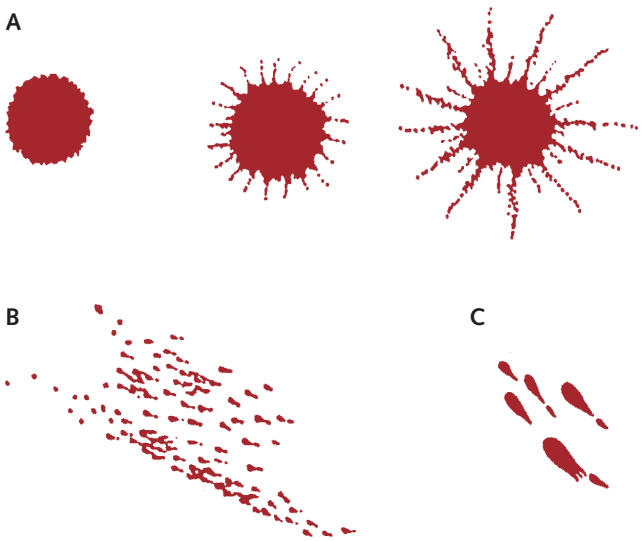


Fig. 3.9 Diagrammatic representation of types of blood spots commonly encountered at a scene showing: **(A)** Drops of blood falling vertically on the flat surface. Depending upon the distance of the fall and the recipient surface, they may have scalloped edges at shorter distances or more secondary spatters with a longer fall; the peripheral spatters growing finer and larger in number with the increase in length. **(B)** Jetting or spurting of blood. **(C)** The splashes caused by striking the surface at an angle usually appear like spears or exclamation marks depending upon velocity and angle of the fall; the 'dot' of the mark points towards the direction of path of the drop of blood.

powder is a known carcinogen. The negative test is of more value as it rules out blood but the positive test suggests that further steps be taken to confirm the nature of the stain because vegetable stains, salivary stains, pus, rust, etc. can also give positive reaction with this test. Phenolphthalein test is more specific for blood but comparatively less sensitive.

Confirmatory tests for blood include the following:

- Microscopic examination
- Microchemical tests, namely:
  - Haemochromogen crystal test (Takayama test).
  - Haemin crystal test (Teichmann test).
- Spectroscopic examination.

Microscopic examination: When frank blood is available, it can be examined as such under the microscope. In case of stain, the extract should be prepared. The stain may be scraped out with the help of a blunt pointed blade and dissolved in normal saline solution before examination. If the stain is on the cloth, the piece of cloth may be left overnight dipped in normal saline to get the stain extract. The stain extract thus obtained may be placed on the glass slide and covered with a cover-slip and observed under the microscope. Intact red blood cells confirm the stain to be of blood. Human red blood cells are circular, biconcave, non-nucleated and are of average diameter of 7 μm. All mammalian red blood cells are circular, biconcave and non-nucleated except that of camels. In fish, avians and amphibians, the red blood cells are oval, biconvex and nucleated.

Staining of the film of the extract with **Leishman stain** will provide added information about white blood cells also, which may help to know the sex of the origin of the blood by counting Davidson bodies in the polymorph cells.

Microchemical tests: Of both the tests named above under this head, **haemochromogen crystal test** (Takayama test) is more dependable but is comparatively more time consuming. These tests are based on the property of haem (iron) part of the haemoglobin to form characteristic coloured crystals with certain reagents, and these crystals can be appreciated microscopically. (Pink feathery crystals of haemochromogen and dark brownish rhomboid-shaped crystals of haemin are diagnostic of blood.) False negative results may be obtained in both the tests (*i*) if the stain extract is contaminated with some chemical, (*ii*) if the stain is very old or decomposed or (*iii*) if the reagents are very old/defective.

Spectroscopic examination: It is more reliable both for the recent and old blood stains. Less than 0.1 mg blood is sufficient. The blood stain is dissolved in normal saline or dilute ammonia and is placed in a small glass test tube, which is then kept between the spectroscope and the source of light. The solution of blood has the property of absorbing some of the rays from the spectrum of light producing characteristic dark absorption bands that vary with the type of the blood pigment present.

Whether the Blood is of Human or Animal Origin This includes Serological Testing of the blood. Various methods in use are gel diffusion, antiglobulin consumption test, isoenzyme methods and precipitin electrophoresis.

Age of the Stain Colour and nature of the stain can help in ascertaining the age of the stain:

- Fresh stain on light-coloured cloth appears bright red, is moist and sticky.
- Turns reddish brown in 24 hours.
- More than 24 hours—it is dark brown and black on longer duration.

Whether Arterial or Venous Recently shed arterial blood is bright-red and venous blood is dark-red. Bleeding from arteries has a sprouting effect (jet like ejection/spurting), while bleeding from a vein occurs passively (Fig. 3.9B).

Whether of Antemortem or Postmortem Origin Antemortem bleeding causes coagulation when the blood partly solidifies with separation of serum. The clot can be taken out en masse from the spot, and the stained area after removal of the clot usually retains the impression of fibrinous network owing to the process of clot formation. On stretching, the clot can be separated in scales due to presence of fibrin. Postmortem solidification occurs without proper coagulation change and the clot cannot be taken out en masse. On removal from the

Table 3.17 Source of Blood and Appearance Thereof

Source	Appearance
Stomach or gastric bleeding	Chocolate coloured due to presence of acid haematin and is acidic in reaction
Nasal bleeding	Blood mixed with nasal mucus and hair, etc.
Haemoptysis	Bright red (being oxygenated in the lungs) and frothy (due to some churning effect with the inspired and expired air). Reaction is alkaline
Menstrual blood	Dark coloured fluid blood with foul smell and often with endometrial debris. Acidic in reaction. Vaginal epithelial cells may be present
Abortion	Dark clotted blood. Endometrial and placental debris with some foetal remnants present

spot, it does not leave the impression of fibrinous network. On stretching, the clot becomes powder.

Source of Blood Table 3.17 describes source of blood.

Distribution Pattern of Blood The pattern of distribution of blood at the scene may give an insight into the actions and activities of the victim and the assailant and may be of particular value in evaluating direction of travel of blood spots, the distance travelled, etc., as shown in Figs. 3.9A, B and C.

Sexing of the Blood Stain As written earlier, Leishman staining of the extract of the stain can help in ascertaining the sex by studying the presence of drumstick bodies (Davidson bodies) in the polymorph cells.

Grouping of the Blood Stains Whether the blood or blood stain belongs to group A, B, AB or O can be known by testing for antigen (agglutinogen) present on the surface of blood cells if the cell structure is intact or indirectly by testing for the antibody (agglutinin) present in the serum. According to the antigens present on the surface of red cell membranes, blood can be classified into four major groups, i.e. Group A, B, AB and O. The serum contains antibodies known as anti-A and anti-B. Red cells with antigen A will react with serum containing anti-A antibody and will result in clumping of red cells and this is **known as agglutination**, which forms the basis for blood group testing. Similarly, the red cells with B group antigen will react with anti-B antibody. The red cells containing A and B antigens will be agglutinated by both anti-A or anti-B antibody. The blood in which red cells have neither A nor B antigen is known as Group O. This group, therefore, is not agglutinated by either of the antibody. That is why Group O blood can be transfused to any person, and the person having blood Group O is known as **Universal donor**. Similarly, Group AB is called the **Universal recipient** because there are

Table 3.18 Blood Grouping

	A	B	AB	O
Anti A	+	–	+	–
Anti B	–	+	+	–

+represents presence of agglutination.
– represents absence of agglutination.

no antibodies in the serum and, therefore, the person belonging to this group can receive blood of any group.

Methods for determining blood group

- **When the red cells are intact**, then direct agglutination test with the help of known antisera can be undertaken either by the tube method or by the tile method. **In the tube method**, red cells are washed with normal saline and suspended for sometime. One drop of cell suspension is added to equal volume each of anti-A, anti-B and O-group serum in separate tubes and left for an hour or two at room temperature. Presence/absence of agglutination is appreciated to know the blood group as mentioned earlier. **In the tile method**, one drop of cell suspension and one drop of each antisera are mixed separately in different well of the tile, shaken by rod and observed for clumping (agglutination). The results are mentioned in Table 3.18.
- **When the red cells' structure is damaged** and they cannot be subjected to agglutination test, then the techniques like absorption inhibition, mixed agglutination and absorption elution are the proper techniques for determining blood group.

Medicolegal applications of blood groups: The knowledge of manner of inheritance of blood groups carries an obvious application in cases of disputed paternity and maternity. Blood groups are inherited as per Mendel Laws of Heredity. The two important principles in this context are:

(1) A blood group antigen cannot appear in a child unless it is present in one of the parents.
(2) If one of the parents is homozygous for a particular blood group antigen, that antigen must appear in the child's blood.

Thus, from the above first point, we get that if the blood cells of child contain A antigen, then at least one of its parents have A antigen in his or her blood cells. From the second point, we get that if father or mother has homozygous A antigen (AA), then it must be present in the blood cells of the child (Table 3.19). (The only chance of exception of the above two theories is the chance of mutation that may occur in 1 in 50,000 newborns.)

Blood group evidence has been accepted by the courts in cases of disputed paternity for more than 50 years, but it is only in the last few years that a legal framework has been recognised in this direction. The courts can give directions for

Table 3.19 Inheritance of ABO Blood Groups

Blood group of		Blood group of children	
Parent 1	Parent 2	Possible	Not possible
O	O	O	A, B, AB
O	A	O, A	B, AB
A	A	O, A	B, AB
O	B	O, B	A, AB
B	B	O, B	A, AB
A	B	O, A, B, AB	None
O	AB	A, B	O, AB
A	AB	A, B, AB	O
B	AB	A, B, AB	O
AB	AB	A, B, AB	O

Table 3.20 Chance of Exclusion of Non-fathers

System	% Individual chance of exclusion	% Combined chance of exclusion
Red cell antigens		
ABO	17.6	17.6
MNSs	32.1	44.1
Rhesus	28.0	59.8
Kell	3.3	61.1
Duffy	4.8	63.0
Lutheran	3.3	64.2
Kidd	4.5	65.8
Serum proteins		
Haptoglobins (Hp)	18.1	72.0
Group specific components (Gc)	15.9	76.5
Ag	14.2	79.8
Gm^1Gm^2	7.8	81.4
Km^1	4.1	82.2
Red cell enzymes		
Phosphoglucomutase (PGM)	24.5	86.6
Red cell acid phosphatase (EAP)	21.0	89.4
Glutamate pyruvate trans aminase (GPT)	18.7	91.4
Esterase D (EsD)	9.0	92.1
Adenosine deaminase (ADA)	4.5	92.5
Adenylate kinase (AK)	4.2	92.8
Glyoxalase 1 (GLO)	18.0	94.1
Total combined chance of exclusion		94.1%

the blood testing if one of the parties involved in the proceedings made an application and if, either party refuses to comply with the directions, the court is empowered to draw any inference from this failure, if it thinks proper to do so under the circumstances.

The question of **disputed paternity** may arise in the following circumstances:

- Alleged adultery and suits for nullity of marriage. When the child is born in lawful wedlock and the husband denies that he is the father of the child and seeks divorce on this ground.
- Blackmailing: When a child is born out of lawful wedlock and the mother accuses a certain man as the father of the child but the man denies the accusation.
- Suits for maintenance of illegitimate children.

The question of **disputed maternity** may arise in the following circumstances:

- When two women claim the same child.
- When there has been an allegation of interchange of a child with another in the maternity home or hospital.
- In case of a kidnapped child, when the woman who has kidnapped the child claims to be the mother. She may name a friend as alleged father.
- In case of suppositious child, when a woman pretends pregnancy and delivery, and brings forth a suppositious child to pass it off as her own.

Exclusion of Paternity: Exclusion of paternity falls into two classes:

- First-order exclusion: Where the child possesses a blood group antigen that is absent in both the mother as well as the putative father. As for example, supposing the child's blood group gene is B and that of mother's and putative father's are O. It follows that child's blood group B gene could not have come from either the mother or the putative father, so he is excluded from paternity.
- Second-order exclusion: Where the putative father is homozygous for a blood group antigen, he must pass that antigen on to all his children. But the antigen is not present in the child in question. So, non-paternity is indicated.

Non-exclusion of Paternity: Where no exclusion of paternity is obtained, it is of value to the court to be provided with some indication as to the likelihood of paternity. False conclusions may be reached if blood relative of the alleged father is involved or the parties hail from an isolated community in which inbreeding may have occurred, resulting in a gene uncommon in the general population being more frequent.

With the present range of tests, over 90% of falsely implicated men can be excluded from paternity so that a non-exclusion result in itself carries significant evidentiary value to the mother. Blood group systems commonly employed for the purposes of exclusion of paternity are listed in Table 3.20 and another important system, HLA system, has been described below:

The HLA System consists of a series of antigens present on the lymphocytes. The chance of excluding a falsely accused

man using a comprehensive range of HLA genetic markers is about 96%. This high exclusion rate makes it the most important system for use in paternity problems, but a number of practical difficulties limit its usefulness. A particular difficulty is due to short life-span of the lymphocytes so that many samples may become unsuitable for testing. Others are the cost factor and lack of well-equipped laboratories and well-trained experts.

Medicolegal Importance of Blood Examination In **Civil Cases** like paternity/maternity issues, divorce and nullity of marriage, compensation cases related to workmen's welfare considerations or civil negligence issues arising in hospital or medical practice. In **Criminal Cases** like identification of the victim or the offender of a crime of assault, homicide, sexual offences or where death occurs due to rash or negligent act of the offender, etc. (In case of homicide, if the blood stain present on a lethal weapon matches with the blood stain present on the wearing apparel of a suspect on the one hand and blood of the victim on the other, then relationship between the offence of homicide, the victim, the offender and the offending agent gets authenticated.)

Mass Disaster

Every accidental death is a disaster to the individual family involved and (to them) is of the same dimension irrespective of how many others were similarly affected at the same time. Every accidental death has its preventive aspects and, often, these lie in the hands of the pathologist (Mason, 1989). **WHO has defined disaster as** an occurrence that causes damage, ecological disruption, loss of human life or deterioration of health and health services on a scale sufficient to warrant an extraordinary response from outside the affected community or area. A 'Mass Disaster' is considered to have occurred when the number of casualties occurring in a single event exceeds 12. (This figure may be modified in accordance with local criteria as applicable in a country or a city.)

CLASSIFICATION

A. Natural:

(i) Non-biological—earthquake, cyclone, flood, drought, heat wave, volcanic eruption, landslide, and most recently, 'the tsunami'.
(ii) Biological—disease epidemic, mass poisoning (food/liquor).

B. Man made:

(i) Accidental—transportation (road, rail, sea, river, and air), building collapse, mining accidents, dam bursts, food poisoning, fires, football tragedies like Ibrox Park, Heysel, Hillsborough, crush tragedies at Mecca, etc.
(ii) Industrial—fires, explosions, leakage of toxic substances/gases.
(iii) Civil disturbances—riots and demonstrations.
(iv) Warfare—conventional (bombardment, exchange of fire, shelling) and non-conventional (nuclear, biological and chemical warfare, terrorism).

Objects usually include:

(i) Retrieval, reconstruction, examination and early disposal of the bodies [it may be worth focussing that the medico-legal expert may become open to criticism (or even to action) for pain and suffering brought about by the delay in releasing a body to its next of kin].
(ii) Establishing identity.
(iii) Conducting autopsy, wherein practicable, and to establish the cause of death [all necessary factual evidence needs to be obtained at the time of autopsy, including samples for histology, toxicology, odontology, radiology and DNA analysis (depending upon the case)].
(iv) Seeking evidence in the form of some foreign material/fragment (of bomb or detonator) that may be embedded in the bodies.

MANAGEMENT

It is primarily a teamwork of the civil administration and its agencies (especially the police) on one hand and a multi-disciplinary medical task force usually comprising of clinicians, nurses, paramedical staff, odontologists, radiologists, forensic scientists, mortuary assistant, and funeral officials, etc., on the other hand, resources of the Armed Forces may be called-in to reinforce efforts. Over the past couple of decades, many countries have established protocols for the management of disasters, often with the setting up of Disaster Victim Identification (DVI) teams. Recently (after 'tsunami disaster'), Indian Government also constituted the National Disaster Management Authority under the Chairmanship of the Prime Minister with a Vice Chairman and five members. This authority aims at bringing about a change in orientation from a relief-centric approach to a holistic multi-disciplinary and multi-sectoral approach.

Because the circumstances differ so much from incident to incident (as described above), it is impossible to try and anticipate every contingency and draw detailed plans to cope with them (Walsh, 1989). Hence, protocols need be 'simple and flexible'. Commonly involved considerations may be as under:

Isolation, Demarcation and Protection

Isolation, demarcation and protection of the site by the security cordon and entry of the team through some predetermined route. Involvement of by-standers and other officials need be checked. An experienced Lieutenant Commander of the New York City Police Department once remarked that the greatest problem he encountered in protecting the scene was

the unauthorised attendance of police personnel and high-ranking officials coming along to help. This occurred particularly in the more 'high-profile' cases, which were attended by a great deal of publicity. Further, as remarked by Geberth, "such visitors either contaminated or destroyed valuable evidence or got in the way. They frequently left behind material, initially thought to be valuable trace evidence, which was consequently, needlessly examined". Other organisational features have been well canvassed and a vade mecum has been published by the Royal College of Pathologists.

Identification and Investigation

Identification and investigation are not different exercises but are complementary in at least two respects, viz.:

- In accidental cases, identification of the individual may contribute towards cause of the accident in case the deceased had the history/record of having some pre-existing disease or some toxicological problem (age and experience go hand in hand, as do age and atheroma).
- It may afford some explanation towards the pattern of injuries (especially when sitting arrangements are available).

Some regard the exercise of identification as the major priority, as was the case at Mount Erebus. The Royal College of Pathologists places it first in the list of medicolegal expert's responsibilities. **Others take the view** that identification is ultimately the responsibility of the police and that the medicolegal expert's obligation to provide such information/evidence of identity as available stands ancillary to his/her responsibility towards investigation.

Visual recognition may not be helpful because of severe disfigurement (due to trauma, burning or decomposition, etc.). Further, the emotional factors involved in visual recognition make it unwise to accept either a positive or negative response in such circumstances. Major disaster usually involves an international dimension, and it is imperative to be sensitive to customs and traditions of each community in order to avoid future action.

Belongings can be helpful and may include clothing, jewellery, and personal documentation. Clothing found on a body can be of great value in establishing identity but must not be accepted as a positive proof in isolation. The corpse needs be searched and undressed very carefully (even the badly incinerated corpse may have a fragment of recognisable underclothing in the depth of the inguinal fold, axillary fold, natal clefts, etc.). Such clothing may also contain evidence of the use of a fire accelerant. Timperman (1991) in discussing the Zeebrugge ferry disaster put forward a proposition that the clothing need be removed by medical personnel, believing that the cadaver was entitled to privacy in the same way as is the living victim/patient. Jewellery being relatively fire-resistant can be very helpful, especially when some unusual and strongly identifiable fragment is available. However, it being secondary evidence must not always be assumed that the item belongs to the deceased or will be recognised by relatives (ACPO EP, 1999). Personal documents carry potential value but again, being secondary evidence, should not in their own be used to assume the identity of the deceased. The compact and protective way of carrying documents can often afford a great protection from fire and water, and every tiny fragment can be useful and therefore be handled with care.

Medical and X-ray evidence may contribute or confirm identification. Clearly, the more unusual the medical or post-surgical condition is, the more positive will be the identification of the body. Comparison of X-rays taken in life may be used when there is some abnormality. Alternatively, normal structures may be of such a variety that they can almost provide proof of personal identification—the cranial sinuses and the pituitary fossa are good examples.

Odontology has proven to be very helpful, especially when fire or putrefaction has destroyed the soft tissues. Obviously, the method needs obtainable dental records. Seventy-three percent of the deceased were identified dentally in the Mount Erebus disaster that involved predominantly Western and Japanese people and included a high proportion of New Zealand adults who had notorious amount of dental work done on them prior to fluoridation.

Fingerprinting is another method of personal identification with well-established criteria for reliability. Obviously, fingers or fingerprints may be destroyed in the event of fire or severe mutilation. It is for this reason that some recommend retaining of heel print records too, the heel being relatively better protected from fire or decomposition by shoes or boots. [In the Mount Erebus accident wherein all 237 passengers and 20 crew died, 1 passenger, though badly burnt about the face had a wallet in his hip pocket that contained a plastic identity card including fingerprints, which matched the fingerprints of the body.]

DNA-profiling has been shown to be of great value in identifying a wide range of human fluids and tissues, chiefly because DNA molecules remain highly stable in stains, are present in virtually all human cells and are extremely polymorphic.

Management of Survivors

In case of survivors, establishing a system of Triage to determine priority for evacuation is the need of the hour. Colour-coded tokens may be hung around the neck of the injured. The colours recommended are the following: Category I (Red)— requiring resuscitation and emergency lifesaving surgery, Category II (Yellow)—requiring possible resuscitation and early surgery, Category III (Green)—less serious injury not endangered by delay and Category IV (Black)—survival not likely.

THE TSUNAMI DISASTER

On 26th December, 2004, an earthquake struck near the western tip of Sumatra's west coast. The epicentre was located

Table 3.21 Guidelines for Collection, Preservation and Dispatch of Samples for DNA Testing

Material/circumstances	Guidelines for collection and preservation
Maternity/paternity/parentage testing	Blood samples of mother, disputed child and alleged father are required; 2–3 ml blood can be collected in sterile blood collection material (EDTA vials) in the presence of some judicial/administrative authority. Samples should be sent in ice in a thermos flask either by a messenger or through courier, so as to reach the laboratory within 72 hours after collection.
Blood (Now-a-days, FTA paper is being used for collection of blood samples. This is an absorbent cellulose-based paper that contains chemical substances to protect DNA molecules from nuclease degradation and preserve the paper from bacterial growth (Seah and Burgoyne, 2000). Use of this paper simply involves adding a spot of blood to the paper and allowing the stain to dry.	As red cells have no nuclear DNA, sufficient amount of blood must be obtained to extract DNA from the much sparser leucocytes. Preferably 5 ml is taken into an EDTA tube, which not only prevents clotting, but also inhibits enzymes that may break down DNA during storage. The sample should be kept in the plastic tube in the ice-making compartment of an ordinary refrigerator if there is delay for sending the sample to laboratory. Blood stains should either be sent intact on surfaces, keeping as cool as possible before and during transit to the laboratory. Alternatively, stain may be rubbed with a sterile cotton-wool swab moistened with water. This swab is then air-dried without heat, put in a plastic tube/container and frozen (if there is some delay in sending to the laboratory).
Saliva	Saliva is collected in a test tube, diluted with an equal volume of normal saline and placed in boiling water bath for about 10 minutes (for destroying enzymes that inactivates blood group substances), allowed to cool and transferred to a clean, sterile and well-stoppered bottle that should be properly sealed and labelled. Bite marks can be challenging to identify. Early recognition is rewarding because the suspected bite mark can be swabbed for salivary DNA with the help of a sterile saline water-soaked cotton swab (washing or medical treatment of the area, or where the sample has been obtained through clothing may negate the findings).
Buccal epithelial cells	By gently rubbing a wet toothbrush across the buccal surface, the brush can be tapped onto the surface of FTA paper for sample storage and preservation. Alternatively, cells may be collected by using sterile, dry swabs. Two samples are taken; one swab is rubbed on the inside of the left cheek and the other swab is used on the right cheek. The swab should be identified and left to dry at room temperature in a protected area (swabs must not be placed in a container until they are completely dry since the bacteria in saliva proliferate rapidly in moist conditions and will degrade DNA). All such swabs need to be frozen or kept under effective cooling.
Clothing (Personal/bed sheets, bed covers/pillows or pillow covers, etc., as the situation may be)	All wet or dry clothes with wet stains should be dried in air, transferred gently into strong sterilised paper bags, sealed and labelled. It is preferable that each piece of clothing be folded inward, placing a piece of clean paper against any stain, so that the stains are not in contact with the bag or other parts of clothing. If delivery to the laboratory is delayed, store in a cool dry place that is secured.
Hair	In rape (and in other sexual offences, if necessary) cases, loose (transferred) pubic hair should be collected from the victim and the accused before collecting their controls. Control/reference samples (for comparison with questioned hair) need be collected from the victim, the accused and other involved persons (the hair sample preferably be full stranded and should represent the body part concerned). Hair mixed with blood, body fluids or other tissue should be air dried and packaged in thick envelopes, sealed and labelled properly.
Swabs/smears	Sterile cotton is used for absorption of vaginal/urethral fluid by touching suitable regions of vagina/urethra, air dried, placed in sterile test tubes, sealed and labelled properly. Before drying, swabs may be used for preparing smears on plain sterile slides, without rubbing it as the latter may cause the spermatozoa to break and thereby give a false negative result. The slide is then air dried, covered with another slide and wrapped in paper for sending it to laboratory after proper sealing and labelling. (No fixative should be used.)

(Contd.)

Table 3.21 (Continued)

Material/circumstances	Guidelines for collection and preservation
Seminal and vaginal fluids	Liquid semen found in the vagina or elsewhere should be recovered with a fine pipette, placed in a small plain tube and preserved under effective cooling. Seminal stains on small items of fabric or any other small objects need to be kept as cool as possible during transit.
Autopsy samples (The fresher the tissue, the better it is. However, rapidly developing new techniques are allowing the recovery of recognisable DNA from old material. In a recent Welsh murder, Professor Alec Jeffreys was able to confirm the identity of a young girl buried in a garden 8 years previously, from dried bone marrow recovered by him from the completely skeletonised remains.)	Deep **muscle tissue** samples are most suitable as it is most resistant to decomposition. Two skeletal muscle fragments need be selected (weighing about 10 gm and approximately 2 cm wide) from the best preserved area of the body. Place the fragments in a plastic container having a wide mouth and screw-on lid. The next choice is the cardiac muscle. **Teeth:** After completing dental chart, at least four teeth need be selected (preferably molars), wrapped in a dry paper or cloth and labelled properly. **Hair:** Between 10 and 15 hair with roots should be pulled from the subject, wrapped in dry paper or cloth and labelled properly. **Postmortem blood:** A sample of about 10 ml of blood should be drawn into a tube containing an anticoagulant (EDTA type).
Charred corpses Despite external appearance, the stability of DNA at high temperatures allows genetic analysis in corpses, where charring is not complete, by using fragments of skeletal muscle from deep regions of the body and from semi-solid blood that remains inside cardiac cavities.	**Bones:** Preferably long bones, like femur or humerus, should be selected. In addition, the bone like sternum or ribs can also be used. Sticking decomposed tissue should be removed, and clean bone should be packaged in the evidence box/container (if a bone saw is used, it is preferable to use a new blade each time to avoid DNA contamination. If it is not practicable, a used saw blade should be washed repeatedly with bleach and soap solution). **Teeth** (preferably molars) remain the tissue of election.
Special precautions: (a) Wear gloves and change them often, (b) use disposable instruments or clean them thoroughly before and after handling each sample, (c) avoid touching an area where you believe DNA may be present, (d) avoid talking, sneezing or coughing over evidence, (e) avoid touching your face, nose and mouth when collecting and packaging evidence, (f) keep evidence dry and transport it at specified temperature and (g) ensure that the chain of custody is maintained at all times, etc.	

155 miles southeast of the provincial capital Banda Aceh, the worst affected country in this disaster. The vast amount of the damage caused by the earthquake was due to the resultant tsunami—a series of giant waves triggered by the shift in earth plates 10 km under the water surface. Waves travelled some 3000 miles and affected the coast of many countries. The worst affected was Indonesia reporting 220,153 deaths, Sri Lanka coming the next, reporting 31,147 deaths.

As reported, The Forensic Services encountered great difficulty in identification, display, handling, storage, recording and disposal of large number of dead bodies. Mass embalming was resorted in order to overcome lack of storage facilities. Other features of forensic importance included public display of bodies for identification, photographing and recording of valuable anthropological data with collection of personal effects and disposal in mass graves. [A well-defined portion of the cemetery was allocated for disaster victim identification system with a separate plan for the DVI graves and numbers were allocated to the graves. In each coffin, photograph of the body, location where the body was found, external description with particular reference to special marks, clothing, deformities, fingerprints, odontological data, postmortem DNA profile, etc. were kept in a weather-proof case. Jewellery and clothing with corresponding number were kept in sealed bags in the custody of police. A concrete monument was erected on each grave carrying the identification number. Coffins were also uniquely marked to enable identification in the event of re-exhumation.]

Collection and Preservation of Biological Material for DNA-Profiling

The essence of DNA profiling in forensic work is comparison between two samples. One is the **'test/evidence'** sample from the crime scene such as semen from the vagina of the rape survivor or blood-stained clothing or objects with which he or she has been in contact, and the other is the **'control/reference'** sample such as blood, hair or tissue provided by a known person, for instance a victim or suspect/a group of suspects. The sensitivity of DNA technology is changing the strategy of sample collection since sensitive low-copy-number DNA methods are being developed allowing the detection of

subnanogram amount of DNA. It is, therefore, extremely important to avoid cross-contamination from the personnel collecting the samples (Collection of reference samples from living subjects requires legal authorisation and informed consent given by the person providing the sample. In the case of minors or mentally disabled persons, there should be parental or custodial consent in addition to legal authorisation. However, exception may be exercised in circumstances falling under Section 53 CrPC]. Guidelines are being furnished in Table 3.21.

In OJ Simpson case, the defence essentially put the crime laboratory on trial as the main point of discussion, as generated from the circumstances, was that there could be possibility that some of the DNA drawn from the suspect could have accidentally mixed with the DNA retrieved from the crime scene. The case involved 'double-murder' that occurred in June, 1994 in West Los Angeles (USA). The victims were the beautiful white wife, Nicole Brown of black Heisman trophy winner and football great Orenthal James Simpson and a handsome Ronald Goldman, a waiter. The Los Angeles Police Department (LAPD) found enough evidences at the murder scene and in Simpson's vehicle (Ford Bronco) and in his nearby mansion. This led the police to focus quickly on Simpson as their primary suspect. However, Simpson was acquitted by the court by giving him 'benefit of doubt' emerging, amongst other factors, from the doubts appearing in context with the integrity of 'evidence sample' and the 'reference sample' of blood. Some similar contention formed the basis of acquittal of the accused at the trial court in the **Priyadarshini Mattoo case** (the victim, a Law Student, was allegedly raped and strangled to death by the accused, son of a highly placed official). However, things turned different at the appellate court.

4
CHAPTER

Death and Its Medicolegal Aspects (Forensic Thanatology)

After going through this chapter, the reader will be able to describe: Death | Presumption of death and survivorship | Somatic and molecular death | Brain death with its medicolegal aspects, especially in relation to organ transplantation | Suspended animation | Mode, manner, mechanism and cause of death | Estimation of 'time since death' from the immediate, early and late changes after death, and factors influencing such changes | Medicolegal aspects of immediate, early and late changes after death | Differentiation between postmortem staining and bruising, hypostasis and congestion, rigor mortis and cadaveric spasm, rigor mortis and conditions simulating rigor mortis, etc. | Postmortem damage by predators | Entomology of the cadaver and postmortem interval

Death is the end of dying. It is a process rather than an event except in the exceptionally rare situations where death may be almost instantaneous such as in case of crushing of the brain in a vehicular accident, death in a nuclear explosion or in a bomb blast, etc. Need usually arises to ascertain specific time of death whether in family or in business affairs. Inheritance of property and acquiring a business often revolves around this. Legal systems usually contain provisions regarding the **'Presumption of Death'** and **'Presumption of Survivorship'** depicting the importance of these aspects. Sections 107 and 108 of IEA lay down that if it is shown that a person was alive within 30 years, and there is nothing to suggest the probability of his death, it is presumed that he is still alive unless proof be produced that the same person has not been heard of for 7 years by those who would naturally have heard of him, had he been alive. The onus of proving that the person is dead is shifted to the individual who asserts such fact. An example in this context may be cited of the disappearance of the Prime Minister of Australia while swimming on a rocky, deep seacoast near his home. It was followed by assumption of power by his successor after a few days when he was presumed legally dead, but uncertainty may prevail in certain circumstances where there is no obvious explanation for the disappearance in contrast to such factually based presumptions and hence the legal provisions.

Black's Law Dictionary defines **death** as, 'The cessation of life; the ceasing to exist defined by the physicians as a total stoppage of circulation of the blood and a cessation of animal and vital functions consequent there upon, such as respiration, pulsation, etc.' Here, the emphasis was placed on the cessation of respiration and circulatory function, but it was obvious that all systems would fail quickly after any of the vital functions had failed, viz., nervous system, circulatory system and the respiratory system. That is why these are known as atria mortis, **death's portals of entry**.

This **unified or interdependent concept of death** lingered on for centuries until recent times when the medical-care advances have made it possible to maintain respiration and circulation for long periods through heart–lung machines. However, the tragedy is that the irreversible damage to the brain often occurs during the short period when breathing/circulation has been suspended. Serious permanent impairment can occur with only 4–6 minutes of oxygen deprivation, and total loss of function may often occur when deprivation exceeds 6–10 minutes. Hence, there appeared the development of concept of 'brain death'.

Brain Death

The first proposal to determine brain death by permanent loss of consciousness is generally assigned to Mollaret and Goulon in France in 1959. However, it was Ad Hoc Committee of Harvard Medical School that examined the definition of brain death in 1968. The Committee was composed of 13 members—ten were doctors representing various disciplines of medical science and three were non-doctors, i.e. a lawyer, a theologian and

a historian of science. It recommended three criteria for determining permanent non-function of the brain:

- Unreceptivity and unresponsivity
- No movements or breathing
- No reflexes

And an added confirmatory test proposed was 'a flat' or isoelectric electroencephalogram. The fact that EEG is an objective test, while all other require subjective clinical judgements by the doctors, substantiates its strength.

The criteria of Harvard Ad Hoc Committee have since been generally accepted throughout the world. Currently, brain-stem has been the focus of attention where vital centres are situated because various strata of brain behave differently in their response to oxygen deprivation. Therefore, circumstances may be there, where cortex has been damaged but the lower brain including brain-stem is still functioning. In such a state, the victim will exist in a 'vegetative state', **the so-called 'living cadaver'**. The victim can remain in deep coma for a considerable period; may be for years. However, when brain death spreads below the tentorium, i.e. when base of the brain including midbrain, pons and medulla suffer damage, the loss of vital centres and consciousness will cause the victim not only to be irreversibly comatose but also to be incapable of spontaneous breathing. Without medical intervention, the cardiac arrest invariably follows within minutes and then the usual process of 'cellular death' progresses. Once irreversible damage to the brain-stem has been established, the victim is dead in the somatic sense, though not yet dead in the cellular sense. It is through this 'physiological gap' that the advances in removing the organs from the cadavers for the transplantation purposes have broken through.

With the passage of **Transplantation of Human Organs Act, 1994** (the Act was enacted in July 1994 and notification was issued in Gazette of India on 4th February, 1995), India has also given statutory sanction to the concept of brain-stem death. The Act defines a 'deceased person' as one in whom permanent disappearance of all evidence of life has occurred by reason of brain-stem death or in the cardio-pulmonary sense, at any time after the live-birth. Brain-stem death has been defined as the stage at which all the functions of brain-stem have permanently and irreversibly ceased. **The brain-stem death needs to be certified by a board of doctors consisting of the following:**

- The registered medical practitioner in charge of hospital in which brain-stem death has occurred.
- An independent registered medical practitioner being a specialist to be nominated by a registered medical practitioner specified in clause (i) from the panel of names approved by appropriate authority.
- A neurologist or a neurosurgeon to be nominated by a registered medical practitioner specified in clause (i) from the panel of names approved by an appropriate authority.
- The registered medical practitioner treating the person whose brain-stem death has occurred.

Certain preconditions to be fulfilled before certifying the brain-stem death are:

- The cause of irreversible brain-stem damage (either from a period of hypoxia, trauma, illness or toxic insult) producing non-responsive coma, must be clearly established. Following reversible causes must be excluded:
  - Intoxication
  - Depressant drugs
  - Muscle relaxants
  - Primary hypothermia
  - Hypovolemic shock
  - Metabolic or endocrinal disturbances
- The patient must be examined by a team of doctors at least twice, with a reasonable gap of time in between (say about 6 hours or so).
- None of the doctors who participate in the diagnosis of brain-stem death should have any interest in the transplantation of an organ being removed from the cadaver.

The structural and functional damage of brain-stem may be diagnosed depending upon the following observations:

- Dilated fixed pupils, not responding to sharp changes in intensity of incident light.
- Absence of motor responses within the cranial nerve distribution on painful stimulation.
- Absence of corneal reflexes.
- Absence of vestibulo-ocular reflexes.
- Absence of gag reflex or reflex response to bronchial stimulation by a suction-catheter passed down the trachea.
- Absence of spontaneous breathing.

TRANSPLANTATION OF HUMAN ORGANS ACT

For the purpose of retrieval of human organs from the dead body for therapeutic purposes, Section 5(1) and Section 5(2) of the Transplantation of Human Organs Act come into operation. In accordance with Section 5(1) of the Act, a dead body lying in a hospital or prison and not claimed by any of the near relatives of the deceased person within 48 hours from the time of the death of the concerned person, the authority for the removal of any human organ from the dead body which so remains unclaimed may be given by the person in charge of the management or control of the hospital or prison or by an authorised employee of such hospital or prison. However, in accordance with Section 5(2) of the Act, no authority shall be given under sub-section (1) if the person empowered to give such authority has reason to believe that any near relative of the deceased person is likely to claim the dead body even though such near relative has not come forward to claim the body of the deceased person within the time specified in sub-section (1).

MEDICOLEGAL CONSIDERATIONS OF BRAIN DEATH

For legal and medical purposes, an individual who has sustained an irreversible cessation of functioning of brain, including the brain-stem, is dead. A determination of death must be made in accordance with the criteria outlined earlier.

Another aspect that deserves consideration is the criteria to be followed in switching off the heart–lung apparatus. Sustaining life by artificial maintenance of circulation and respiration inherits some legal implications. Considering the death to be a permanent and irreversible cessation of functions of the three interdependent vital systems of the body **(the 'tripod' of life)**—the nervous, the circulatory and the respiratory systems—will not help in deciding as to when the artificial aids should be stopped as these systems are functionally interlinked. It is obvious that artificial aids may be applied in the hope that natural circulation or respiration may be resumed after the 'aids' are continued for sometime. But natural respiration may not be resumed even after the use of artificial aids for considerable periods. Therefore, where lies the line of demarcation, i.e. when the artificial aids to be stopped so that the doctor may not get involved in the offence of culpable homicide not amounting to murder or one of rash and negligent act, if he has removed the 'aids' indiscriminately. In such crucial affair, the decision for permanent withdrawal of the artificial aids should preferably be taken after consultation with another doctor. Ordinarily, it is sufficient to wait for 10–15 minutes. If no evidence of spontaneous functioning of respiration/circulation is available for such a continuous period, the doctor(s) is/are justified in disconnecting the artificial aids because the serious permanent impairment of brain cells can occur with only 4–6 minutes of oxygen deprivation and total loss of function generally supervenes when the deprivation exceeds 10 minutes.

Somatic and Molecular Deaths

Two phases of death have been recognised, namely, the extinction of personality or the death of the body as a whole (soma means body) when there is cessation of vital processes of the body. This is referred to as somatic death (systemic or clinical death), which is followed by progressive disintegration of body tissues and is called as cellular or molecular death.

In the absence of circulation and respiration, different cells die their molecular deaths at different times after the somatic death. Death of the brain cells stand first that are most sensitive to oxygen deprivation and therefore usually begin to die within about 5 minutes of somatic death. Then may come the organs like lungs, liver, kidneys and heart, which need to be removed for transplantation at the earliest possible, maximally within about an hour. The striped muscles can survive for hours and tissues like hair and nails for days. **Practically speaking,** the organs like lungs, liver, kidneys and heart, etc. have to be removed for the purposes of transplantation even before occurrence of somatic death by maintaining the donor on artificial aids after declaration of the brain-stem death because any lack of oxygenated blood-supply will soon make them unsuitable for transplantation purposes.

Suspended Animation (Apparent Death)

Ordinarily, the diagnosis of death does not pose any difficulty if the observations of cessation of respiration and circulation can be made with sufficient accuracy and for a sufficient period. Obviously, one should not make such decisions hurriedly as there are numerous accounts in the literature of premature pronouncement of death. Mullan et al. (1965) described two cases of barbiturate poisoning in patients who had been certified dead but were subsequently found to be alive. Polson et al. (1985) described a case of a young woman of 23 years of age, who was found on a beach near Liverpool and was declared dead by a local doctor. The pathologist who appeared later at the scene also agreed with the view. When the body was taken to the mortuary, one of the persons noticed the flickering of an eyelid and formation of a tear. She was immediately covered with clothing and shifted to the intensive care unit, and eventually recovered fully.

Such cases emphasize the importance of examining the body carefully before death is certified. Failure to detect heart beat or respiration by auscultation must be accompanied by the demonstration of electrocardiogram and electroencephalogram. The condition where the person may appear to be dead due to the fact that the vital functions are at such a low pitch as to be minimum compatible with life is known as suspended animation or apparent death. It may be encountered under the following circumstances:

- **As a voluntary act (death trance):** Life is not incompatible with temporary suspension of heart beat. The limit of tolerance will vary with the degree of oxygenation of blood at the time of suspension, metabolic rate and body temperature, etc. Under usual conditions, longer than 3–5 minutes arrest of heart beat is irrecoverable. But condition of suspended animation is practicable, and its practice is popular amongst the yogis who can maintain their vital processes to the minimum through their physical and mental exercises and restraint.
- **In hypothermia:** Operations are being undertaken after lowering the body temperature artificially and can be extended to an hour or so without heart beat, yet normal rhythm will return on warming.
- **In bodies removed from water:** Visible respiration may be absent for some periods and doubt often may occur as to presence of life or not.
- **In newborn infants:** Infants, particularly in case of 'birth in a caul', may not show any obvious signs of life yet prompt resuscitation may bring them to life.

- **In electric shocks:** The individual may impart every appearance of death but continuous artificial respiration may be helpful in restoring life. In some jurisdictions of the United States, it is still mandatory that in such cases the resuscitative measures be continued till livor mortis becomes manifest.
- **Vagal inhibitory reflexes:** Narcotic poisoning, hanging, catalepsy, hysteria, sunstroke, concussion and severe 'syncopal attacks' of various kinds are notoriously likely to cause conditions simulating death. On all these occasions, greatest care must be exercised in order to avoid the calamity of premature certification and the resuscitative measures should be continued until definite signs of death are evident.

Mode, Manner, Mechanism and Cause of Death

Confusion often arises in appreciating these terms and their proper interpretation, particularly amongst the doctors who are in infancy in the medicolegal work. This is extremely significant as one has to declare the 'cause of death' at the end of the 'autopsy report' and even otherwise in relation to the documentary certification of death.

The Mode of Death refers to the abnormal physiological state that existed at the time of death. According to **Bichat**, there are three modes of death depending upon the system most obviously involved, irrespective of what the remote cause of death may be. These are:

- **Coma,** i.e. failure of functions of brain.
- **Syncope,** i.e. failure of functions of heart.
- **Asphyxia,** i.e. failure of respiratory system.

Gordon postulated that vital body functions depend upon availability and utilisation of oxygen by the body tissues, and tissue anoxia of any type (anoxic, anaemic, histotoxic or stagnant) finally leads to cardiac failure and death. Bishop's stand point is on mode of dying, i.e. on the three proximate causes of death, while Gordon lays stress on the pathogenesis. Thus, fundamental pathological changes though vary in degree but will be more or less uniform in all forms of death. Hence, essentially the two classifications do point to the same goal. Bishop's classification, providing a descriptive picture of the mode of dying, is useful to the lawyers and laymen for interpretation of medical evidence as to cause of death. Gordon's classification is useful to the forensic pathologist to understand the pathogenesis in different forms of death of medicolegal importance. In most cases, the mode is unhelpful in describing and understanding the underlying fundamental aetiological process. Therefore, the terms like 'cardiorespiratory failure' or 'heart attack' or 'syncope', etc. are undesirable unless further qualified by basic pathological condition.

The Manner of Death refers to the 'design'/fashion in which the cause of death came into being. If death results from some disease, the manner of death is 'natural' and if by injury, then the manner of death is 'violent'/unnatural. Violence may be accidental, suicidal or homicidal in origin, depending upon the circumstances attending the episode. The manner of death, here in India, is determined by the court after examining all aspects of the case including the evidence of the doctor and his interpretation of the findings. In the United States and some other countries where there is 'Medical Examiner System' the manner of death is also expected from the doctor after evaluating the scene of crime/incidence and the victim.

The Mechanism of Death refers to the physiological derangement or biochemical disturbance in relation to death. It includes such entities like metabolic acidosis and alkalosis, sepsis, toxaemia or paralysis, etc.

The Cause of Death: The determination of cause of death following autopsy is an interpretive exercise depending upon the sound evaluation of the anamnestic data, circumstantial evidence (in India, it is furnished by the police), morphological evidence of disease and/or injury and the results of any additional laboratory studies (if need be). **Cause of death** is the injury, disease, or combination of the two that initiates a train of physiological disturbances (brief or prolonged), resulting in the termination of an individual's life. **Immediate cause of death** is the disease or injury present at the time of death that caused person's death, whereas the **proximate cause of death** is the original natural disease process, injury, or event that led to a string of uninterrupted train of events (time interval may be spread over weeks, months, or even years), that eventually led to the individual's death. However, this link/connectivity between injury and death gets weakened or broken if in the intervening period, the individual has completely recovered from the injury or has died from an unrelated condition. Labelling cause of death as 'cardio-pulmonary arrest,' 'respiratory arrest,' or renal/hepatic failure, etc. is unacceptable. Use of such seemingly inappropriate expressions invites scrutiny (such expressions may be used in clinical settings, not in the autopsy diagnosis).

It is not unusual that the information gathered at the time of autopsy is not enough to properly list 'the cause of death'. In such a situation, the opinion can be kept pending and the same is given when results of other investigations (such as toxicology, histology and/or some other test) become available. It is, therefore, understandable that **certainty as to cause of death** depends on many variables. Some categorisation in this context may be as follows: (*i*) cases where the examination including laboratory studies reveal cause of death with a degree of 'probability nearing certainty'—the circumstances are not necessarily incompatible with life but the investigations including laboratory studies reveal no other reasonable explanation for death (e.g., advanced heart disease or poisoning deaths where the poisoning/drug demonstrates non-fatal range);

(*ii*) cases where cause of death approaches 'probability' as interpreted from the anamnestic facts, the postmortem and other findings being non-specific (e.g., deaths from electrocution and epilepsy); (*iii*) cases where neither anamnestic data nor the findings or laboratory studies help in arriving at some sufficient evidence and cause of death merely remain 'conjectural' (e.g., most anaesthetic deaths behave in this manner); and finally (*iv*) cases where cause of death remains 'undetermined' from the circumstances (highly decomposed/skeletonised body), autopsy as well as laboratory studies. However, the negative anatomical and chemical findings carry significance in dispelling allegations of injuries or poisoning that might have been alleged to have caused or played some role towards death.

Estimation of Time Since Death

Most of the books have given the heading as 'Estimation of Time of Death' but I have specifically chosen the heading as 'Estimation of Time Since Death' because the experience shows the fallibility of all the methods and commands that a reasonable range of latitude be allowed for any of the methods, whether considered individually or in concert, and the doctor should, therefore, wisely avoid making dogmatic statements regarding this duration of postmortem interval.

Fortunately, even the courts, for most practical purposes, require establishing a relatively broad time-range to surround the moment of death. It is very rarely that a more precise moment of death becomes necessary. It is further stressed that the longer the interval between the moment of death and the time of examination, the wider become these limits.

IMPORTANCE OF TIME SINCE DEATH

Determination of reasonably accurate time since death has a bearing on the issues of **'alibi'** and **'opportunity'**. If a suspect can prove that he was remote from the victim when the fatal incidence occurred and thereby his innocence may be implicit. Conversely, if it is shown that the lethal attack occurred when the suspect was known to be in the neighbourhood of the victim, he thereby had an opportunity for committing the crime. When several suspects are being sorted out, the estimation of this postmortem interval may be extremely helpful in the screening procedure to exclude some putative killers, who could not be able to approach the victim at that particular time and may help in giving more weightage towards the others whose movements/activities happened to coincide with the estimated time. Therefore, any doctor, while reporting on such issues, must be wary of relying on any single observation so as to be able to withstand intense cross-examination in the courts, many months or may be many years after the reporting. **In the civil cases also,** it may have implications. As stated in the beginning, the matters concerning transfer of estate or property may hinge upon this time of death. Other matters may revolve around insurance and compensation claims following accidents or assaults.

Before taking into account the various methods in estimating the time since death, once again it may be projected that from the medicolegal standpoint, it is unfortunate that the length of time, required to attain a particular degree or type of postmortem change, cannot be categorically furnished because the timings of onset and the rates of change are usually governed by unpredictable endogenous and exogenous factors. Nevertheless, careful observations of all the varying phenomena/sources of information, influencing the postmortem interval, can yield some reliable data within reasonable range of time.

The signs of death or changes after death or the methods of estimating time since death may traditionally be studied under the following heads: immediate, early and late.

IMMEDIATE SIGNS OF DEATH

Insensibility and loss of voluntary power are concomitants of death but may be found in cases where the death is merely apparent, a fact inviting sustained efforts at resuscitation, as has amply been stressed earlier.

Cessation of respiration and **Cessation of circulation** are the other immediate signs of death but again deserve caution as outlined under Suspended Animation (Apparent Death). If on careful auscultation by the stethoscope, heart sounds are not appreciated for a continuous period of 5–10 minutes, it is acceptable evidence of death. Difficulty may arise if the sounds are feeble or chest wall is thick or in cases of emphysema. In case of doubt, ECG will settle the issue. A flat ECG for a continuous period of 5–10 minutes is accepted as an evidence of death.

Various subsidiary tests for testing presence or absence of circulation are Magnus Ligature Test, Icard Test, Diaphanous Test and Fingernail Test. Similarly, there are some tests for determining the presence or absence of respiration, namely, Mirror Test, Feather Test and Winslow Test. All these procedures are of historical interest only and of little academic importance presently. Prolonged listening with the stethoscope over the trachea or lung fields is the time-honoured procedure.

EARLY CHANGES AFTER DEATH

Facial Pallor and Changes in the Skin

Due to stoppage of circulation after death, blood drains out of the small vessels to big ones and thereby face usually appears pale. The skin becomes lustreless, pale, ashy-white and also loses its elasticity. That is why that postmortem lacerations or incisions do not show gaping to any appreciable extent.

In cases of death associated with agonal spasm and where there has been obstruction to the venous return due to compression over the neck or in cases of traumatic asphyxia, face

remains congested, bluish-black for sometime after death. Yellow discolouration of skin due to jaundice and reddish-pink colouration due to carbon monoxide or HCN poisoning usually persist for sometime after death.

Primary Flaccidity of the Muscles

Muscles lose their tonicity and become flaccid, loose and lax. The jaw drops, limbs fall flat and limp, thorax collapses, the sphincters relax, and there may be involuntary passage of urine and faeces, though the muscles are physically capable of responding to electrical/mechanical stimuli during this phase.

Changes in the Eye

They include the following:

- **Loss of corneal reflex:** After death, there is loss of corneal and conjunctival reflexes, but this may be noted in all forms of deep insensibility, e.g., narcotic poisoning, general anaesthesia, epilepsy, etc. and therefore should be interpreted cautiously.
- **Opacity of the cornea:** The cornea loses its glistening appearance, becomes dull and opaque. The glistening appearance of cornea may get dimmed even before death, as in uraemia, cholera, narcotic poisoning, wasting diseases, etc., whereas the cornea may preserve its glistening appearance for sometime after death as in cases of death due to carbon monoxide or HCN poisoning.

  The eyelids usually close after death due to primary flaccidity of the muscle, but the flaccid muscles usually fail to produce complete occlusion and therefore where the sclera remains exposed, a film of cell-debris, mucus and dust settles steadily on each side of the cornea within a few hours, becoming reddish-brown and then occasionally almost black, to which the name **'Taches Noire De La Sclerotique'** has been given.
- **Flaccidity of the eyeball:** Intraocular tension decreases rapidly after death as it depends upon the arterial pressure. The eyeballs feel progressively softer and tend to sink into the orbital fossa. This flaccidity may easily be appreciated by simple palpation. Nicati (1894) invented an instrument to measure intraocular tension. He estimated that during life the tension could vary between 14 and 25 gm. But when the heart ceased to beat, the tension fell to about half and fell as low as eighth after about half an hour of death and was nil by 2 hours after death. Reduction in intraocular tension allows distortion of the pupillary shape by gentle palpation on the globe, which is not observed during life.
- **State of the pupils:** Though the iris responds to chemical stimulation for hours after death, the light reflex is lost as soon as the brain-stem nuclei suffer ischaemia. The iris contains a large portion of the muscular tissue, which loses its tone rapidly after death and the iris usually relaxes into a state of equilibrium, assuming a mid-dilated position, though the state may alter later on as a result of onset of rigor mortis. There may be unequal dilatation of the pupils, but this has no bearing upon the cause or manner of death.
- **Changes in the retinal vessels:** Ophthalmoscopic examination of retina provides one of the earliest positive signs of death. After death, the blood stream in the retinal vessels becomes segmented as the loss of blood pressure causes the blood stream to break up into segments. This condition of 'trucking' is considered a valuable early sign of death. The phenomenon occurs all over the body but the retina is only accessible for direct viewing.

  Wroblewski and Ellis (1970) studied retinal and corneal changes in 300 patients. 'Trucking' was exhibited in one or both the eyes in about one-third patients within an hour of death. Clouding or haziness of the cornea was observed by them at 2 hours in three-fourths of their subjects. They concluded that static segmentation was a postmortem change and on the other hand, any obvious movement in the columns of blood might be due to persistence of circulation. They opined that static fragmentation and clouding of cornea were each indicative of death within previous couple of hours.

Algor Mortis (Postmortem Cooling)

It may be stated that the first recording of body temperature for confirming the death of a person was used by Dowler (1849–1850). Presently, recording of rectal temperature is often a quite useful step in the investigation of any death occurring in suspicious circumstances, unless where the external appearances indicate the body would have cooled to the temperature of surroundings. It must also be emphasised that the usefulness of the temperature estimations resides only in the cold and temperate climates where the body loses heat so long as it is in equilibrium with the environmental temperature, whereas in tropical areas, the postmortem fall in body temperature may be minimal or even absent and in some torrid climates, the corpse may even warm up after death.

When life ends, after a short interval, the body starts losing heat. The usual temperature of a healthy adult at rest is about 98.4° F (37° C) when determined by mouth, whereas the temperature at the rectum under same conditions is about 99° F and in the axilla, about 97° F. Further, there are usually individual and daily variations up to 1–1.5° F. The temperature also shows variations during the different timings of the day. It will be less in the morning and higher in the afternoon. Exercise may also have influence upon the body temperature but this however drops to normal in about half an hour. Of all the changes that occur in a dead body, the cooling of the body to the temperature of its surroundings was the first to be used as an index of the time of death. A low body temperature is a sign of either death or hypothermia. It may be reasonable to assume that a rectal temperature of 21° C (70° F) is presumptive evidence of death or a moribund state.

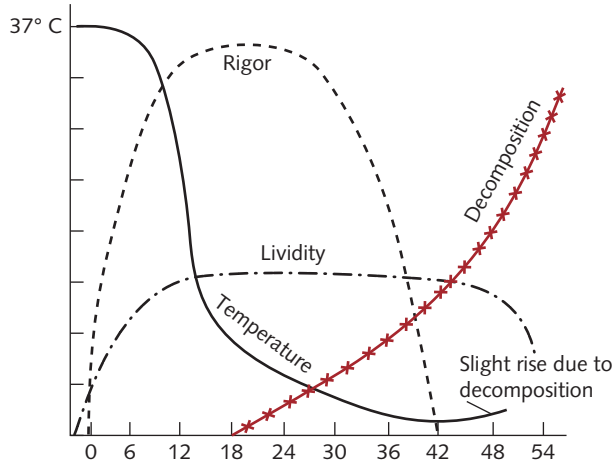


Fig. 4.1 Graph showing some factors helpful in estimation of time since death. This graph shows the approximate rate of cooling of the body after death, the approximate time of onset and progress of postmortem lividity, the approximate time of onset, duration and offset of rigor mortis, and the approximate time of onset and progress of decomposition. All these times are approximations because all these changes are subject to a number of variations.

In 1958, Fiddes and Patten worked out a formula applying repeated observations of the difference between the rectal temperature and the surroundings, assuming that the rate of cooling of the corpse would follow Newton's law. However, Marshall (1962–1974), in an extended series of studies with many coworkers showed that the body does not follow Newton's law of cooling but rather that the curve of cooling exhibited **'sigmoid shape'** with an initial plateau forming an upper flattened or slightly sloping part of the double exponential curve (Fig. 4.1). (Metabolic heat production does not cease uniformly and some heat generation continues for sometime after death due to cellular metabolism, mainly in the liver. Therefore, the skin surface may start losing heat immediately after death but the 'inner core' of the body cannot begin to cool until a 'temperature gradient' is set up and hence the rectal temperature will not show any fall for sometime after death. This is responsible for the well-known initial plateau.) This plateau is followed by a straight position of varying length and slope corresponding to the period of quickest cooling and finally the graph again flattens out into the lower shelf as the temperature differential between the body and the atmosphere approaches zero.

In cold or temperate countries where the difference between the body temperature at the time of death and environmental temperature is reasonable, hourly recording of temperature is of more value. **For temperate countries**, formula of Marshall and Hoare can be applied with some degree of satisfaction. The pre-requisites are (*i*) the atmospheric temperature should be around 60° F, (*ii*) body should better be uncovered, (*iii*) the limbs should be outstretched and (*iv*) body-built of the subjects should be taken care of. The formula provides different *rates of fall* of body temperature for corpses of different body-builts, as given in Table 4.1.

Table 4.1 Rates of Fall of Body Temperature in Subjects of Different Body Builts

Time passed since death	Thinly built subjects	Average built subjects	Fatty built subjects
Up to 3 hours	1⅓° F	1° F	5/6° F
3–6 hours	1⅔° F	2° F	1⅔° F
6–9 hours	2⅓° F	2° F	1⅓° F
9–12 hours	1⅔° F	1½° F	1⅓° F
12–15 hours	1⅓° F	1⅓° F	1⅓° F

In tropical countries, where the difference between body temperature at about the time of death and the atmospheric temperature is not high, the determination of time since death by recording temperatures is not considered to be a good criterion. Therefore in our country, Marshall and Hoare formula is not advisable to be applied except in some hilly areas where the atmospheric temperature is low (nearing 60° F). Glaister (1962) in 11th ed. of *Forensic Medicine* and K Simpson in Taylor's (1965) 12th ed. have suggested that in an average adult, the overall rate of fall of temperature in air may be 1.5° F per hour for the first few hours in temperate climates but in tropical climates, it may be approximately 0.75° F per hour. But these are broad generalisations and should be used with great circumspection.

The following formula may be used to estimate roughly, the time since death:

$$\frac{\text{Normal body temperature} - \text{Rectal temperature}}{\text{Rate of temperature fall per hour}} = \text{Approx. number of hours since death}$$

For recording the temperature of the dead bodies, the traditional method of measuring postmortem temperature is by inserting a mercury thermometer (chemical thermometer) with graduation from 0 to 50° C into the rectum. The bulb must be inserted at least 10 cm into the rectum. The reading should be recorded after sometime when it has become stabilised and it is to be recorded when the thermometer is in situation. Multiple readings should be taken at an hourly interval without withdrawing the instrument. Alternative sites may be axilla, deep nasal passage or intra-abdominal (sub-hepatic) regions. While taking the temperature of the cadaver, the temperature of the surroundings should also be recorded. Joseph AEA and Schickele E (1970) prefer to use the term **'torso cooling'** as the course of cooling varies from region to region of the same body.

Modern measuring devices include thermoelectric couple, which registers temperature accurately with least stabilising time. It may be connected to a computerised recorder, which can analyse some other sites at regular intervals.

Certain incompatibilities between morbid presentations (anatomical and clinical) warrant specific comments so as to interpret the findings documented at autopsy and the clinical course of the victim. There is no denying the fact that recent thrombotic occlusion of some major coronary artery may be unhesitantly labelled as the cause of sudden death. In this context, the severity of stenosis incompatible with the continuity of life is debatable. Davies and Popple (1979) consider that 85% stenosis is the minimum reasonably associated with sudden death. Absence of demonstrable myocardial infarction in such cases may be attributed to the brief interval between the vascular occlusion and death or the alternative blood supply provided by the effective collateral circulation. Conversely, no recent vascular occlusive lesion may be found in a person dying suddenly from stenosing coronary atherosclerosis. In this concern, Spain and Bradess noted a positive relationship between survival time and appearance of recent coronary thrombi. Only 17.5% of 80 subjects who survived less than 1 hour following the attack showed recent thrombi. On the other hand, 36.4% of 22 individuals who survived for 1–8 hours and 57% of 100 subjects who survived longer than 8 hours had recent thrombi.

APPROACHING THE CAUSE OF DEATH

Determination of cause of death may better be a multi-approach phenomenon and collective interpretation of all the findings/information including the laboratory studies, as enumerated below.

Historical Data

Presence of relatively chronic cardiovascular disease alone cannot confirm that the cardiac disease, in fact, gave rise to pathophysiological phenomenon leading to death. The common causes, i.e. pain, fear, apprehension, emotional stress, anger or the like situations leading to dysfunction or inability of the heart to respond to a demanding situation may occur without a conclusive anatomical evidence in the heart. Designation of cardiovascular disease as a primary cause of death with reasonable certainty, therefore, needs evidence confirming association between symptoms and signs of acute cardiac dysfunction and death.

In cases of concurrent heart disease and trauma, witnesses may have a key role on the basis of observation of symptoms and signs observed by them. Evaluations depending upon the statements of witnesses showing symptoms and signs of cardiac dysfunction preceding/during/immediately succeeding the episode and eventual death may finally be interpreted properly in the light of the circumstances. The final outcome, of course, rests with the court. However, many cases of sudden deaths may skip unwitnessed or the witnesses may die in the ensuing accident/trauma, etc. In such cases, the cause of death may be gathered from the presence of significant cardiovascular disease at autopsy, absence of potentially lethal trauma and other information available from the scene.

Autopsy Documentation

Autopsy documentation of findings is essential for determining the role of heart disease in fatal trauma or role of trauma in relatively expected or predictable deaths resulting from chronic disease that appears to offer no immediate threat to the life of the victim.

Needless to emphasise that a detailed and meticulous autopsy is mandatory inclusive of laboratory studies. Problem may be experienced in quantifying the amount of stenosis at autopsy because the walls of the vessels are lax and collapsed after death, which are patent during life, the patency being maintained by the intraluminal blood pressure. However, relative degree of stenosis may be observed and evaluated.

Interpretations

The goal in all these cases is to arrive at the heart disease—trauma situation as objectively as possible. After compiling and carefully examining the entire data as detailed above, the interpretations may be grouped as under:

- **Observations supporting 'cardiovascular disease' as the primary cause of death:**
  - Historical data including observations of the witnesses as presented in the 'Inquest Papers' consistent with acute cardiac dysfunction preceding death.
  - Autopsy documentation of specific potentially lethal cardiovascular disease.
  - Autopsy documentation may reveal nonfatal traumatic lesions.
  - Toxicological analysis is nil or may reveal nonfatal quantities.
- **Observations supporting 'trauma' as the primary cause of death:**
  - Historical data including observations of the witnesses not revealing any evidence of acute cardiac dysfunction.
  - Autopsy documentation reveals potentially lethal traumatic lesion.
  - Autopsy may reveal any chronic cardiovascular disease that does not seem to offer any immediate threat to life.
  - Toxicological analysis is nil or may reveal nonfatal quantities.
- **Observations supporting 'toxic data' as the primary cause of death:**
  - Historical data including statements of the witnesses not concrete.
  - Autopsy documentation of chronic heart disease that does not seem to offer any immediate threat to life.
  - Autopsy may reveal some nonfatal traumatic lesions.
  - Toxicological analysis reveals fatal amounts of the agent used.

POSTMORTEM DEMONSTRATION OF MYOCARDIAL INFARCTION

The demonstration of early myocardial infarction is of considerable forensic importance, especially in the exclusion of some unnatural cause of death.

A great number of deaths due to occlusion or stenosis may not show any evidence of myocardial infarction as detailed earlier. These deaths may result from ventricular fibrillation or damage to the conducting system.

Macroscopic Appearances

Authorities differ in their opinions as to time of occurrence and progression of changes in the infarct with the passage of time. However, general broad criteria may be as follows.

- **For the first 12–24 hours:** It is generally impossible to detect myocardial infarction with the naked eye within first 12 hours after the coronary occlusion. By 24 hours, the characteristic pallor area of infarction with the swelling of the area may become apparent because the swollen fibres squeeze the blood from the vessels lying among them. The bundles of the muscles seem separated and on cutting the ventricle at the autopsy table, the affected muscle shows more coarsely fibrillar arrangement than the normal area.
- **From about the end of first day and progressing through second and third day:** The colour usually changes to brownish-purple that progresses through a reddish blush until the muscle becomes necrotic and assumes yellowish appearance at about 24–48 hours after the occlusion. An alternative stage may sometimes appear after 24 hours, called the **'tigroid appearance'** depicting alternate indistinct bands of red and pale areas. The fully developed infarct is yellowish as the blood vessels in the area of infarction also undergo necrosis and haemoglobin, which is thus liberated, diffuses into the surrounding necrotic muscle, giving it a reddish or greenish hue. This is a patchy process and therefore the area may show appearance of small punctate haemorrhages. Associated with these changes in the infarcted muscle, acute inflammatory reaction occurs in the neighbouring surviving muscle so that the infarct is surrounded by a zone of hyperaemia.
- **3–10 days:** Progressive lysis and removal of the dead muscle leads to softening and thinning of the area of infarction, this process being at its peak about the 10th day. Rupture usually occurs at this stage.
- **Beyond 10 days:** During this time, the process of fibrous repair dominates, and the infarct is slowly converted into a fibrous scar. Fibrosis of the tissue is apparent to the naked eye after about 2 weeks, progressing ultimately into a fully established dense white fibrous scar. However, it is not uncommon to see an area of muscle showing a mixture of changes, e.g. small white fibrous scars set in a zone of yellow infarcted muscle.

Microscopic Appearances

Conventional formalin-fixed paraffin-embedded sections offer considerable assistance in the detection of the infarction, though postmortem autolysis may make the picture complicated. The following features are usually observed in sections stained by haematoxylin and eosin.

One of the earliest changes is the eosinophilia of the muscle cytoplasm, which begins to occur some 6 hours after the onset of infarction and is best seen in the lightly stained specimens. Eosinophilia or hyperchromasia can be accentuated by placing a green filter in the light path of microscope. When looked at with ultraviolet light, such fibres usually 'autofluoresce' as yellow against greenish background. Side by side appears the swelling of the muscle fibres and the granularity of the cytoplasm. The former causes the reduction in the normal inter-cellular spaces due to oedema of the cells. Later on, the cell outline becomes indistinct, the stage therefore may be called as blurring of the cell membranes. (Loss of integrity of the sarcolemma leads to release of intracellular proteins such as myoglobin, lactic dehydrogenase and creatine kinase from the myocytes into the extracellular space. Ion gradients are also dissipated, tissue potassium decreases as the contents of sodium chloride increase.)

By 24 hours, the myocytes are deeply eosinophilic and show the characteristic change of coagulation necrosis. However, it takes several days for the nucleus to disappear totally. Time of appearance of polymorphonuclear leukocytes is variable. They generally get attracted to the necrotic myocytes and reach their maximum concentration in the infarcts after 2 days.

By 2–4 days, the muscle cells become more clearly necrotic and nuclei disappear.

By 5–7 days, the acute inflammatory response has abated so that the periphery of the infarcted region shows phagocytosis of the dead muscle by macrophages. Fibroblasts begin to proliferate and collagen formation is evident.

During 1–3 weeks, collagen deposition proceeds further and the newly sprouted capillaries are progressively obliterated. Fibrosis of the tissue is apparent to the naked eye after about 2 weeks, progressing ultimately into a fully established dense white fibrous scar. (The changes described are those as observed experimentally in the dogs by inducing myocardial ischaemia and reported in the literature.)

Frozen Section Histochemistry

It may be of value; the most commonly used techniques are lactate dehydrogenase (LDH), succinic dehydrogenase (SDH) (Wachstein and Meisel, 1955; Aronsen and Pharmakis, 1962), malate dehydrogenase (MDH) and nicotinamide adenine dinucleotide diaphorase (NADD) (this was formerly called diphosphopyridine nucleotide diaphorase—DPND). Details of the standard methods for demonstrating these enzymes are given by Pearse (1972).

Sahai and Knight (1976) described a very simple fluorescent technique for demonstrating early myocardial damage. It has the advantage that frozen sections are not necessary; ordinary formalin-fixed, paraffin-processed material is satisfactory. Lie et al. (1971) reported that a basic fuchsin technique using formalin-fixed, paraffin-processed sections could be used to detect early hypoxic damage to myocardial fibres and may be expected to provide evidence of infarction as early as half to one hour after the event. Olsen (1974) has found the method reliable. Mcvie (1970) believed that measurement of the ionic ratio (K^+/Na^+) by a simple technique could be useful in the diagnosis of early myocardial infarction.

Gross examination with macroenzyme techniques has shown only the triphenyl tetrazolium chloride (TTC) method to be of some value. It is possible to highlight the necrotic area by immersion of the tissue slices in a solution of TTC. It imparts brick red colour to the intact area, i.e. noninfarcted myocardium where the dehydrogenase enzymes are preserved. Because dehydrogenase enzymes are depleted in the area of ischaemic necrosis (they leak out through the damaged cell membranes) and infarcted area is revealed as an unstained pale zone. Infarction can thus be identified at about 4 hours but the results appear to be variable.

HYPERTENSIVE HEART DISEASE

Hypertension may kill a person in a number of ways such as cerebral stroke, renal failure, ruptured aneurysm and, of course, primary heart failure. The primary heart failure due to hypertension is usually responsible for the so-called 'cardiac asthma' or 'paroxysmal nocturnal dyspnoea' produced by the massive pulmonary oedema due to hypertension.

Cardiac hypertrophy beyond acceptable weights, especially over 400 gm, is a common finding. Such hypertrophy is usually associated with severe coronary artery disease, and death appears to be due to ischaemia of the muscle. Hypertrophy of the left ventricle occurs as it has to work against the higher pressure in the systemic arteries. The muscle fibres increase in length and thickness (concentric hypertrophy). Sometimes the overall weight of the heart may be normal, yet there is a relative left ventricular thickening.

CARDIOMYOPATHIES

They represent an uncommon group of causes of sudden death. Though they are quite rare but fall in the forensic field due to the fact that the sufferer suddenly drops dead without any warning. The outstanding feature is the enlarged heart in the absence of hypertension or valvular lesion. The victims are usually young adults.

Three main types differentiated on the morphological basis are congestive cardiomyopathy, hypertrophic obstructive cardiomyopathy and obliterative cardiomyopathy. Out of these, hypertrophic obstructive cardiomyopathy is most often associated with sudden death. The blocks of tissues for histopathological examination should include from each of the lateral walls of right and left ventricles and from the interventricular septum. Isolated myocarditis as a cause of sudden death was recorded by Corby (1960).

NONATHEROSCLEROTIC CORONARY ARTERY DISEASE

There are a variety of nonatherosclerotic diseases and congenital abnormalities of the coronary arteries that can be potential causes of sudden death. Because they tend to manifest within the childhood and adolescent groups, their possibility should be entertained in case of sudden apparent natural death in subadults. Anomalies may include erratic origin of coronary arteries, abnormal location of the ostia, ostial stenosis and/or ostial ridges and acute angle take-off of the proximal position of a coronary artery, etc. **Coronary artery spasm** (Prinzmetal angina/variant angina) is typically seen as angina at rest accompanied by demonstration of ST-segment elevation on ECG with a reversible decrease in the luminal diameter of a coronary artery on angiogram. Individuals with atherosclerotic artery disease are greatly victims of this entity. Difficulty for the autopsy surgeon is that a diagnosis of coronary artery spasm cannot be approached through autopsy findings. It requires previous medical history plus clinical documentation of the spasm. **Spontaneous coronary artery dissection** is a rare cause of death. It has no known association with hypertension but tends to affect women more than men. The dissection usually affects outer third of the media or between the media and adventitia. Traumatic dissection of a coronary artery is exceptionally rare. However, when encountered, will reveal history and/or autopsy evidence of blunt impact injury involving the anterior chest wall. **Cardiac conduction system disorders** can also be cause of sudden death. Where there is gross or histologic evidence of a disease process that is also capable of affecting conduction system of the heart, the examination of the system can be of merit. Furthermore, past medical history should also encourage one to go in for such an examination. A detailed examination of the conduction system can involve processing and examining hundreds of slides from a single case. **Wolf–Parkinson–White (WPW) syndrome** is another known entity caused by an accessory pathway, or multiple pathways of conducting tissue extending between the atria and ventricles. Occasionally, accessory pathways also permit retrograde transmission of an electrical impulse from the ventricles to the atria, creating a re-entrant circuit and potentially lethal tachyarrhythmias. Autopsy diagnosis through serial sectioning and examining both atrioventricular rings is beyond practicality. All such lethal arrhythmias arising out of conduction system disorders often occur during exercise, and in particular, during swimming. Therefore, one must entertain the possibility of such disorders when a good swimmer 'drowns' for no apparent reason.

Some Other Causes of Sudden Death Having Medicolegal Significance

DISEASES OF THE CENTRAL NERVOUS SYSTEM

The heart and brain share the notoriety of organs giving rise to sudden natural death, with heart being responsible for the bigger share. Furthermore, a number of diseases traditionally considered as primary central nervous system pathologies are, practically speaking, diseases of the cardiovascular system (e.g., ruptured berry aneurysm, hypertensive intracerebral haemorrhage, etc.). Sudden death may occur under following CNS circumstances.

Meningitis

Acute meningitis is a well-known but relatively uncommon cause of sudden death. Hyperacute bacterial meningitis in the very young and the elderly can lead to death within 24 hours of onset of symptoms. Mechanisms (occurring singly or in varying combinations) leading to production of sudden death may include (*i*) cerebral oedema, (*ii*) obstruction to the flow of CSF and (*iii*) systemic collapse. Inflammatory bacterial mediators can produce a direct toxic or cytopathic effect leading to cytotoxic oedema. Additionally, bacterial toxins affecting neurons can increase metabolic demand leading to hypoxia and injury to the blood–brain barrier that may further result in vasogenic oedema. At autopsy, purulent exudates may not be apparent on gross inspection of the brain. Microscopic examination in untreated cases will usually demonstrate subarachnoid spaces infiltrated by leukocytes and bacteria. Postmortem blood cultures and/or cerebrospinal fluid cultures need be taken in such cases. A swab of the purulent meninges can be submitted for culture if cerebrospinal fluid is not obtained.

Cerebral Tumours

Although majority of tumours involving central nervous system are diagnosed clinically within time, yet occasions may be there where primary or secondary involvement of the system may become evident after an apparently sudden death. Further, detection of a tumour may at times be an incidental finding in an individual dying of some other cause. A case has been reported wherein a police officer who was shot while on duty and subsequently died had a subependymoma in the fourth ventricle. He had no known previous symptomatology. Mechanisms, acting singly or in varying combinations, through which sudden death may be caused may include (*i*) simple mass effect with increasing size, (*ii*) acute haemorrhage into the tumour causing a sudden increase in the mass effect with herniation etc., (*iii*) blockage of the ventricular system leading to acute noncommunicating hydrocephalus with rapid rise in intracranial pressure, (*iv*) compression of anatomic regions critical to cardiac or respiratory functions and (*v*) precipitation of an epileptic seizure with resultant apnoea and/or cardiac arrhythmia, etc.

A causal relationship between trauma and tumour is extremely difficult to establish. However, one may come across such cases and occasional claims be litigated. The prerequisites to the assumption that any given case of tumour was caused by trauma rests in the fulfillment of **Ewing's postulates** (James Ewing, a US pathologist, 1866–1943): (*i*) evidence of previous integrity of the injured part, (*ii*) the injury must have been of sufficient severity to produce tissue disruption, (*iii*) the tumour must have originated in the part of the body that sustained the injury, (*iv*) it must be of the histologic type that could originate from the cells that have been disrupted by the trauma and (*v*) there must be proof of reasonable time interval between injury and appearance of the tumour.

Epilepsy

Epilepsy can cause sudden death during seizure or even otherwise. Seizures limited to autonomic nervous system without motor involvement (paroxysmal autonomic dysfunction) have been held responsible for the possible mechanism of death in some epileptics found dead in bed without 'any evidence of major convulsions having occurred'. Therefore, absence of tongue-biting or faecal or urinary incontinence does not exclude the occurrence of epileptic fits.

In many deaths, mode of death may be apparent, such as asphyxia during a fit in the bed when the face may be pressed into the pillow and saliva and mucus may be observed at the mouth and nostrils. Epileptics may also suffer in other obvious ways: loss of consciousness and/or muscular control during the seizure may lead to occurrence of traumatic incidents like drowning, vehicular accident or due to results of obstruction of respiratory tract due either to aspiration of gastric contents or to the peculiar position of the individual following the attack.

Hirsch and Martin (1971) suggested that death may sometimes be related to acute disruption of brain-stem, cardiac or respiratory functional control or both as a result of an epileptic discharge and cited a number of cases where witnessed sudden deaths in epileptics had occurred without manifestations of a major seizure except for a brief tonic phase.

Autopsy should always include a search for evidence of any 'bite marks' on the tongue, examination of clothing for evidence of incontinence of urine or faeces or presence of any vomitus. Blood should always be sent for chemical analysis because withdrawal of barbiturates can also lead to convulsions and, therefore, negative results may show that a known patient had failed to follow the treatment. Examination of the brain usually fails to reveal any lesion in idiopathic epilepsy, which may enable the doctor to form an objective diagnosis of epilepsy.

Characteristic history of convulsions, ideally supported by abnormal electroencephalographic changes, is needed to base the diagnosis. However, in the absence of witnesses to the terminal episode, the autopsy diagnosis may be based upon the reliable

history and conducting complete autopsy so as to exclude any other anatomic or chemical cause of death. Any cause of post-traumatic epilepsy may also be searched in the brain.

For deaths due to rupture of berry aneurysm and intracerebral haemorrhage see discussion *Head Injury* in the Chapter "Regional Injuries".

DISEASES OF THE GASTROINTESTINAL SYSTEM

Diseases of the gastrointestinal system ordinarily do not tend to present as sudden death. However, their signs and symptoms do warrant medical attention. Avoiding or ignoring such signs and symptoms, at occasions, can create a situation wherein the death may appear to be sudden. Bleeding gastric or duodenal ulcers are one of the most common gastrointestinal diseases that may present in this manner. Another presentation leading to sudden death may be peritonitis arising from ulcer perforation. In such cases, it is wise to check the gastric antrum and the duodenum as these are the most likely sites of gastrointestinal perforation. In pre-autopsy X-ray, free air will be demonstrable in the peritoneal cavity, although it may not necessarily be located in the subdiaphragmatic region as seen in an upright antemortem abdominal X-ray.

DISEASES OF THE ENDOCRINE SYSTEM

Diseases of the endocrine system again do not tend to present as sudden natural death ordinarily. However, it is surprising that at occasions, how some individuals can simply adjust to or put up with the symptoms of acute-on-chronic endocrine imbalance and subsequently may die. Those with known endocrine disorders can die rapidly of an acute exacerbation of their disease, usually by some superadded infection or by poor compliance with treatment. In known diabetics, it is always wise to check the postmortem vitreous glucose level, even when there seems to be some other plausible cause of death. A high vitreous glucose level (>200 mg/dl or so) is likely reflective of hyperglycaemia. As reported in an analysis of >6000 vitreous fluid specimens, no nondiabetic had vitreous glucose level >200 mg/dl (because vitreous glucose levels decrease after death, a low vitreous glucose level is not considered significant in most circumstances).

PULMONARY EMBOLISM

See under chapter 'Complications of Trauma'.

VAGAL INHIBITION

Also known as vasovagal attack, reflex cardiac arrest, nervous apoplexy, instantaneous physiological death or syncope with instantaneous exitus or primary neurogenic shock. This state is characterised by sudden stoppage of heart following reflex stimulation of vagus nerve endings. There is a wide network of sensory nerve supply to the skin, pharynx, larynx, pleura, peritoneum covering the abdominal organs or extending to the spermatic cord, uterine cervix, urethra, etc. These receptor nerve endings form the afferent pathways for the reflex action and pass through the lateral tracts of spinal cord, effect the local reflex connections over the spinal segments and then travel to the vagus nucleus in the brain. The vagus nucleus has connections with sensory cerebral cortex and thalamus, besides the spinal cord, as stated. The efferent then originate from there and affect the heart through the related branches.

Such deaths occur with dramatic suddenness within seconds or at the most in a few minutes. The loss of consciousness is usually instantaneous on these occasions and death follows soon afterwards. Consequently, the mobility is negligible and the victim is likely to be found in the posture/position in which he/she was at the time of death. The condition, therefore, is characterised by fulminating circulatory failure that may be attributed either to reflex slowing/stoppage of heart, reflex vasodilatation leading to profound fall in blood pressure or a varying combination of both the mechanisms.

The victims are usually young adolescents of nervous temperament but anyone may be susceptible. The factor responsible for initiating or triggering the vasovagal phenomenon may be a minor trauma or relatively simple and harmless peripheral stimulation at the vulnerable sites upon the body as described earlier. Obviously therefore, **a variety of circumstances have been incriminated as precipitating factors, as outlined below:**

- Sudden pressure over the neck, especially over the region of carotid sinuses as may be operating in occasional cases of strangulation and hanging (carotid sinus is a dilated part of the wall of the carotid artery and contains numerous nerve endings from the glossopharyngeal nerve and communicates with the medullary cardiovascular centre and dorsal motor nucleus of vagus in the brain, related with the control of blood pressure and regulation of heart activity). Such deaths are of considerable medicolegal significance as death may ensue under the circumstances in which there had been no intention to kill. In some instances, it may be reasonable to regard such deaths as borderline between a natural and an accidental death.
- Sudden blow on the abdomen or scrotum, larynx or genital organs.
- During intubation of, or from impaction of food/some other material into the larynx.
- During minor surgical procedures involving penetration of pleura or peritoneum for tapping purposes, stretching of peritoneal sacs, dilatation of urethra or of a muscle sphincter and dilatation of the cervix in instrumental abortion.
- Sudden cerebral concussion or blow on the back of the neck.
- Sudden immersion of body in cold water. Here, vagal inhibition may act in several ways, i.e. a sudden in-rush of cold water into the nasopharynx or larynx, sudden blow of water upon the abdomen as in horizontal entry into the water with a consequent blow upon the abdomen, etc. Keatinge (1969)

found sudden rises in arterial pressure and vagal output in men exposed to ice-water showers. Bradycardia and ventricular ectopic beats have also been reported by the ECG studies in the volunteers during the first few minutes of their immersion in cold water.

- Sudden death may also be seen as occurring with intense fear, fright, emotions, from extreme unpleasant/horrible sight or smell.
- The reflex gets accentuated by a high state of emotional tension and also in many conditions that lower the voluntary cerebral control of reflex responses, like mild alcoholic intoxication, some degree of hypoxia or partial narcosis due to incomplete anaesthesia.

Autopsy

The examination of body does not disclose any typical postmortem findings by which to diagnose death due to vagal inhibition. Consequently, the diagnosis is made only on the basis of circumstantial evidence and careful exclusion of other causes of death. The availability of accurate observations by the reliable witnesses surrounding the circumstances of death is of paramount value in ascertaining the cause of death, provided that the elimination of natural disease, poisoning/chemical analysis or some other obvious cause has successfully been carried out. Occasionally, a frank admission of inability to arrive at the cause of death may be declared.

Stress and/or emotion related death: Severe emotions/physical stress can elicit powerful physiological responses and may predispose a person to a sudden and unexpected death. Explanation may reside in the concept that severe physical exertion, emotional stress, or some life-threatening situation exerts stress on the heart through the release of catecholamines. This stress may get compounded if the individual is under the influence of some sympathomimetic drug. Since heart and central nervous system are neurally linked (both parasympathetic and sympathetic nerves innervate the heart contributing to the formation of cardiac plexus), the cardiac–neural interactions can help to explain such deaths as well as those with sudden seizure disorders or subarachnoid haemorrhage. Arrhythmias may arise secondary to an overactivity of the sympathetic nervous system, or subsequent to rapid shifts between sympathetic and parasympathetic effects. It has been documented that myofibrillar degenerative changes in the cardiac myocytes occur in individuals dying of assault, but there is no clear fatal physical injury. The term 'human stress cardiomyopathy' has been applied to these histological changes. 'Somewhat delayed death', at occasions, may be explainable on the concept that the levels of catecholamines continue to increase during the first few minutes after cessation of physical activity (attainable up to 10 times higher than normal). This coupled with electrolyte imbalance (plasma potassium levels have been shown to rise during exercise and then falling rapidly minutes after the exercise) play pivotal role in inducing cardiac dysfunction. This has been termed as **post-exercise peril**. Further, medical intervention may be another factor interfering with the 'vulnerable period'.

It is possible for a person **to be scared to death through heart attack**. If the death occurs as a result of a crime, the death may be considered a homicide, even though no physical injury was inflicted. Death is usually sudden and most likely due to lethal dysrhythmia. Usually, some cardiac abnormality has been reported in such cases. However, in cases where there is no demonstrable cardiac pathology, one may also consider acute vasospasm of a coronary artery, precipitating dysrhythmia.

Sudden Death in Infancy

This has been given various names but presently most commonly accepted is 'Sudden Infant Death Syndrome' or 'SIDS'. It is known as Cot Death in Britain and Crib Death in North America. The definition put forward by Beckwith is generally followed, "The sudden death of any infant or young child that is unexpected by history and in whom a thorough necropsy fails to demonstrate an adequate cause of death".

INCIDENCE

It is estimated that incidence of death from sudden infant death syndrome is about 1.8 per 1000 live births in the United Kingdom; 90% of all infants who die being less than 8 months. The syndrome is also recognised in other countries. Rates in the range of 1.5–3 per 1000 live births are quoted in countries like New Zealand, USA, Ireland and Canada. SIDS is now most common cause of infant mortality in the first 12 months of life in the Western countries.

Identified risk factors include the following:

- Winter season: Higher rate of incidence in the winter season.
- Respiratory disease: Deaths occurring particularly in the regional occurrence of respiratory disease.
- Male child: SIDS has a male predominance.
- Higher incidence in cities than in rural areas.
- A characteristic age distribution with three-quarters of the cases between 4 and 6 months.
- An increased incidence in twins, in babies of low birthweight, among off-springs of young mothers.
- Poor living conditions.
- Bottle-fed babies.
- Most fatalities seem to occur during the night, the babies being found dead in the morning, the child being previously healthy or only mildly unwell.

PATHOPHYSIOLOGY

There are multiple theories, but each provides the possible cause in a proportion of infants. The older concepts of

'overlaying' or 'suffocation' or ' inhalation of vomitus' usually account for small number of unexpected deaths and have been discredited.

- Prolonged sleep apnoea associated with age, low birth weight and infections may be the cause in some cases.
- It has been observed that human infants who are given cow's milk at the first feed choke more frequently than those given human milk, but this observation is absent in older babies.
- Airway obstruction from the blocked nose may operate in some cases.
- Respiratory viruses found in about 25% of cot deaths may cause a rapidly fatal infection or trigger sudden apnoea.
- The findings of most studies in England support the view that sudden infant death is essentially a mode of death in children suffering from some occult illness.

AUTOPSY

Detailed history and examination of the scene may be essential to exclude child abuse or an 'accidental death'. Birth weight and full measurements must be taken. Gross examination should be meticulous including all the orifices. Swabs need to be collected from air passages, and blood from heart may be preserved for microbiological culture. Complete histology studies should be carried out. Middle ears should be opened and swab obtained. A separate piece of lung may be sent for virological culture. Petechial haemorrhages on the visceral pleura, epicardium and thymus may be seen in a few cases but may also be agonal in nature from terminal respiratory efforts against the obstructive airways. The findings of gastric contents in the air passages may not be used as a cause of death in itself, which may again be agonal or actually postmortem in origin. It should be evaluated in conjunction with the other attending circumstances.

6 CHAPTER

Asphyxial Deaths

After going through this chapter, the reader will be able to describe: Asphyxia and its types | Asphyxial stigmata | Suffocation and its types | Medicolegal aspects of carbon monoxide and carbon dioxide | Medicolegal aspects of smothering, gagging, choking, traumatic asphyxia, burking, etc. | Mechanism of death by compression of the neck | Types, cause of death, autopsy findings and the circumstances of hanging | Types, cause of death, autopsy findings and circumstances of strangulation | Mugging, garroting, *bansdola*, palmar strangulation, etc. | Types, mechanism of death, cause of death, pathophysiology, and diagnosis of death in drowning | Floatation of body in water | Circumstances of drowning

The term **asphyxia** commonly means 'lack of oxygen'. However, etymologically, the term has been translated from the original Greek, implying 'pulselessness/absence of pulsation'. How the lack/absence of oxygen is related to pulsation may be explainable on the fact that the air (pneuma) necessary for maintaining life is carried through the blood (i.e., through the oxy-Hb) and therefore, this movement of air obviously will come to a standstill when movement of blood ceases, i.e. pulselessness occurs. Hence, failure or interruption of one function is inevitably linked to the other.

Hypoxia is a general term referring to inadequate supply of oxygen to the tissues or an impairment of the cellular utilisation of oxygen for any reason, whereas **hypoxaemia** refers only to decreased carriage of oxygen in the arterial blood.

The term **anoxia** implies 'absence of oxygen' and is often incorrectly used to indicate any condition characterised by defective or insufficient oxidation of the body tissues. Barcroft (1920) using this term, divided the situation into three groups:

Anoxic anoxia, i.e. prevention of oxygen from reaching the lungs.

Anaemic anoxia, i.e. inability of blood to carry sufficient oxygen due to low haemoglobin contents.

Stagnant anoxia, i.e. where the circulation of blood is impaired so that there is lack of oxygenated blood transport to the tissues.

Later on, a fourth group called the **histotoxic** was added (Peters and Van Slyke, 1931). In the histotoxic anoxia, oxygen—although freely available in the bloodstream—cannot be utilised by the tissues. It can further be subdivided into:

Extracellular, i.e. tissue oxygen enzyme system is poisoned. Classic example is cyanide poisoning, in which the cytochrome-oxidase system is interfered with. The effects of most of hypnotic and anaesthetic drugs may also be included in this because they depress cellular enzyme activity.

Pericellular, i.e. oxygen cannot gain access to the cell because of the decrease in the cell membrane permeability that may be seen in lipid soluble anaesthetic agents like halogenated hydrocarbons, e.g. chloroform, halothane, etc.

Substrate, i.e. there is inadequate food for efficient metabolism by the cell.

Metabolite histotoxic hypoxia, i.e. the end products of cellular respiration cannot be removed thereby preventing further metabolism as in uraemia or carbon dioxide poisoning.

Adelson defined asphyxia as, "the physiologic and chemical state in a living organism in which acute lack of oxygen available for cell metabolism is associated with inability to eliminate excess of carbon dioxide". Even in some textbooks on physiology, the definition of asphyxia is extended to these two elements, i.e.:

Hypoxia implying inadequate supply of oxygen to the tissues, and hypercapnoea implying an increase in the carbon dioxide tension in the blood and tissues. (The normal levels of oxygen in the arterial blood (PO_2) with a 95% saturation of haemoglobin range from 90 to 100 mmHg at the age of 30 years to 65–80 mmHg at 60 years or more.) Reduction to 60 mmHg results in hypoxia even though the haemoglobin is 90% saturated; 40 mmHg represents severe hypoxia and death might be expected when the level falls to 20 mmHg [Eastham, RD (1971), *Biochemical Values in Clinical Medicine,* 4th ed., Bristol: John Wright and Sons].

However, **from the medicolegal point of view**, it may be useful to categorise the asphyxia into two broad groups—mechanical and non-mechanical. **Mechanical asphyxia** may

be taken to mean that the flow of air into the body is interfered through some physical impediments. It may be considered depending upon the location of respiratory blockage (Fig. 6.1 and Flowchart 6.1):

- Pressure upon the exterior of the neck as in cases of hanging, strangulation, etc. Although such deaths are not predominantly asphyxial, as will be revealed later while discussing these individually, yet they may be considered under this entity of mechanical asphyxia.
- Obstruction of the airways from the exterior, i.e. when the mouth and/or nose, is/are obstructed through some means as in cases of suffocation, smothering, etc.
- Obstruction of the airways from the interior, i.e. when the obstruction happens to occur in the internal respiratory passage as in cases of gagging, choking, etc.
- Pressure upon the chest leading to a sort of mechanical fixation of chest sufficient to prevent adequate respiratory movements as in cases of traumatic asphyxia.
- Submersion deaths may be viewed as a complex form of mechanical asphyxia, as the mechanism of death involves an asphyxial element due to occlusion of air passages by fluid as well as some biochemical changes in the blood.

Non-mechanical asphyxia may be taken to mean physiological impediments/disturbances where there occurs exclusion of oxygen by its depletion and replacement by another gas or by chemical interference with its uptake and utilisation by the body itself or where there is insufficient oxygen in the atmosphere itself. Examples may be carbon monoxide poisoning, cyanide poisoning, etc. (Flowchart 6.1).

Asphyxial Stigmata

The traditionally preached signs of asphyxia are due to pathological changes resulting from lack of oxygenation of tissues, i.e. hypoxia. The effect of hypoxia on tissues is mainly two-fold,

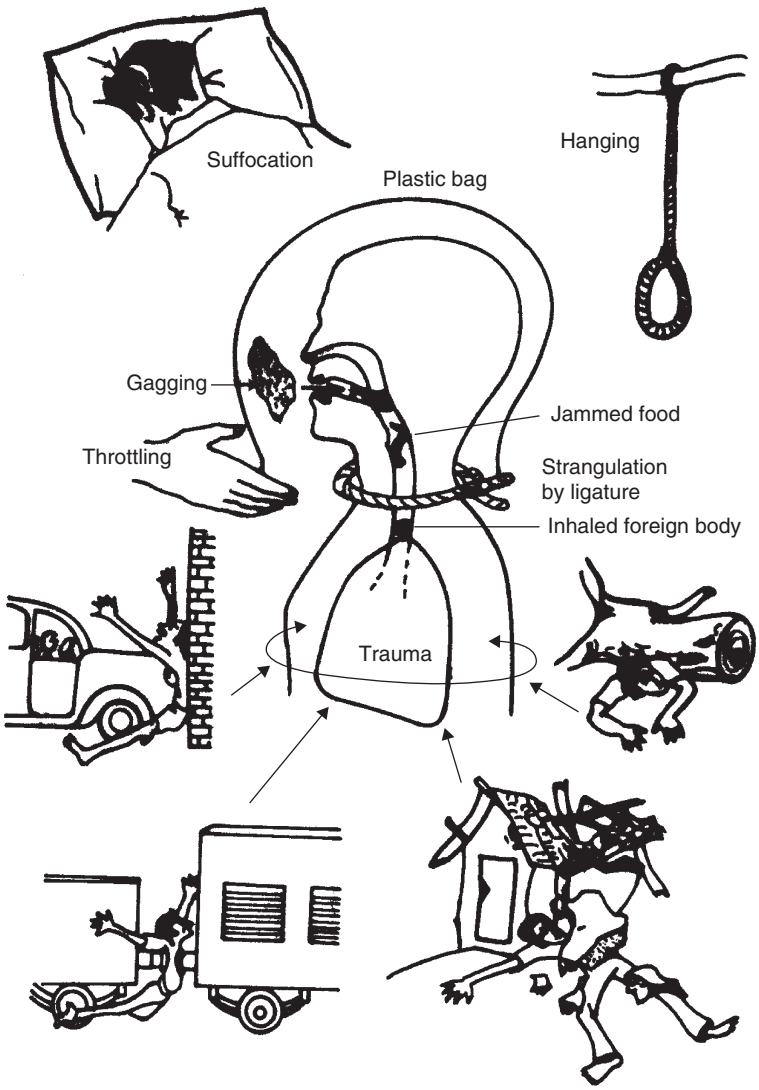
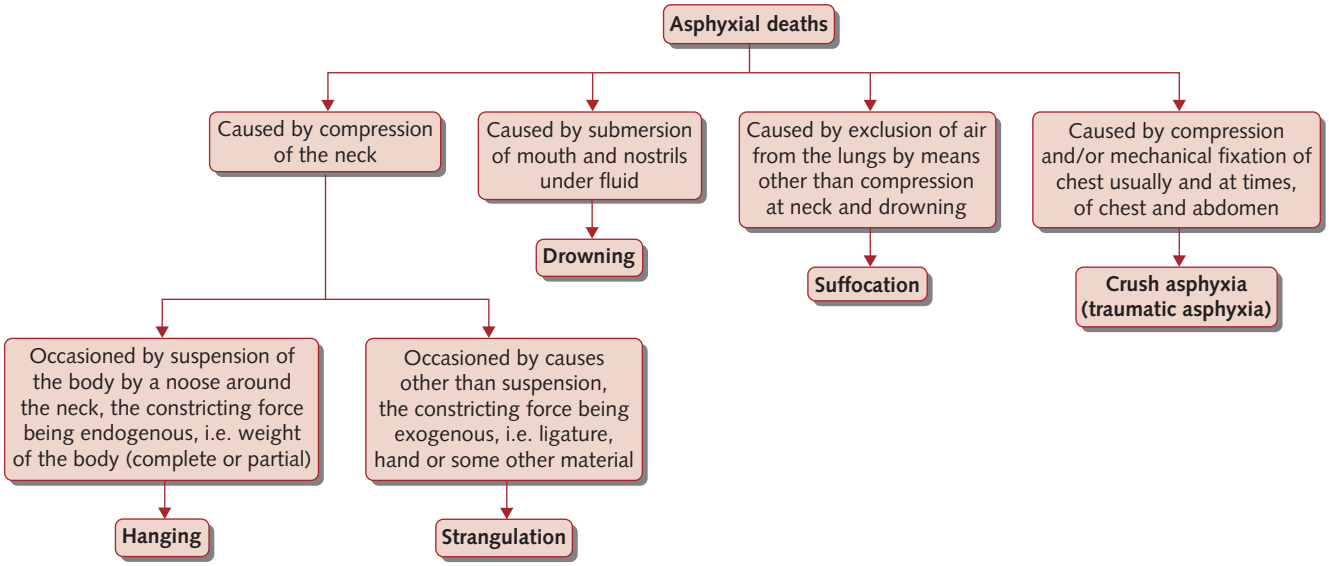


Fig. 6.1 Diagrammatic representation of the mechanism of some asphyxial deaths of common occurrence in the medicolegal field.



Flowchart 6.1 Death associated with mechanical and non-mechanical asphyxia.

viz., nonspecific and specific. The nonspecific effect eventually leads to development of parenchymatous degeneration of tissues. The specific effect leads to development of changes (the so-called 'asphyxial stigmata') like cyanosis, increased capillary permeability, petechial haemorrhages, etc. These may be taken to serve as a warning towards death due to mechanical asphyxia (Flowchart 6.2).

Suffocation

Suffocation literally means '**to die as a result of not being able to breathe or to have difficulty in breathing, etc.'**. Therefore, it may be regarded as a general term, indicating that form of asphyxial death which is caused by deprivation of oxygen by any of the following means (Fig. 6.1):

- By lack of oxygen in the atmosphere, the so-called environmental suffocation.

OR

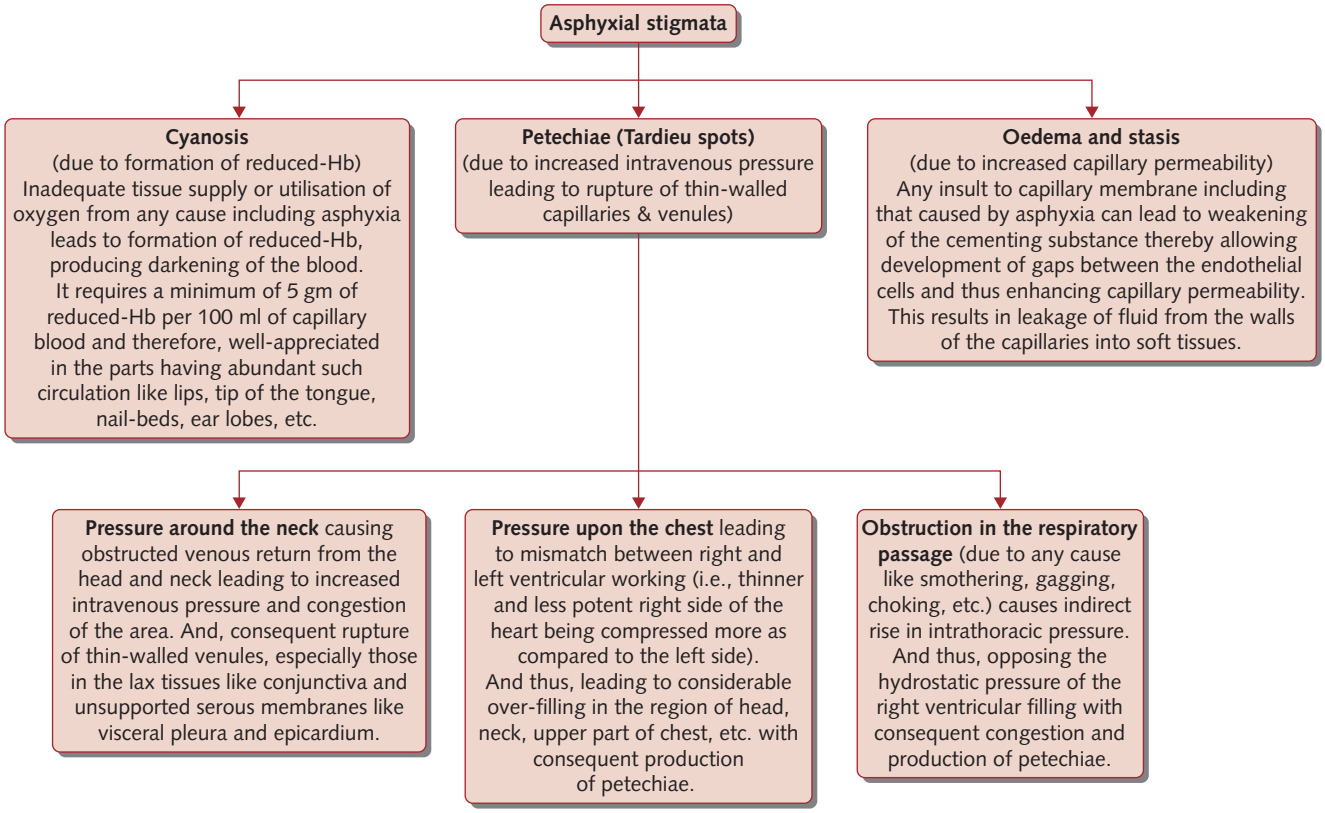
- From obstruction of the air passages by means other than compression of the neck and drowning.

Lack of oxygen in the atmosphere may occur in a variety of ways:

- Commonly by physical replacement of oxygen by other gases or by chemical changes such as combustion. Examples may be the presence of toxic gases in the environment such as carbon monoxide, cyanide, carbon dioxide, etc.
- Decompression such as cabin-failure of aircraft at high altitudes leading to sudden fall in the partial pressure of oxygen and hence reduced penetration through the alveolar walls.
- In domestic circumstances, death may ensue due to accumulation of carbon monoxide in the room where heating apparatus with burning of coals has been left burning inadvertently or otherwise throughout the night, especially when the room is lacking adequate ventilation or when a gas cylinder is left open/incompletely closed.
- Children may get suffocated when they become locked in old disused refrigerators or when they hide themselves into some box/trunk during play.
- It may also be seen in tanks of the ship or other industrial metal chambers where oxygen is replaced by nitrogen.
- Lastly, there may occur circumstances associated with high altitudes. Undoubtedly, the most important environmental factor at high altitudes, from the point of view of its biological effects, is the decreased oxygen tension of atmospheric air. Other variables may include barometric pressure, wind velocity, changes in temperature during the day (which are much greater at high altitudes), lower relative humidity and greater ultraviolet and cosmic radiations, etc. An oxygen concentration of 16% or less is considered to be dangerous. It is more or less at an altitude of 3000 m (9840 ft) above the sea level where statistically significant physiologic and anatomic differences are usually observed. Studies carried out during climbs of Mount Everest have proved that after some training, man can function without resorting to supplementary oxygen up to 8600 m (28,200 ft). Anatomophysiologic studies suggest that at more than 4750 m above sea level the possibilities of achieving a stable homeostatic balance are increasingly reduced.

Carbon Monoxide

Carbon monoxide is a major and ubiquitous component of fire atmospheres. It is colourless, tasteless, nonirritative, inodorous gas, which is lighter than air. Various sources include



The above stigmata are in no way peculiar to asphyxial mode of death. Visceral petechiae may be found after death from causes other than respiratory obstruction, especially when the death has been associated with convulsions or congestive heart failure. Hence, before they can be interpreted as originating from mechanical asphyxia, other characteristics like evidence of mechanical interference with respiration and local amplification of the hypoxic signs depending on the location of respiratory blockage/interference must be searched. [**Tardieu**, a Parisian Professor (1866), stressed that the petechiae in the thorax are often restricted to the visceral pleura, especially in the interlobar fissures and around the hilum. He described these spots in the bodies of infants who he claimed had been overlain.]

Flowchart 6.2 Asphyxial stigmata.

inefficient combustion of coal, exhaust gas of the automobiles, fuel gas and explosion gas. Being completely miscible with air, it is likely to readily spread all over by ordinary air movements. It is a highly poisonous gas, which is absorbed through lungs and avidly combines with haemoglobin to form a stable compound, known as carboxyhaemoglobin (COHb) and thus producing 'anaemic hypoxia'. Its high affinity for Hb (about 250 times than that of oxygen) is primarily instrumental for its toxic effects. An additive factor influencing the toxicity of CO is the fact that the presence of COHb causes a shift to the left of the oxygen–haemoglobin dissociation curve. As a consequence, there is increased bondage of Hb with oxygen and thus at any given PO_2, the release of oxygen will be reduced as compared with conditions where COHb is not present. When death is solely from CO poisoning, the COHb concentrations, stated to be compatible with death from acute carbon monoxide poisoning, are usually in the range of 50–60%. In interpreting lower concentrations of COHb in fatal cases, it should be remembered that hypoxia may enhance the toxicity of CO and that the CO may be an interactive factor in the presence of other toxic substances and physical and thermal trauma. Patterns of COHb found in differing fires may reflect the circumstances of an individual specific fire. Exposure time and exposure concentration, however, remain the major factors affecting degree of COHb concentration in an individual. Alcohol, barbiturates and many other drugs are other factors that potentiate the toxic effects of CO. For example, a CO saturation of around 40%, from which a healthy victim would probably recover after temporary incapacity, may prove fatal in the presence of a blood alcohol concentration of about 0.2%.

Where absorption is slow and life persists longer, autopsy samples may show more than 50–60% saturation. The variation in the fatal concentrations is wide and irregular. This may be exemplified from the fact that when two or more victims die in the same environment, the COHb concentrations of the bodies may be totally different, even in the persons of the same age group carrying similar physical health.

MEDICOLEGAL CONSIDERATIONS

Poisoning by CO is mostly accidental. The decrepit, the diseased, the drugged and the drunk are more often involved in

accidental poisoning. Accidents may occur in connection with inefficient combustion of coal/wood in ill-ventilated room(s), leaky gas pipes and taps in the dwellings and motor car exhausts in small garages. Many deaths in house conflagrations are caused by inhalation of smoke rather than by burns. These fatalities are largely occasioned by CO poisoning, though other lethal gases like cyanide, phosgene, etc. may be partly responsible. Some **victims of house fires may die remote from the flames and may be overcome in different rooms or even on different floors**. This may be explained on the basis of ability of CO to percolate considerable distances. Further, as the gas is lighter than air, it is likely to be present in significant quantities in the upper reaches in the vicinity of a fire. That is why it is always advisable to crawl on the floors to get out of the scene than to walk or run.

Suicidal poisoning by CO frequently occurs in Western countries. The deliberate introduction of fumes into the interior of a car by means of a pipe attached to the exhaust or by running the engine in a closed garage is a common method of suicide in those countries. Accidental death can also occur in the latter circumstances if the victim is unaware of the toxic nature of the exhaust gases or fails to make adequate provisions for ventilation when working on a vehicle. In India, suicide by CO is rare.

The use of this gas for homicidal purposes is also rare. However, a murderer may turn on a gas tap when his victim is asleep in the bedroom and thus suffocate him to death.

The human foetus is particularly sensitive to CO because of several differences from the adult. **Firstly**, greater contents of haemoglobin leading to greater COHb in the foetus than the corresponding maternal blood COHb under steady conditions. **Secondly**, the partial pressure of oxygen in the foetal blood is lower as compared to the adult. **Thirdly**, the foetal oxygen–haemoglobin dissociation curve lies to the left of the adult curve, resulting in greater tissue hypoxia at equivalent COHb concentrations. It is also considered that foetal half-life of elimination of CO is longer than in the mother. Hence, acute exposure to CO concentrations that are nonlethal to the mother have been reported to be associated with foetal loss or permanent neurological sequelae in the foetus.

Forensic expert should be cautious about a couple of confusing features of CO poisoning, i.e. occasional blistering of the skin of dependent areas such as calves and buttocks that may be mistaken for burns because their rupture leaves a red, raw surface that later dries to a brown parchment-like area. They are not specific to CO toxicity and are the result of cutaneous oedema in a state of profound coma leading to immobility and impaired venous return. Second circumstance includes the tendency of the dying victim to wild, flailing movements inside the room disturbing clothing and furniture to give an impression of violent quarrel, thus creating an erroneous suspicion of murder in a death due to accidental or suicidal CO-poisoning.

AUTOPSY

The most striking appearance is imparted by the colour of the skin, especially in areas of hypostasis. The typical **'cherry-pink'** colour of the COHb is usually evident if the saturation of blood exceeds 30%. When the victim is anaemic, the colour may be faint or even absent because insufficient Hb is present to demonstrate the colour. In racially pigmented victims, the colour may obviously be masked, though it may still be appreciable on the lips, nail-beds, tongue and palms and soles. Cyanide poisoning and exposure of dead body to cold (refrigeration, etc.) may cause redness/pinkness similar to CO poisoning. This cherry-pink discolouration changes to greenish and to brownish/blackish with the progression of putrefaction. However, putrefaction exercises little effect on COHb, which is extremely stable. There is no evidence that CO is evolved during putrefactive process and may be detected in the blood several days after death from poisoning by CO by using sophisticated laboratory techniques. Autenrieth detected CO in the blood of an adult 2 months after he died from poisoning by coal gas. Laguma describes a case in which CO was detected chemically and spectroscopically in the fluid content of the pleura and abdomen of the woman whose body was exhumed 7 months after death, which occurred suddenly from poisoning by CO from a defective oven. When no blood is available at autopsy, an aqueous extract from bone marrow, lung, brain, spleen or other organs or tissues containing blood may still allow determination of COHb content by gas chromatography.

In delayed deaths, i.e. in cases who survive an acute episode of CO poisoning but die later from complications/other causes, bilateral necrosis of basal ganglia and globus pallidus has been reported. These structures are particularly vulnerable to anoxaemia due to their unique blood supply. Identical lesions have also been reported in barbiturate poisoning and in cases of marked arteriosclerosis of the vessels of the corpus striatum.

Carbon Dioxide

It is a heavy, colourless, inodorous gas and is a constituent of the atmospheric air in which it exists to an extent of 0.04%. It is given off in the process of respiration, combustion, fermentation and decomposition of animal matter. It is also evolved in the neighbourhood of lime kilns owing to decomposition of carbonates. Cases used to occur in the olden days where vagrants sleeping near the lime kilns for having warmth were sometimes asphyxiated by this gas. This gas, being heavier than air, tends to accumulate at the bottom of old wells, damp cellars, mine shafts, brewer's vats, grain pits, ship holds, etc. Therefore, if a victim is seen lying unconscious at the bottom of a well or a pit used for storing grains, an attempt should be made to discharge oxygen from an oxygen holder into the bottom of the well or pit by means of a hose that may not only revive the victim but also displace the carbon dioxide so

that others can descend to render help. Manning et al. (1981) describe the deaths of three men who descended into an open drainage pit to recover a fallen grate lid. Each victim died within minutes of his descent. Analysis of the air samples taken at various levels of the pit revealed that as one descended, there occurred decrease in oxygen level, from 20% at the top to 3% at the bottom. Carbon dioxide, however, increased from the top of the pit and showed a level of 22% at the 6 feet depth of the pit. The accepted lethal level usually ranges from 10 to 20%. Blood CO_2 content determination has no diagnostic significance, since CO_2 readily accumulates postmortem. **Of critical importance**, however, is the analysis of air sample collected from the scene for its CO_2 content.

Suffocation from obstruction to air passages by means other than compression of the neck and drowning may include the following:

- Obstruction of the airways from the exterior, i.e. when the mouth and nose are obstructed by some means, may be by some material or by the application of hands, e.g. smothering.
- Obstruction of the airways from the interior, i.e. when the obstruction happens to occur in the internal airways by some foreign material, may be food or any other material, as in gagging, choking, etc.
- Traumatic asphyxia may also be included here where pressure upon the chest leads to a sort of mechanical fixation of chest thereby preventing adequate respiratory movements.

Smothering

It may be brought about by **any circumstance that prevents breathing by obstruction of the nose and mouth**, as written earlier. Smothering agent is usually fabric, pillow or hand(s). Sometimes, sand, grain, mud, flour, thick grass or vegetation may be responsible for blocking the air passages. Death in such cases may occur either by occluding substance pressing over the nose and mouth or by the passive weight of the head pressing the nose and mouth into the occlusion.

SUICIDE, ACCIDENT OR HOMICIDE

Suicide by Smothering

It is possible by burying the face in the mattress or by lying against the bed clothing so as to obstruct the nose and mouth, particularly when under the influence of alcohol or some drug. The victims are usually mental patients or prisoners. Suicidal smothering can be effected by tying a polythene or similar bag over the head and face. In such cases, the hypoxic features may be slight. There may be few petechial haemorrhages in the eyelids. Internally, there may be found subepicardial petechiae. The circumstances in which the body is found are usually of auto-erotic activity. The process is sometimes combined with the inhalation of 'sniffing substances' such as ether, amyl nitrate, etc. The induction of partial hypoxia as by hanging or any other form of mechanical asphyxia accentuates the sexual sensations during an auto-erotic exercise. Therefore, some cases may be accidental too.

Accidental Smothering

Circumstances can vary according to the age of the subjects, as discussed below.

- **Infants:** An infant, particularly when premature, may be suffocated merely by the weight of the bed clothes covering the nose and mouth or an infant 'born in a caul' may get accidentally suffocated by an intact amniotic sac. A child may get suffocated on turning his face in the cot, face getting buried inside the bed clothing or pillow or mattress, etc.
- **Children and young adults:** Children may get accidentally suffocated while playing with plastic bags and putting them over their heads when the material gets electrically charged and stuck over the face during inhalation. An epileptic child may occasionally get suffocated because of burying the face in the pillow or bed-clothing.
- **Adults:** Accidental smothering may occur in the workers during the course of their occupation. A worker can fall and get buried into semi-solid or finely divided materials like sand, ash, cotton, mud, wool, flour, coal dust, grains, wheat or cinders, etc., when the mouth and nose will get obstructed. Adult deaths may often occur when the individual is already weak or unconscious due to drug or disease. During masochistic exercises or in auto-erotic practices, as described earlier, accidental smothering can lead to death amongst young adults.

Homicidal Smothering

To accomplish homicidal smothering in an adult, there needs to be a great physical disparity between the assailant and the victim or alternatively, the victim must be incapacitated by virtue of disease, age, drink or drug. But it is also practicable if the victim is stunned by a blow prior to the act. Usually, the mouth and nose are closed by hand(s) or clothes or the face may be pressed by a pillow or be thrust into the pillow, mud, sand, sawdust or thick grass, vegetation, etc. Homicidal smothering, however, can be effected in a normal adult in his full senses if the number of assailants is more.

AUTOPSY FINDINGS IN SMOTHERING

When hands have been used as the smothering agent, the evidence of violence is likely to include nail scratch abrasions, bruises (especially the fingertip bruises) and even lacerations of the soft parts of the face. Lips, gums and tongue may show bruising and/or lacerations. Bruising and abrasions may even spread over forehead, cheeks, lower jaw, nape of the neck, etc.,

especially when there has been struggle. Bruising on the inner aspect of lips from pressure against the teeth, with or without bruising of the gums and tongue, **is an important suggestive finding**. The areas must be examined acutely with magnifying glass and confirmed by dissection. Tissues may also be taken for microscopic examination. Occasionally, the injuries may amount to mere ruffling of the skin, which will invite microscopy for its confirmation.

When some soft material, clothing or pillow has been applied gently, there may not be any external signs of violence. Under such circumstances, medical evidence may not be able to go further than the conclusion that the death was attributed to asphyxia. Occasionally, an area of pallor in an otherwise suffused face may be demarcated that may indicate the agent responsible for causing obstruction.

Presence of sand, dust, mud, cotton wool, flour, barley grains, etc. in the mouth and nostrils is highly significant finding. Presence of such particles/matter in the deeper respiratory passage intermixed with fluid and mucus is another crucial finding. Lungs may show congestion, oedema and areas of haemorrhage and collapse with intervening emphysema. Where a struggle develops during smothering, laboured efforts to breathe against the obstructed airways may lead to congestion, cyanosis and facial and conjunctival petechiae. Blood should be examined for drugs and alcohol. Presence of any natural disease should also be taken care of.

CASE: SIGNIFICANCE OF CIRCUMSTANTIAL EVIDENCE IN DEATH DUE TO SMOTHERING

On 19.12.2007, a newly married lady aged about 29 years was found dead in bathroom. A complaint was lodged by the parents of the deceased alleging that their daughter had been killed by her husband and in-laws. The Police conducted investigations instituting Section 498A (husband or a relative of husband of a woman subjecting her to cruelty) and Section 302 (punishment for murder) of the IPC. At the scene of crime, the investigating team did not find any significant evidence suggesting homicide (the body had already been removed to the mortuary). Polygraph test of in-laws was found to be inconclusive. No poison was detected in the viscera or blood of the deceased. Histopathology report revealed congestion of viscera in general with "oedema, congestion and haemorrhage in the lungs". The postmortem report showed presence of two abrasions, viz., (*i*) abrasion with reddish brown scab of 1.0 × 0.1 cm over the bridge of nose and (*ii*) abrasion 1.0 × 0.1 cm over the inner aspect of left half of lower lip with reddish brown scab. Board of doctors who had conducted the postmortem expressed inability to furnish any definite cause of death. The case was thereby referred to another board of doctors at PGIMS, Rohtak. The opinion was, "FIR and inquest papers alleged the cause of death to be strangulation. Although features of strangulation have not been observed in the neck on postmortem examination, possibility of asphyxial death due to smothering cannot be ruled out in view of reddish brown scab covered abrasion on nose and lower lip and marked congestion without associated inflammation in internal organs reported on histopathology. These findings must be interpreted keeping in view circumstantial evidence." (Contributed by Dr. SK Dhattarwal, Professor of Forensic Medicine, PGIMS, Rohtak).

Gagging

It is caused **when some pad or any piece of cloth is thrust into the mouth**. It is usually resorted to prevent the victim from shouting for help, and death is usually not intended. Hence, sometimes victim's hands and legs may be found tied to prevent him from removing the gag and walking for help. At times, it may be homicidal, particularly when victims are infants or individuals incapacitated by alcohol or drug, old, infirm, etc.

The gag not only blocks the mouth but also prevents the entry of air through the back of throat coming through the nostrils. It soon gets moistened with saliva, mucus and oedema fluid and may also get further sucked with inspiratory gasps, thus progressively leading to complete obstruction. Therefore, death in such cases is more likely to be due to pharyngeal obstruction. The autopsy findings will depend upon the intensity of the struggle to breathe and sometimes may be negligible or absent. If the gag has been removed, mucosal bruising, abrasions or lacerations, individually or in varying combinations, may be evident on the lips, soft palate and in the pharynx. There may be traces of material in the mouth and between the teeth.

To achieve some idea whether the particular material has been employed in the process of smothering or gagging, it may be examined for presence of buccal epithelial cells. The normal saliva contains between 200 and 2000 buccal epithelial cells per mm^3 and if the material has been in contact with the mouth, these cells may be demonstrable.

CASE: DEATH OF A '*CHOWKIDAR*' BY GAGGING

On 5th February, 1999, at about 8.30 a.m., on receiving information about the alleged murder of a '*chowkidar*', the police reached the site and found the '*chowkidar*' with hands tied at the back and legs tied about the middle with some cotton cloth (Fig. 6.2). Mouth was stuffed with a brown coloured 'monkey cap' (partly inside and partly hanging outside the mouth), which was secured by a red coloured muffler going tightly around the mouth, covering the nose too. Investigations by the police revealed that the assailants probably intended to commit burglary and to accomplish the same, they had undergone this exercise of tying the limbs and gagging the victim to prevent him from shouting/crying for help. The 'Inquest Papers' showed the FIR, dated 5th February, 1999, under Section 460 IPC, i.e. all persons jointly concerned in lurking, house-trespass or house-breaking by night punishable with death



Fig. 6.2 Photograph showing crossed arms that have been tied at the back with some cotton cloth. A portion of muffler is also evident around the back of the neck.

or grievous hurt caused by one of them. The case showed following features of interest:

- Hands had been crossed and tied at the back.
- Legs tied in the middle.
- Stuffing of the mouth with a 'monkey cap', probably belonging to the victim.
- Securing of mouth and nose by a muffler, again belonging to the victim.
- One whistle was found entangled loosely in the clothing.

Choking

This term refers to the **blockage of internal upper respiratory passages by some solid/semi-solid material**. The common agents may be piece of food, lump of meat, coins, bunttas, buttons, set of false teeth, marbles, corns, etc. Of these, choking by food material deserves special mention.

Food may be drawn into the larynx, either while travelling down in the mouth in the act of swallowing or may be regurgitated from the stomach. **In the former case**, undigested food may be found in the air passages. This is usually seen in old persons and mentally disturbed persons, but can occur in any age group. A popular term **'cafe coronary'** was coined by Dr. Roger Haugen, Medical Examiner of Broward County, Florida for such impaction of food in the respiratory passage. The victim may be observed slumping over the dining table or collapsing suddenly while walking across the room after having meals, with no signs of respiratory distress. The original series of deaths involved well-nourished businessmen dying suddenly and unexpectedly in restaurants and cafes, while sitting or shortly after sitting in their chairs, as if they died of heart attack. Hence, the name 'cafe coronary'. However, autopsy usually reveals a bolus of food in the pharynx or larynx. Many cases have been reported where the victim was merely seen dead while sitting or just after sitting in a chair, the mode of death probably being cardiac arrest due to overactivity of parasympathetic nervous system through the stimulation of branches of vagus nerve supplying the laryngeal and/or pharyngeal mucosa. Fairly high level of alcohol has been found in most cases of cafe coronary deaths. The suppression of gag reflex due to alcohol or drugs makes the individual susceptible to this calamity. The latter case, i.e. regurgitation of the stomach contents into the respiratory passage, deserves a cautious interpretation. Knight (1975) found in a series that 25% of deaths from various causes revealed presence of gastric contents in the air passages.

It may be reasonable to suggest that the finding of gastric contents into the respiratory passage can be due to (*i*) inhaled vomit as a terminal event in asphyxial deaths; (*ii*) disorganised and uncoordinated muscle movements during terminal moments of life, which often result in regurgitation of stomach contents; (*iii*) intoxication and unconsciousness as a result of alcohol/drugs; (*iv*) an after-effect of head injury and (*v*) agonal or postmortem spillage, etc. Vomit inhalation, therefore, may be an incidental finding or a final common event in such cases and may not be related to the cause of death. The appropriate way to diagnose inhalation or aspiration of gastric contents is by copious lung histology when products of digestion are found in the bronchi and bronchioles.

ACCIDENTAL CHOKING

Choking is almost always accidental. Victims are mainly very young or elderly people. It may also be witnessed in children while playing and putting the materials in the mouth suddenly in the act of concealing the same. Infants may 'belch out' clotted milk after a feed and this may fall into the larynx and cause choking.

Individuals in the state of unconsciousness or under the influence of drugs or alcohol or anaesthesia or during the fit of epilepsy may inhale the vomitus and suffer choking. But the usual circumstances occur during the meals, when the food is accidentally inhaled, especially when the victim is laughing or crying or talking to someone during the meals. It may be seen in the mental hospitals where a patient may snatch food from the other and may get choked while finishing the same in haste. The circumstances of 'cafe coronary' have already been described.

SUICIDAL CHOKING

It is very rare but may be possible in cases of a determined suicide or in mental hospitals or prisoners, etc.

HOMICIDAL CHOKING

Choking is a mode of infanticide but not commonly practised. This can possibly be procured when either there is appreciable physical disparity or the victim is incapacitated by disease, drugs, drink or age.

TOLERANCE OF FOREIGN BODY IN THE AIR PASSAGES

Inhalation of foreign bodies usually causes choking and unless promptly removed may lead to death. But cases have been

reported showing that the foreign body may remain lodged in the air passages without giving much trouble. Acute respiratory distress, of course, ensues immediately but once crossed, the victim may suffer little discomfort afterwards. After sometime or after a significant latent period, the foreign body may be discovered while investigating for some trouble. Literature speaks that portions of chicken bones, pins, safety pins and even partial dentures may be lodged in the air passages for relatively long periods without causing serious trouble. A remarkable example of tolerance of foreign body in the air passage is given by Ravenel (1891), where a pin had been retained in the air passages of a patient for about 38 years and its presence was felt by him when it was dislodged during the act of violent coughing. Two inflamed, circumscribed areas, at opposite points on the posterior end of each ventricle were seen and, possibly, the pin had long been lying impacted in the larynx.

CAUSE OF DEATH IN CHOKING

Usual mechanism of death in choking is **mechanical asphyxia** due to obstruction of the respiratory passage interiorly, and the findings of death due to hypoxia may be evident. However, occasionally, the entry of foreign material may produce sudden death due to **reflex neurogenic cardiovascular failure** as detailed under 'cafe coronary'. In such cases, mechanism of death is not airway obstruction, but rather a vagal mediated event. The proposed mechanism is oesophageal distention-mediated stimulation of tenso-receptors in the wall of oesophagus, causing vagal outflow that terminates in the medulla. Here, impulse pathways overlap with those of the cardiac and respiratory pathways, producing bradycardia, dysrhythmia or bronchospasm. Additionally, vagal reflexes may also arise from the pharynx and larynx. All this helps to explain why in some cases of **choking** due to upper airway obstruction death appears to ensue quicker than might be expected from an asphyxial event alone. Another mechanism of death by choking, in occasional cases, may be asphyxia due to **laryngeal spasm**. The cases are usually those where the material is irritant in nature and only partially obstructs the laryngeal lumen and triggers laryngeal spasm. In a case reported by Gardner (1942), a young man was found dead outside his home. He was deeply cyanosed and appeared to have died of asphyxia. Some acid fluid, resembling gastric contents, was present in the air passages but the air passages were not occluded by the fluid. Death was considered to be due to laryngeal spasm. The man had returned home under the influence of drink and had vomited.

Traumatic Asphyxia

The term 'traumatic asphyxia' or 'crush asphyxia' is applied where there is **pressure upon the chest leading to a sort of mechanical fixation of chest and thereby preventing respiratory movements**. Examples include:

- Burial under the rubble of a collapsed building.
- Similarly, burial under sand, grains, coal or minerals or by falling timber or masonry in industrial accidents.
- May also happen during sexual intercourse, especially when one or both the parties are affected by drug or drink.
- When panic in a crowd leads to stampeding.
- Crushing under an automobile.
- Occasionally, it may result from indirect compression while the victim's thighs and knees are driven against the chest, the so-called 'jackknife' position.

AUTOPSY FINDINGS

The **outstanding feature** is the intense cyanosis of deep purple or purple-red colour, confined to the face, neck and shoulders up to the thoracic inlet. It may sometimes extend even lower than the clavicles. Areas of pallor may be seen at the level of the collar, folds or creases in the garments, buttons, braces, etc. The mechanism of this gross-discolouration of upper part of chest and face may be ascribed to the fact that heavy load/pressure upon the chest primarily compresses the thinner and less potent right side of the heart (which receives the blood from the head, neck and face) while the more powerful left side of the heart continues to pump blood. This leads to considerable overfilling in the region of the head, resulting in such a gross discolouration of the face and adjacent regions. Shapiro (1975) suggested that the pressure on the chest forces blood backwards into the major veins, the valveless jugular system allows the blood to be forced upwards to congest the head and face whereas the valves of the subclavian veins prevent the displacement of blood into the arms. The abnormal purplish-red colour of the skin may remain for several days. Poison and Gee ascribed it to the occurrence of haemorrhages into the corium, which is responsible for persistence of colour change in these cases.

In addition to above gross discolouration, other changes usually include congestion and haemorrhages in the conjunctivae and oedema of the conjunctivae. Face, lips and scalp may be swollen and congested. Bleeding from the ears and nostrils may be there.

Internally, lungs are usually dark, heavy and may show subpleural petechial haemorrhages. Right side of the heart and the great veins above the atria are enormously distended, as explained above. Injuries to the chest wall and even the pleural cavities, with or without fractures of the ribs, may also be encountered.

In some cases where the force moves and rolls over the chest, the veins and capillaries of head and neck may get ruptured due to sudden increase in pressure and produce numerous petechiae. Such cases may be seen in industrial and road traffic accidents.

Postural Asphyxia

It is a related condition, where **a person incapacitated by drug, disease, alcohol, etc. lies with the upper portion of the body lower than the rest of the body**. The common

example is of a drunk who slides out of bed so that his head and adjoining region hang down from the edge and the remaining body is resting at an upper level. Such an almost inverted position allows the abdominal viscera to push up the diaphragm and this, combined with the decreased respiratory movements, can cause death with prominent cyanosis, congestion and petechiae in the face and neck.

Burking

This is a particular method of homicidal smothering and traumatic asphyxiation, **named after Burke and Hare** who during the 1820s used to kill their victims by this method, to supply dead bodies to the Edinburgh Medical School for anatomical dissection purposes. The practice was carried out as follows.

The victim, usually a lonely one, either alone or away from the family, was to be invited by the assailants to their house for a drink. When the victim became tipsy, he was made to lie on the ground. Then Burke used to kneel or sit on his chest and close his mouth and nostrils by hand or towel and Hare used to pull him round the room by feet. Hare turned approver and in 'King's evidence' described how Burke used to carry out the work. [Burke and Hare, 1948, Notable British Trials, TW Roughead, 2nd ed., London.]

Death by Compression of the Neck

As described under the broad categorisation of asphyxia, death by compression of neck structures include hanging and strangulation (Fig. 6.3). Other rare circumstances may include blows upon the neck, arm-locks (mugging) and certain circumstances of accidental origin.

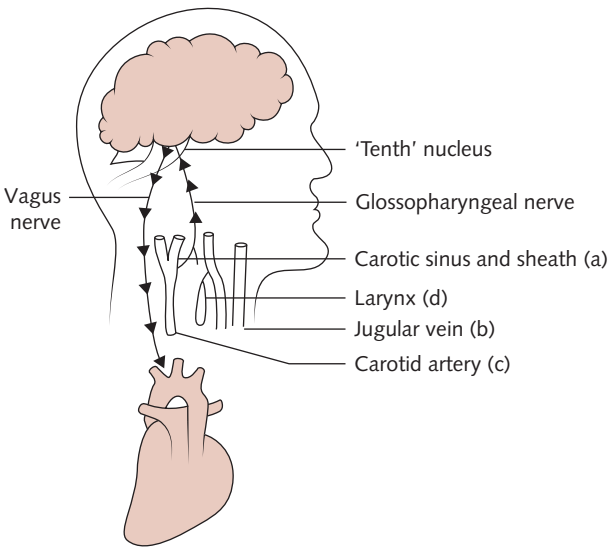


Fig. 6.3 Possible effect of pressure on neck: (a) carotid sinus reflex leading to cardiac arrest; (b) jugular venous compression leading to cyanosis and petechiae; (c) carotid artery compression leading to unconsciousness and (d) airway obstruction leading to hypoxia.

MECHANISM OF DEATH BY COMPRESSION OF THE NECK

A number of anatomical and physiological factors in varying permutations and combinations usually operate in bringing death, that is why it has repeatedly been pointed out that though considered under asphyxial deaths, yet it is not the sole element involved. The closure of the airway is not an essential element of hanging can be gathered from a case reported by Reineboth (1895)—a case of suicide by hanging in a man who had undergone tracheostomy for relief of cancer of the throat. Although he died of hanging, the ligature was above the tracheostomy. The factors are enumerated as under:

- **Occlusion of the airway:** Airway obstruction may result from the combined effects of direct compression of the larynx or trachea and upward lifting of the back of the tongue blocking the pharynx. The cartilages being soft and yielding in nature may permit a good amount of pressure but still inconclusive to result in complete closure of the airway. Brouardel calculated that a force of the order of 15 kg was required to close the trachea.
- **Occlusion of the blood vessels:** Jugular venous system lying superficial in the neck is much more susceptible than the deep seated carotid arteries. Moreover, carotid arteries are largely protected by the sternomastoid muscles and the vertebral arteries are protected by the bony canals in the transverse processes of the vertebrae. Most of the findings of asphyxia met in such deaths originate from this venous occlusion. However, under extreme pressure, as seen in hanging from long drop, even carotid vessels may get affected in which case unconsciousness ensues almost immediately and face may impart pallor look rather than congested.
- **Effects on the nerves of the neck:** Pressure on the baroreceptors situated in the carotid sinuses, carotid sheaths and carotid bodies may result in bradycardia or even total cardiac arrest mediated through parasympathetic system. The carotid sinus, it may be recalled as mentioned under **'vagal inhibition'**, is a dilated part of the wall of the carotid artery at its bifurcation that is situated at the level of the upper border of the thyroid cartilage and is supplied by numerous nerve endings from the glossopharyngeal nerve. It is concerned with the control of blood pressure and heart rate. When the area gets compressed, the impulses pass to the brain through the glossopharyngeal nerves to the vagal nucleus in the brain stem (afferent pathway) and then they return via the branches of the vagus nerve supplying the heart and other organs (efferent pathway). The effects, therefore, are brought about by the stimulation of vagus nerve supplying the heart leading to its inhibition or standstill, if the stimulation is too strong (see also in the

chapter 'Sudden and Unexpected Deaths'). Stimulation of nerve endings in the carotid sinus or adjacent carotid sheath may be effected by direct pressure from hands or ligature in strangulation or in hanging in occasional cases, or from a blow high-up on the side of the neck and death may ensue immediately without permitting enough time for the asphyxial changes to develop. Keith Simpson recorded a case in which a soldier at a dance party playfully 'tweaked' his partner's neck and was shocked to see her falling lifeless to the floor.

- **Combined effects:** As stated earlier, various anatomical and physiological factors may operate in varying combinations leading to death. Typical instance may be searched in manual strangulation (throttling) where the initial pressure for sometime may be sufficient to allow the asphyxial changes to develop but a sudden change in the grip involving the carotid apparatus may lead to death with dramatic suddenness. Therefore, the intensity of asphyxial changes may be present to any degree resting upon the circumstances of death.

Having considered these mechanisms at length, it will be easier to appreciate the varied findings while discussing deaths in cases of hanging and various forms of strangulation and the explanations need not be repeated time and again.

Hanging

Death by hanging is due to compression of the neck as a result of suspension of the body by means of a ligature in such a manner that the weight of the body (or a part of the body weight) acts as a constricting force. It is distinguished from strangulation where the neck is constricted irrespective of any effect caused by the weight of the body. This distinction is of practical importance because hanging is usually presumptive of suicide, whereas strangulation is usually homicidal (Table 6.1). Homicidal hanging or suspension of the victim after murder is quite rare.

TYPES OF HANGING (Fig. 6.4)

On the Basis of Position of the Knot

The term **'typical hanging'** is applied when the point of suspension is placed centrally over the occiput, i.e. the knot is at the nape of the neck on the back. If the point of suspension is at any other position, then the term **'atypical hanging'** is often applied. In atypical hanging, the commonest location for the knot is near the mastoid process or angle of mandible. Occasionally, it may be under the chin.

On the Basis of Degree of Suspension

The term **'complete hanging'** is often used when the feet do not touch the ground or any other material so that the body is completely suspended, the constricting force here is the weight of the entire body. For all other positions, the term **'incomplete or partial hanging'** is used, i.e. hanging in a sitting, kneeling or even lying position. Here, only a part of the body weight acts as a constricting force. It has been reported that a tension of 15 kg (33 lb) on the ligature will occlude the trachea, a tension of 2 kg (4.4 lbs) will compress the jugular veins, a tension of 45 kg will occlude the carotid arteries and a tension of 30 kg will compress the vertebral arteries.

The most common method of self-suspension involves attaching the suspending device (rope, string, sari, *chunni*, wearing apparel, etc.) to a high point such as fan or ceiling beam, etc., and the lower end may be formed into a 'fixed loop or running noose' and is placed around the neck. The victim stands on the chair/stool/table or some other support and either jumps or kicks away the support and gets suspended.

CAUSE OF DEATH IN HANGING

Hanging may lead to death by any one or varying combination of the following:

- **Injuries to the spinal cord:** These usually occur when the hanging is exercised with a long drop. Fracture dislocation of the neck was the aim of **judicial hanging** in Great Britain and is unusual in other forms of hanging. It was found in judicial hanging where a drop of 6 feet resulted in fracture dislocations at the level of 2nd and 3rd or 3rd and 4th vertebrae. Fractures of 1st and 2nd vertebrae are less common. The upper cervical cord is stretched or torn and occasionally separated from its junction with the medulla. This causes immediate unconsciousness although heart action and respiration may continue for up to 10 or 15 minutes. Congestive changes are absent.

  In judicial hanging, some writers speak of 'quick death' in some cases and others speak of 'struggled death'. It would depend upon the nature and mode of application of the ligating material plus other attending factors. A coarsely woven hemp rope may not get tightened well and the noose may bear pressure unevenly, thus allowing some cerebral perfusion to continue. In some cases, element of apprehension owing to the mere thought to execution may induce a high level of circulating catecholamines, encouraging fibrillation. It has been established that heart continues to beat for up to a few minutes following judicial hanging. Usual absence of asphyxial signs confirms that cerebral circulation is cut off rapidly by the sudden suspension of the body.

- **Vagal inhibition:** It has amply been discussed under 'Mechanism of Death by Compression of the Neck'. It should be considered as a possible cause when there are no or minimal congestive findings.
- **Mechanical constriction of the structures of the neck**, as described earlier, may be responsible for death. **Practically speaking**, combined obstructive asphyxia and interfered cerebral circulation is the most common cause of death.

Table 6.1 Differences between Hanging and Strangulation

Trait	Hanging	Strangulation by ligature
Age	More common in adolescents and young or elderly adults	No age limit as it can be effected on any age for enmity, rivalry, etc.
Nature of death	Mostly suicidal	Mostly homicidal
Face	Usually pale and petechiae rare	Congested, livid and marked with petechiae
Tongue	Swelling and protrusion is less marked	Swelling and protrusion is more marked
Bleeding	Bleeding from nose, mouth and ears not common	Bleeding from nose, mouth and ears may be found
Saliva	Often dribbles out of the mouth	Such dribbling is very rare
Neck	Stretched and elongated in fresh bodies	Not so
External signs of asphyxia	External signs of asphyxia, usually not well-marked	External signs of asphyxia well-marked (minimal, if death is due to vasovagal and carotid sinus effect)
Ligature mark	Oblique, non-continuous, placed high up in the neck between the chin and the larynx. Base of the groove or furrow being hard, yellow and parchment-like. Subcutaneous tissue underneath the mark is usually hard, white and glistening	Horizontal or transverse, continuous round the neck, low down in the neck below or across the thyroid. Base of the groove or furrow being soft and reddish. Subcutaneous tissue underneath may be ecchymosed
Abrasions and ecchymoses	(a) Abrasions and ecchymoses round about the edges of the ligature mark are rare (b) Scratches, abrasions and bruises on the face, neck and other parts of the body are usually not present	(a) Abrasions and ecchymoses round about the edges of the ligature mark are common (b) Scratches, abrasions, fingernail marks and bruises on the face, neck and other parts of the body are usually present
Injury to muscles of neck	Rare	Common
Carotid arteries	Internal coats usually ruptured in violent cases of a long drop	Damage to internal coat is rare
Larynx and trachea	Fracture of larynx and trachea—rare and that too in judicial hanging	Fracture of larynx and trachea comparatively more common
Hyoid bone	Fracture of the hyoid bone less common	Fracture of the hyoid bone is uncommon (may be common in manual strangulation, i.e. throttling)
Thyroid cartilage	Fracture is less common	Fracture is more common
Cervical vertebrae	Fracture and/or dislocation common in judicial hanging	Fracture and/or dislocation—rare
Sexual assault	Evidence of sexual assault may rarely be seen in homicidal hanging	Sometimes evidence of sexual assault

FATAL PERIOD

Usually depends upon the mechanism of death. In case of judicial hanging or hanging from a long drop resulting in injuries to the spinal cord, death is almost instantaneous, respiration ceasing abruptly though the heart may continue to beat for few minutes. The same is with the vagal inhibition, where death ensues within seconds or occasionally up to a few minutes. In other mechanisms, unconsciousness occurs almost instantaneously, although death may not ensue for few minutes. That is why it is remarked that 'once launched upon suicide by hanging, there is no retreat'. It is not preferable to go into saying, 'within so and so minutes' because the circumstances are so varied (attributable to the victim, type and nature of the ligature material and its mode of application, type of hanging—both on the basis of position of the knot and degree of suspension) that they advise to avoid giving a categorical timing.

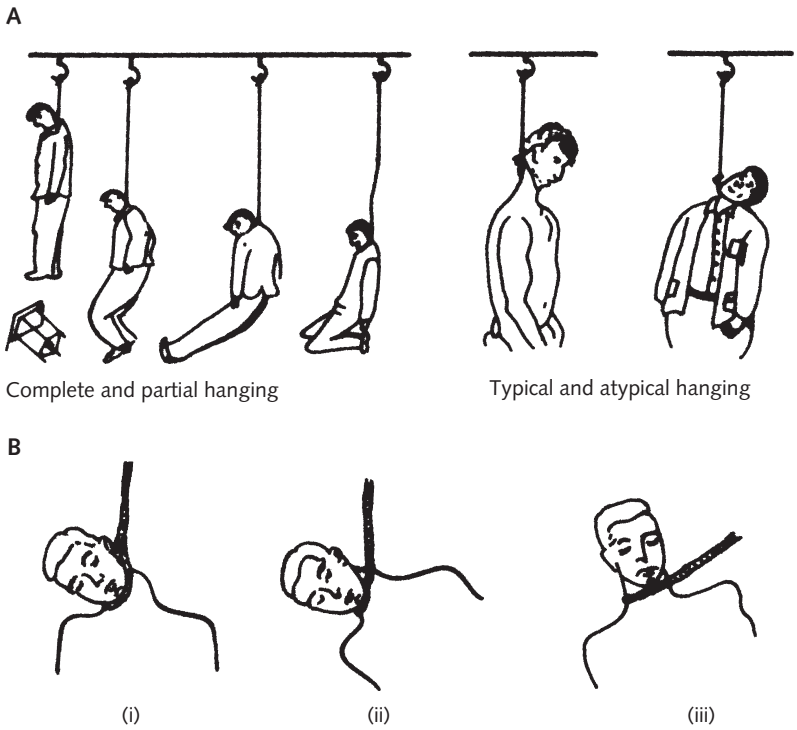


Fig. 6.4 **(A)** Types of hanging. **(B)** The position of the hanging mark on the neck: **(i)** Usual position with fixed noose and high suspension point. The mark rises high and may show a gap. **(ii)** If a slip-knot is used, the tightness of the deeply impressed loop tends to find the smallest circumference on the neck and may be lower and more horizontal. **(iii)** If the suspension point is low and the subject leans away, the mark can be horizontal.

AUTOPSY FINDINGS

These will depend upon the predominance and the combination of the mechanisms of death described above. These may be considered as external and internal and are further subdivided into general and local.

General External Findings

Face

Facial appearance may vary as per the mechanism of death, viz.:

- If death is due to vagal inhibition or injury to the spinal cord, face will appear pale.
- If due to asphyxia, the face will be flaccid and congested.
- If due to apoplexy (venous congestion), the face will be markedly congested, eyes suffused and petechiae may be noticed over forehead, face and temples.
- If the suspension is complete with feet off the ground, the pressure on the neck is such that all blood supply to and from the head is interrupted, i.e. even the deep seated carotid arteries may get occluded and therefore the face looks pale, asphyxial signs may be minimal and petechial haemorrhages are relatively uncommon.
- If suspension is incomplete (hangings may be effected from low suspension points as from door knobs, bed ports and any other easily available low securing point), it may lead to slow asphyxia, the face will be dusky-purple, congested and often swollen because the pressure upon the neck will be insufficient to occlude both the arteries as well as veins and only the veins that are lying superficial as compared to the arteries will be occluded. This obstructs the backflow of the blood from the head and makes the face dusky purple, eyes protuberant and cause petechial haemorrhages over the face, conjunctivae, forehead and temples, etc.

Eyes

May be closed or partly open or may be protruding. More often they are protruded and petechial haemorrhages may be noticed over the subconjunctival region and inner aspects of the eyelids. Etienne Martin (1950) described a state called as 'La Facies Sympathique' with right eye remaining open with dilated pupil and the left eye closed with small pupil (Lopes C, 1945, *Portugal Medical*, 29, 361). This may be due to pressure upon the cervical sympathetic, the eye on the same side remaining open and pupil dilated.

Tongue

It is usually swollen and blue and usually forced in between the teeth when the jaw is closed or the tip may be found projecting between the teeth. The protruding part of the tongue is usually

dark brown or even black due to drying. Slight haemorrhages or bloody froth may sometimes be seen at the mouth and nostrils.

Saliva

It is often found dribbling from the angle of the mouth, opposite to the side of the knot. This may be due to stimulation of salivary glands or congestive hypoxia. Salivation may not occur when the death is due to vagal inhibition or injury to the spinal cord. Evidence of dried marks of dribbling of saliva is suggestive of antemortem hanging but its absence alone will not suggest that the body was suspended after death. Moreover, it is more likely to be noticed at the scene of suspension rather than on the autopsy table.

Neck

It may be found stretched and elongated in prolonged complete suspension or in cases of long drop. It may become freely movable from side to side.

Hands

These are usually found clenched. Sometimes hands may show presence of fibres or of any other material, allegedly involved as a suspending agent.

Genitals

Engorgement of the penis usually occurs due to hypostasis with emission of semen at its tip. Similarly, the turgescence of vagina accompanied by discharge of blood-stained fluid may be noticed in females. Urine and faeces may escape due to relaxation of sphincters.

Postmortem Staining

If the suspension is in the upright position, the postmortem staining will be limited to lower half of the body including the hands, forearms and the region surrounding the genitalia. If the body has remained suspended for sometime, scattered petechial haemorrhages may be seen in the skin of the legs and in other areas of lividity resulting from excessive engorgement, i.e. they simply represent accentuation of lividity. Some blood may also be found under the body of the victim due to rupture of the engorged blood vessels and therefore may not be taken for foul play. But if the ligature is cut down and the body is made to lie on the floor within about the period during which the 'so-called' fixation of postmortem staining develops (see Hypostasis), secondary areas of hypostasis will develop in the then dependent areas, though original areas may also be appreciated on careful observation.

Cyanosis

Deep cyanosis is usually noted when the suspension is from low point and the ligature deeply set or when the ligature has broken between the knot and the point of suspension. The lips, fingertips, nail beds, tip of the nose, and ear lobules will be blue and livid in appearance.

Local External Findings

The mark on the neck is the principal external sign of hanging and therefore requires detailed description. It can almost always be distinguished from ligature strangulation unless under rare circumstances where the ligature breaks at a point between the knot and point of suspension and the victim is found in some open space under a tree or something like that.

In most hangings, fixed loop is applied where the mark appears in the form of a groove or furrow, being deepest opposite to the knot. When fresh, the groove may be considerably less conspicuous than after drying. The mark is generally yellowish or yellowish-brown shortly after death but often gets dried due to exudation of tissue fluid after sometime and assumes parchment-like consistency. The mark may show an impression of the pattern of the material used as a ligature, which is usually best appreciated by oblique lighting and the use of a magnifying glass. The knot is usually observed either on the side of the neck or in the nape of the neck. Quite rarely, it may be under the chin. The width of the groove may be equal or rather less than the width of the ligature. The mark almost never encircles the neck completely, being deficient at the nape of the neck due to hair intervening between the ligature material and the skin and the firmer nature of neck structure at the back. There may also be some gap near the site of the knot due to pull on the knot from the point of suspension above. When the knot is in contact with the skin, it usually forms an inverted 'V', the apex of the 'V' corresponding with the site of the knot. The location of the groove is nearly always above the larynx and can be traced going obliquely upwards on either side. This may be explained on the basis of the fact that when hanging takes place in vertical position, the ligature will obviously slip up until it is held by the jaw. There may be narrow haemorrhagic border, though interrupted, suggesting its antemortem nature. This was the description in a typical case of hanging. However, **there may be multiple factors influencing the appearance of the mark as may be enumerated below**. (After describing the manner of application of the ligature around the neck and the condition, type and location of the knot, the ligature material should be removed by cutting it away from the knot so as to keep the knot intact to be presented in the court later on. It should then be properly labelled, sealed and handed over to the police.)

Composition of the Ligature Material

Since most of the victims are suicides, they usually hang at the spur of moment with little premeditation, using any type of ligature, which is most readily accessible. Articles used as ligature may include rope (which may be made of cotton, jute,

coconut fibre, etc.), *dhoti*, sari, *chunni,* turban, bed sheet, cord of pyjamas or dressing gowns, belts, brace, neck tie, scarves, bootlace, towel, torn pieces of wearing apparels, etc.; these may be employed in hangings from a low point of suspension.

If the ligature material is tough and narrow, the mark is expected to be deep and prominent but if the material is soft and broad, the mark is less prominent and less deep. When a folded cloth has been used, there may be great disparity between the appearance of the neck mark and the size of ligature. When the fabric is pulled tight, certain parts of it become raised into ridges, which form the ligating surface and only these may be reproduced on the skin. When nylon, terylene or silk fabrics are used, they may leave a mark of insignificant width. Unusual ligature materials should arouse suspicion. A man, who hanged himself from a pine tree, used its roots passing over a low branch of the tree as a ligature (Gulbis, 1939). A patterned material may leave an imprint or impression upon the skin.

Mode of Application of the Ligature

Any deviation from the running noose or a noose fixed by a granny or reef knot demands a careful interpretation. Occasionally, there may be more than one turn around the neck and/or more than one knot, imparting corresponding complexity to the mark on the neck. A running noose can tighten at the time of suspension and may then produce a mark that takes a horizontal turn but it is likely to be above the thyroid cartilage.

Position of the Knot

As described earlier, the common sites for the knot are the right or left side of the neck or at the occiput. Suspension by a knot below the chin is very rare.

Course of Ligature Around the Neck

It may also make a difference. As written earlier, the loop is likely to slip up until it is held by the jaw and therefore the location of the mark is above the larynx in a vast majority of hangings. It may be at the level of the thyroid cartilage in about 15% and below the cartilage in about 5% of hangings (cited by Etinne Martin, 1950).

Period and Degree of Suspension

If the period of suspension is more, i.e. the body is discovered late in the hanging posture, the mark is likely to be prominent and parchmentised. Further, in case of total suspension, i.e. complete hanging, the ligature mark is more prominent as compared to partial hanging. It has already been pointed out that successful hangings may be accomplished from low point of suspension as from door knobs, bed posts or other easily available low securing point. Thus, the victim may kneel, sit, slump back or forward or lie prone with only the face and chest off the ground. The point of suspension in Hurphy's case (1881) was only 17 inches from the floor. The victim was a woman aged about 77 years who hanged herself by a ligature of 40 inches long attached to the leg of the kitchen table and she was found lying on her chest on the floor. When the hanging is affected from a low point of suspension, the mark on the neck may take a horizontal course, at about the level of upper border of larynx and the congestive changes are well pronounced due to lessened constriction force.

Slipping of the Ligature

If the ligature is originally fastened at a lower level and during suspension it slips up to be held under the jaw, this may prevent formation of a deeper ligature mark. On the other hand, a wider impression or chaffing or abraded area may be noticed due to such frictional displacement of the ligature material.

Weight of the Body of the Deceased

Obviously, the mark is likely to be more prominent and deep if the weight of the body is more and vice versa.

Ligature Mark may not be Evident

The ligature mark may not be evident if soft broad ligature material is used or if anything like long beard or clothing intervene between the skin and the ligature material or if the point of suspension is very low. If something, say a collar or a muffler or some ornament upon the neck or any other article, interposes between the ligature material and the skin of the neck, the ligature mark may be deficient or less prominent upon that area.

Scratches and abrasions, if present, must excite suspicion. Rare possibility of these lesions having been produced by the victim himself/herself, in an attempt to pluck at the ligature (reflex action to preserve life) could exist but however distinct abrasions, especially the crescentic nail marks, may suggest manual strangulation prior to hanging. The fact, as mentioned earlier, that the skin can be abraded during upward slipping of the ligature should also be kept in mind.

General Internal Findings

These are remarkably less in cases of hanging. Depending upon the mechanism of death, there may not be any finding as in the case of death due to vagal inhibition, or general findings of asphyxia in cases where death is due to asphyxia.

Local Internal Findings

Schrader (1940) drew attention to the artefacts likely to be encountered from seepage of blood from the neck vessels and recommended that neck should be drained of blood by removing the brain and dissecting the heart before reflecting the skin of the neck. Prinsloo and Gordon (1951) confirmed the importance

of these artefacts and recommended dissection of the neck structures in situation. The dissection of the neck may be carried out by a V-shaped incision, the apex of the V being at sternoclavicular joints.

- The subcutaneous tissue under the ligature mark is usually dry, white and glistening—more so if the body has been suspended for a long time and there has been a long drop (complete suspension). There may be petechial haemorrhages in the adjacent tissues above and below the ligature mark, especially in congestive asphyxial mode of death. There may be bruising in the subcutaneous tissue and the muscles deep to the mark, but this is by no means invariable.
- Occasionally, muscle fibres of platysma and sternomastoids may get ruptured, especially in long drops or complete hangings.
- Damage to the intima of the carotid arteries, usually around the region of the sinuses with extravasation of blood in their walls, particularly in case of a long drop, may be found.
- Gordon et al. (1982) suggested that a portion of the skin and deeper tissue in relation to the ligature mark should be examined microscopically for evidence of tissue reaction. Careful and detailed microscopic examination may reveal the presence of effusion of red cells but no evidence of tissue reaction, which takes some hours to develop.

Damage to the Hyoid Bone and Larynx

In order to appreciate the injuries to the hyoid bone and larynx, it would be much better if something is discussed about the forensic aspects of anatomy of the hyoid bone and larynx (Figs. 6.5 and 6.6).

The **hyoid bone** is almost a U-shaped structure and is composed of the central horizontal portion, i.e. the body, to which are attached 'greater horns' through a natural joint with the body. The greater horn lies behind the front part of sternomastoid, 3 cm below the angle of mandible and 1.5 cm from the midline. The greater horns, in early life, are connected to

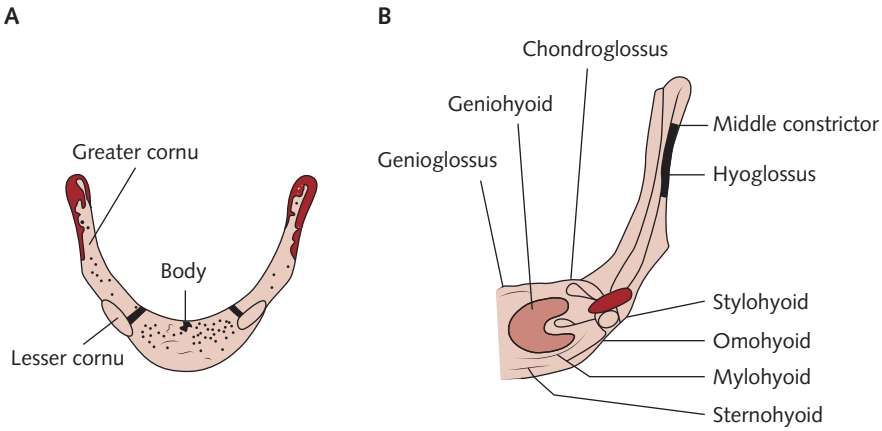


Fig. 6.5 The hyoid bone: **(A)** anterosuperior aspect; **(B)** drawing of the left half of the hyoid bone to show the muscular attachments (superior aspect).

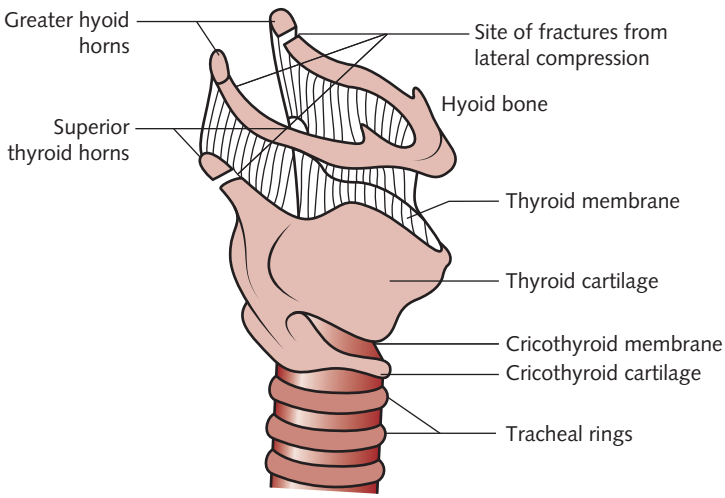


Fig. 6.6 The forensic anatomy of the larynx. The hyoid and thyroid bones fracture either from direct lateral compression or from traction from the thyroid membrane when it is compressed.

the body by cartilage but after middle life, these are usually connected to the bone. There are two 'lesser horns' situated close to the junction of the greater horns with the body that carry no forensic significance. The hardening of the bone usually is related to age but there can be considerable variations and sometimes even elderly people show only slight ossification. Body may get calcified as usual but the horns may calcify irregularly; both in space and time. Evans and Knight (1981), in a series of 110 excised hyoids, found complete fusion of the greater horn with the body in 39 subjects and partial fusion in 14. The youngest subject in whom fusion occurred was 18 years of age. At the other end of the age range, there were subjects in their eighth and ninth decades where no fusion was found.

The **larynx** occupies the front of the neck, and its position varies with age and sex, being opposite to the 3rd to 6th cervical vertebrae in adult males and somewhat higher in adult females. It is composed of nine cartilages—thyroid, cricoid, epiglottis and smaller pairs of cuneiform, corniculate and arytenoids. From forensic point of view, thyroid and cricoid are most important.

The **cricoid** is shaped like a signet-ring with the signet part at the back. Ossification occurs late and is frequently incomplete. Being less accessible and very often remaining cartilaginous, it is rarely injured and may only be injured where there is an application of considerable force with anteroposterior compression against the spine.

The **thyroid** comprises of a central shield-shaped body, which is angled forward at some 90° in males and around 120° in females. This is a susceptible structure as it lies in the front and covered merely by the skin and fascia. At the back of the body, upper and lower horns (superior and inferior horns) are attached to it. The superior horns are firmly attached to the hyoid bone through thyrohyoid ligament. It consists of a hyaline cartilage and tends to become ossified as the age advances. Ossification is generally considered to commence around 25 years, although it is quite variable and one may find a thyroid still completely cartilaginous in old age. Furthermore, the ossification varies in incidence as well as in degree, increases with age and often occurs earlier in men than in women.

The hyoid bone may get fractured or fracture-dislocated in hanging. The fractures usually involve the greater horns, which are likely to break at about the junction of their outer third and inner two-thirds. There is lack of unanimity of opinion regarding the frequency of hyoid bone fracture in hanging. Reuter (1901) reported this fracture to be relatively common, present in 60% of cases in 'typical' hanging and 30% of 'atypical' hangings. Etienne Martin (1933) had made a collection of these fractured bones and described the event as 'assez frequente' in hanging. Smith and Fiddes (1955) remarked that 'the hyoid bone is practically never injured'. The discrepancy may be due to the ages of the victims and the extent of search. The fracture is more frequent in persons over 40 years, i.e. where the hyoid bone is likely to be ankylosed. Weintraub (1961) found the hyoid bone fractured in 9 out of 33 cases. The fracture of the hyoid bone may fall under any of the following groups:

- **Anteroposterior compression**, where the distal fragment is displaced outwards, and periosteum may be torn on the inner aspect.
- **Side-wise compression**, where the distal fragment will be bent inwards, and periosteum may be torn on the outer aspect. Here, one or both the horns (cornu) may be fractured due to compression on one or both the sides, as was found by Weintraub where one horn was fractured on inner side and the other on the outer side.
- **Traction or avulsion or tug fracture.** The hyoid bone is drawn upwards and held rigid by powerful muscles attached to its upper and anterior surface (Fig. 6.6). Violent lateral or downward movements of the thyroid cartilage or the pressure between the cartilage and the hyoid bone will exercise traction through the thyrohyoid ligaments, leading to the fracture of the bone.

The fractures are usually associated with at least some haemorrhage but this is not invariable in hanging, and the absence of haemorrhage does not necessarily mean that the body was suspended after death. This absence of haemorrhage may be explained on the suggestion that the circulation may be compromised during hanging and hence the absence of haemorrhage even when the hanging is during life. Obviously, the other possibilities like postmortem origin of the fracture or fracture occurring during the autopsy procedure need to be taken care of and the findings should be appreciated in conjunction with the other injuries.

Regarding the involvement of thyroid cartilage, opinions again vary. As per Taylor's *Principles and Practice of Medical Jurisprudence* (13th ed., 1984) fractures of superior horn of the thyroid cartilage are approximately equal to fractures of the greater horn of the hyoid bone and are related to the state of ossification of these structures. In a series of 80 cases, age varying from 18 months to 81 years, Polson and Gee found the fracture of the superior horn/horns of thyroid cartilage in 37 cases (almost 50%).

The article entitled 'Analysis of Neck Injuries in Hanging' (retrospective study of 175 cases of suicidal hangings during a 5-year period) published in '*The American Journal of Forensic Medicine and Pathology* Volume 24, Number 2, June 2003 by Nikolic et al. revealed the following:

- The most frequent injuries were the muscle haemorrhages brought by direct pressure as well as indirect stretching of these structures.
- The neck blood vessel injuries were rare. When present, there was higher tendency of their occurrence to the ipsilateral side related to the location of the ligature knot suggesting their production due to traction rather than direct pressure on the blood vessel.

- Frequency of hyoid bone fracture was more in victims older than 30 years (victims varying from 10 to 87 years formed the sample of their study). Factors like age, point of suspension and width of ligature carried considerations.
- Fractures of the superior horns of the thyroid cartilage were the most frequent injuries of the solid neck structures. Factors like age (significantly more frequent in individuals older than 30 years), location of the knot, location of the ligature, length of ligature and possible swing carried considerations. It may also occur due to indirect force, i.e. caused by stretching of thyroid ligament and thyroid membrane.

WHETHER THE HANGING IS ANTEMORTEM OR POSTMORTEM

As already stressed, the absence of tissue reaction to the ligature and the lack of congestive changes may not be taken as evidence that the body was hanged after death. Circumstances in which the body was found may be of much help. A detailed autopsy supported by necessary laboratory investigations may be quite rewarding. The following points will be of help:

- The presence of any other possible cause of death or presence of injuries that could not have been self-inflicted.
- An unacceptable distribution of hypostasis, i.e. hypostasis is not in keeping with the suspension.
- Evidence from the ligature mark: The mark on the neck is the principal sign though not the conclusive one. If the mark is dried and brown in appearance, it may have been applied before or soon after death. Exercising caution from the experiments of Casper (who concluded that a mark of hanging where hanging took place during life can also be produced if the body is suspended within a couple of hours or even longer after death), the evidence/findings, etc. must be scrutinised in entirety. Gordon et al. (1982) suggested that a portion of the skin and deeper tissue in relation to the ligature mark should be examined microscopically for evidence of tissue reaction. Careful and detailed microscopic examination may reveal the presence of effusion of red cells but no evidence of tissue reaction that takes some hours to develop.
- Evidence from presence/absence of saliva: As stressed earlier, dried marks of dribbling saliva are suggestive of antemortem hanging as it is occasioned due to pressure upon the salivary glands but its absence alone will not suggest that the body was suspended after death. Moreover, it is more likely to be noticed at the scene of suspension rather than at the autopsy table. Slight haemorrhage or bloody froth is sometimes seen at the mouth and/or nostrils.

SUICIDE, ACCIDENT OR HOMICIDE

Suicidal Hanging

Hanging is ordinarily presumed to be suicidal unless the circumstantial and other evidences are strong enough to rebut the presumption. The age of the victim may be anywhere between extremes of life. Both sexes are almost equally prone. Often a suicide failing in other methods may lastly resort to hanging. In these cases, evidence of some other adopted means may be forthcoming. Fibres of the ligature material like jute fibres, etc. may be found in the clenched hands. The following points may be of help:

- Corroboration of suicidal hanging may be gathered from the facts like presence of a suicide note in the handwriting of the deceased, place of occurrence being a secluded place, easily approachable point of suspension and easily accessible ligature material usually some household articles or belongings of the victim.
- Presence of blindness or even some physical infirmity may not prevent suicidal hanging. Cases have been reported where gagging, tying or fixing the limbs, stabbing or cut-throat injuries and even the attempt by firearm injury was resorted but being unsuccessful, hanging was followed as a last resort.
- Presence of poison in the body of the deceased does not contradict suicide by hanging. In a couple of cases in the author's series, alcohol (though in nonfatal doses) was present.
- An insane person may disturb the articles in the room and may splash the blood by inflicting injuries to self and finally may hang himself or herself, the scene simulating a homicidal hanging.
- Presence of injuries upon the deceased does not always suggest homicidal hanging. The injuries might have been originated while cutting down the body or bruising over the chest might have been sustained during attempted resuscitation and occasionally minor injuries in the form of abrasions might have been self-produced during an attempt in plucking at the ligature.

Figures 6.7 and 6.8 represent two different cases of suicidal hanging, the important features have been described in the figure captions.

Accidental Hanging

Hanging can occur accidentally, while at work, during playing, exhibiting hanging exercises or showing some performances in the circus, etc. (Fig. 6.9).

- During auto-erotic masochistic exercises, erotic fantasies may be deliberately induced by partial cerebral ischaemia, achieved by some form of hypoxia or pressure upon the neck. Hypoxia may be produced by masks, pads on the face or enveloping the face; but self-suspension or incomplete strangulation is more often practised by the individual. Victims are usually adolescent males, usually found nude or wearing female clothing. They arrange a situation by placing a noose around the neck and create a state of incomplete asphyxiation in themselves by pulling the other end of the noose across some arrangement. In the course of such



Fig. 6.7 Photograph showing 'hanging body' of the victim. Note the marked hypostasis at the lower portions of the arms and legs due to prolonged hanging. Ligature used was of 'running noose type', and the other end of the rope was fixed at the fan. Stool used to achieve the requisite height was probably kicked-off and seen lying upside down. Presence of towel upon the face, one wrist being secured by some cloth with a running round and the other end of the cloth being rounded loosely upon the other wrist and presence of blood stains on the floor initially led the police to think of homicide. (Slight haemorrhage or bloody mucus may

practices, the victim can die if he fails to release the pull. At times, mirrors or cameras may be used to see the events by themselves. Nude photographs or pornographic literature may also be found at the place of occurrence. Such exercises of sexual asphyxias speak of some sexual perversion and some mental eccentricity and in most of the cases, death is accidental in origin. Mislabelling the same as suicide or occasionally homicide may have implications in insurance and inheritance, etc.

- It may occur in the individuals while at work; for example, in the factories when a worker working at a height falls off accidentally and gets hanged on a sling or rope or sometimes a labourer who carries load on this back secured by a strap may slip and cause accidental hanging or strangulation.
- During climbing ladders or railings, if one loses foothold one may fall and the clothing may get caught by a bough or spike that may be drawn across the neck.
- Children can get accidentally hanged due to slippage from the restraining apparatus, while crawling away or by clothing being tightened around the neck, etc.
- Accidental hanging can also occur when a person's neck gets compressed below the chin by getting suspended by a steering wheel of a motorcar, tail board of a lorry or cart, edge of a sofa or arm of a chair, etc.

Homicidal Hanging

It is not usual but not unknown also. A few cases have been reported in the literature; for example, six cases by Reuter (1901) and single reports by Klauer (1933), Weidemann (1940) and Mayne (1942).

Unless the victim is an infant or an adult but incapacitated by drug, disease or drink or rendered unconscious by a stunning

a short period and were removed from the drowning medium alive and gasping. If a dead body is deposited in water or when the death in water is not due to drowning, then though the diatoms may be able to reach the lungs by passive percolation but not to the distant organs because of absence of circulation. Therefore, the organs examined routinely are lung, liver, brain and bone marrow.

The detailed studies of Thomas, Van Hecke and Timperman have shown that the method is reliable provided sufficient precautions are exercised to prevent contamination at each stage in the process of demonstration of diatoms from the organs of the drowning victims. Two main points of criticism, as put forward by some critics, remain:

- Some known cases of death from drowning have shown no diatoms.
- Diatoms have been found in the organs of persons who have died from causes other than drowning (probably depending upon the fact that certain foods, notably the shell fish, contains large quantities of diatoms that can therefore be taken along with the food and reach the distant organs after penetrating the intestinal lining and gaining entrance into the portal vein tributaries or lymphatic channels).

These objections can satisfactorily be taken care of by:

- Comparing the number and variety of visceral diatoms with their number and variety present in the alleged medium of drowning.
- Taking into consideration the type of drowning, the amount of water inhaled, the season of the year and the other attending circumstances.

Method for Demonstrating Diatoms

Thomas and his associates described a technique for the detection of diatoms in tissues. 2–5 gm of the tissue or about 40 gm of the bone marrow from the shaft of a long bone or from the sternum may be taken by means of gynaecological curette. The marrow is placed in a Kjeldahl flask in which it is chemically digested by adding small quantities of concentrated nitric acid at a time. The contents are heated for about 1–2 hours. This yields a transparent yellow fluid with a supernatant disc of fat. The yellow fluid is next centrifuged. The centrifuged deposit (usually hardly visible to the naked eye) is to be poured on a slide and examined while still wet under a cover-slip.

Water from the drowning medium should always be examined for diatoms (Fig. 6.16). While collecting the sample of water from the reservoir/lake/river, etc., it is advantageous to take a fairly large volume of water (1–2 litre) and add a few drops of iodine solution to kill the micro-organisms and allow to stand overnight. Decant with care and preserve the concentrate for examination.

Comparison of number, nature and distribution of diatoms observed in the visceral organs/marrow with the number, nature and distribution of those observed in the alleged medium of submersion will be rewarding to label the death due to drowning. However, as said earlier, the type of drowning, the season of the year, the amount of water inhaled and other attending circumstances should also be taken into consideration. Some recent research in Japan claims that, using detergent or enzyme digestion instead of destructive acid, even soft-bodied algae and protozoa can be recovered from the tissues in drowning.

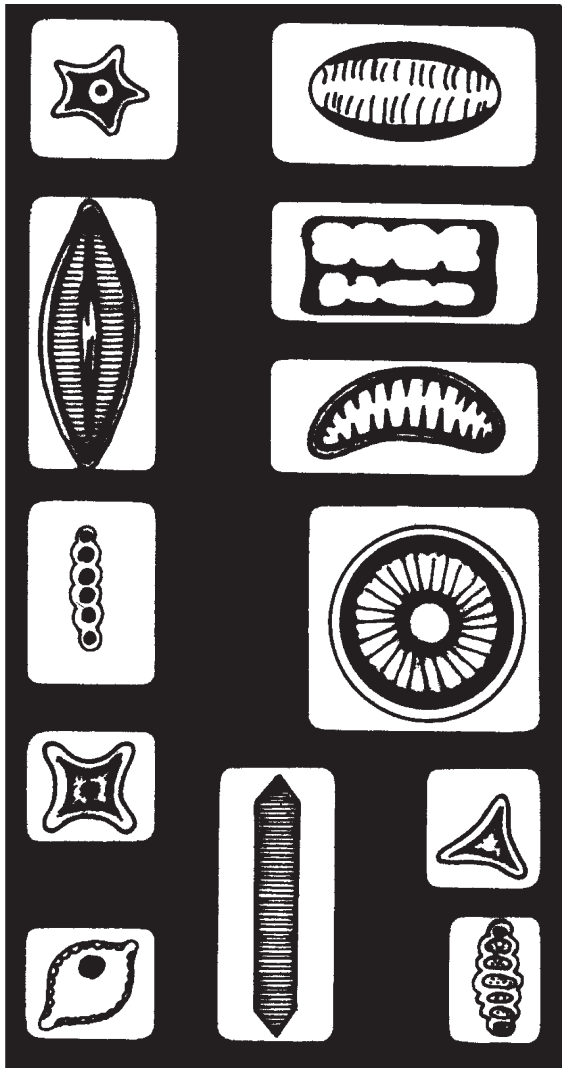


Fig. 6.16 Different types of diatoms.

R V Verrier, 1964 unequivocally throws light on the significance of diatoms: A yacht disappeared in the English channel. It was the month of January. Six weeks later, body of a man was seen on the Belgian coast. It was transferred to England and was identified by means of a surgical scar and finger prints as one of the men who had sailed with the missing yacht. Postmortem examination posed problem due to advanced decomposition. However, there was no antemortem injury upon the body and only natural disease was renal calculi. Study for the diatoms showed number of diatoms in the lungs,

liver and bone marrow of both the femora. These diatoms and silt from the organs and marrow were found to be similar to those present off the Kent coast where the missing yacht was thought to have abandoned. Hence, it is suggested that when carried after observing due precautions at each step, the diagnosis of drowning could be made even in putrefied bodies where the anatomical recognition of drowning is least possible. Further, some evidence about the site of drowning might be gathered from the ecological typing of the diatoms.

FLOATATION OF BODY IN WATER

At occasions, the issue of time interval after which a body makes its appearance on the surface of water assumes importance. The prosecution may allege some specific period that may not be sustainable by scientific data. **The specific gravity of a human body** is determined by the combined specific gravities of different parts. The only element of body that is lighter than water is fat. The specific gravity of fat is 0.92, and it is considered that in an averagely built adult, it constitutes about 5% of the weight of the body. The buoyancy of the lungs and the lightness of the fat are counterbalanced by the weight of the skeleton so that the naked human body has a tendency to sink in water. This obviously follows that women are generally of lower specific gravity than men because of smaller/lighter skeleton and greater proportion of fat and hence can float more readily. Infants and young children also float more readily. When the living body is immersed in water, expansion of chest further lowers the specific gravity and it differs so little from that of water that a little motion/movements of the hands and/or feet will be sufficient to keep the individual on the surface. The condition of the lungs plays an important role in influencing buoyancy of the body, that is why a person with large and capacious chest tends to float more easily than one with a smaller contracted chest. Obviously therefore, a scream/shriek leading to an almost emptied chest at the time of fall is unfavourable to floating. Nature of clothing on the person may also make a difference. Loose light clothing may serve to buoy up the body, whereas heavy clothing may cause it to sink. Again here, women are better placed as their loose clothing usually traps air that helps them float readily.

Generally therefore, as discussed above, a recently dead unclothed body is heavier than water and sinks when immersed. After a variable period, the body will rise again and float upon the surface. This period of floatation is influenced by both endogenous and exogenous factors. **Endogenous factors** may include specific gravity of the body, age, sex and prior physical status, etc. **Exogenous factors** may include nature of water (whether salt or fresh, polluted or clean, stagnant or running, etc.), temperature of water, season of the year and other conditions facilitating putrefaction. Therefore, with the development of sufficient gases of decomposition, the body rises to the surface and usually floats with belly upwards owing to abundance of gases in the gastrointestinal tract. The head and face have a tendency to remain at a lower level than the rest of the body as the head is relatively heavy. This favours early gravitation of blood into the head and face and hence more marked decomposition. The body tends to assume a characteristic posture—the trunk being uppermost, head and limbs hanging passively. If the developed gases happen to escape, the body may again sink, but may again rise as a result of formation of more gases. However, some dead bodies may not float because of being entangled in vegetation/weeds or any other impediments. **Conversely**, a body may sink like a stone if weighted by some apparatus or stones or heavy boots etc. Rarely, a body may become disintegrated (if attacked by some fish or crabs or the like) before the conditions tending to promote floating are established. In India, floatation of the body usually occurs by about 24 hours in summer and 2–3 days in winter.

SUICIDE, ACCIDENT OR HOMICIDE (Fig. 6.17)

Majority of deaths due to submersion are either by accident or by suicide. The victim of accidental drowning is usually a child or an adult male, whereas the suicide may be committed either by a male or female adult. Admittedly, the medical data are of secondary importance against the other collateral evidence. However, the medical evidence as to the actual fact of death from drowning is of critical significance in this direction. The deceased might have been stunned prior to or during fall into



Fig. 6.17 Officials attempting to reconstruct events of a case through dumping a crude dummy in the sewerage pipeline. The case pertains to death of a lady constable (aged about 27 years and about 8-month pregnant) by falling into a manhole, which was allegedly un-covered. The police asserted that the corporation staff was liable to be prosecuted against for negligence. As the issue of accident, suicide or homicide assumed alarming significance, every effort was made to untangle the problem, including reconstruction of events through dumping a crude dummy and correlating its passage through the sewerage pipeline under circumstances attending the case.

water or might have been so intoxicated as to have been unable to help himself or might have succumbed to vagal inhibition due to fright or shock from sudden immersion or death might have ensued from some totally independent natural event such as coronary catastrophe, epilepsy or otherwise, when the position of the victim at about the time of death was such as to cause a fall into water. All such situations have already been discussed at length.

Injuries upon the body must be attended carefully. Injuries may be sustained before and/or during and/or after entry into water or may have been inflicted through physical violence. They may be grave enough to account for death or may be slight but of much medicolegal significance depending upon their nature and distribution. However, pressure marks by the collar or tie or by woman's neckwear or by some other clothing that may become more pronounced as the body becomes distended owing to decomposition may not be misinterpreted. The principal problem hangs about the issue, whether the injury or injuries is/are result of accident or design, and in framing an opinion, compatibility or incompatibility of such injuries with the circumstantial evidence available at the scene must be taken care of. Significant surface injuries may sometimes be caused under water when the body has been carried by the current of water against mechanical forces. Therefore, presence of injuries should not lead to a hasty suspicion of foul play. Conversely, the victim may be pushed or chased into water and bear no evidence of assault. Recognition of injuries being antemortem or postmortem is of paramount importance. Ordinarily, they may not present any difficulty in differentiation as the injuries sustained/inflicted after death are attended by absence or negligible haemorrhage. At occasions, perimortem production of injuries may pose a problem and further, the presence of body under water (which is likely to wash away the blood) may multiply the problem.

Drowning in shallow water always merits explanation. This may occur accidentally under the circumstances described above. However, under rare situations, the assailant may hold the victim's head in such a position until life is extinct. But its accomplishment requires an appreciable physical disparity between the assailant and the victim except when the victim is a child or is incapacitated by drug/disease or is taken by surprise or is overpowered by more than one stalwart. Marks of violence may or may not be present in such cases. In the case of RV Smith, known as **'the brides in the bath case'**, no less than three women were done to death by drowning in a bath. The assailant had managed to immerse the victim by suddenly lifting the legs up and pushing the head under water. Only in one case, some signs of violence were available, which were in the form of three bruises on the arm.

Ligatures on the body or weights attached to the body may be a good pointer towards homicide but cases have been reported where determined suicides have tied themselves before rolling into water to ensure success. The nature and manner of application of weights to the body and the presence/absence of attending injuries deserve consideration. Constrictions or marks, especially around neck, give strong evidence for homicide provided signs of drowning are wanting. Another situation raising suspicion for homicide may be that there are evidences of struggle on the banks of river/canal from where the body has been recovered and especially when the articles belonging to someone other than the deceased are found on the banks and associated with those known to have belonged to the deceased, or where some fragments of clothing/hair, etc., (not corresponding to his own) are found grasped in the hand(s) of the victim.

Analysis, especially for alcohol, may throw some light on the circumstances because drunkenness is a common cause for accidental immersion. Even good swimmers drown if they are intoxicated. The explanation may be found in the vasodilatation of the skin produced by alcohol, which leads to an increased skin temperature, enhancing the sudden cooling effect. A similar mechanism may be operating when an already exhausted person jumps into the water to cool off, without first letting the skin to cool.

Homicidal drowning is extremely rare. Copeland (1986) reported 10 cases out of a total of 2617 homicides. Homicide by drowning is easier to conduct if the victim is rendered helpless by intoxication or drugs or by violence. In the notorious insulin case, the husband (a male nurse) gave an injection of insulin to his pregnant wife, the wife taking it to be of ergonovine intended to induce abortion. Thereafter, when she lapsed into hypoglycaemic coma, the husband placed her in a bathtub to present the case as of death due to drowning (Bir Kinshaw et al., 1958).

7
CHAPTER

Infanticide and Foeticide

After going through this chapter, the reader will be able to describe: Infanticide and the related law | Primary and secondary issues to be resolved in relation to infanticide | Age of viability and its medicolegal significance | Concept of live birth and separate existence | Proof of live birth | Hydrostatic test and its importance | Other tests for separate existence | Probable duration of life of the child | Autopsy | Cause of death, i.e. acts of commission and acts of omission | Foeticide | Abandoning of children and concealment of birth | Development of foetus

Finding dead bodies of newborn infants in the sewers, alleys, trash dumps, streams, lakes, public lavatories, bushes, dry wells, etc. is somewhat common in large cities. The killing of the newborn infants has been practised from time immemorial for a variety of reasons. **One of the basic reasons**, probably, was the survival of the fittest or the safety of the tribe, i.e. those with some malformations or having less potential value for the family (such as females) were used to be killed. Tribal superstitions—the issue of unlucky child as per astrology and even the leg presentations—used to be the other causes. Today, the social stigma attached to the out-of-wedlock pregnancy is usually the most common motivating factor to resort to the commission of such a crime. Superstitions, poverty and ignorance may be the other factors, especially amongst the village folk.

Infanticide

THE LAW

From the point of view of law, the offences against children may be dealt with on the same lines as if the victim were an adult. **In India**, there is no distinction in law between infanticide and murder, such as exists in many Western countries like England, Germany, France, etc. In English law, there are special provisions dealing with certain offences against children. **The English Infanticide Act, 1938, Section 1, provides:** Where a woman by any willful act or omission causes the death of her child, the child being under the age of 12 months, but at the time of 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 effect of lactation consequent upon the birth of the child, then, notwithstanding that the circumstances were such that but for this act, the offence would have amounted to murder, she shall be guilty of felony of infanticide and may for such offence be dealt with and punished as if she had been guilty of the offence of manslaughter of the child.

The analysis of the above provision reveals:

- The word 'woman' speaks that this benefit of diluting the offence to the manslaughter only extends to the mother—not the father or any other person. If anyone else is involved in the charge in assisting the woman for this crime, he will be charged with murder.
- The words 'causes the death of her child' lay stress that it has to be a 'child', i.e. a person with a separate existence outside the mother's body.
- The child must be under the age of 12 months; though most of the infanticides are committed within hours or a short period after birth, yet this limit has been provided for legal purposes.
- The circumstances leading to the death of the child must be willful (deliberate) act(s) of omission or commission.
- There must be evidence to show that the mother (accused) was suffering from disturbance of functions of mind due to reasons mentioned in the provision. For this purpose, the opinion of an experienced forensic psychiatrist, who had been attending to the accused, should be invited.

Whether the crime is to be treated on the lines of manslaughter (as in England) or on the lines of murder (as in India), certain facts have to be established by a doctor, as amply revealed from the above provisions, before the criminal charge can be brought, namely:

Primary issues:

- The child was capable of survival after birth.
- The child was born alive and had a separate existence outside mother's body.

- The death was caused by willful act or omission. Here the violence inflicted upon the child has to be differentiated from the injuries incidental to the birth, i.e. accidental injuries connected to the birth trauma, whether during or succeeding the birth.

Secondary issues:

- Probable duration of life of the child, i.e. degree of maturity of the child.
- It may also be necessary to prove that the mother has recently delivered, and the period of delivery coincides with the probable duration of the life of the infant and she had been suffering from the disease of the mind due to effects of having given birth to the child or due to the effects of lactation at the time of act of commission or omission.
- Connection between the identities of the child and the mother requires to be traced, i.e. the suspect, in fact, is the mother of the child.

Primary Issues

The **first primary issue** is to show that the child was capable of survival after birth. It comprises two components, i.e. 'capability to survive' and 'after birth'. In the legal sense **'birth'** constitutes complete expulsion of the child from the maternal genital passage irrespective of severance of the cord or delivery of the placenta. Therefore, the destruction of a partially born child (a child whose head is out of the genital passage but legs still within the genital passage and the child has cried after the delivery of the head) is not regarded as infanticide, though it does seem paradoxical from the medical point of view. But this is the law and one has to bear with it. Cases have been reported to have occurred where the killing of the child, one of whose legs were undelivered, was not held to be infanticide and consequently the woman was acquitted. Such cases, however, may be covered under the Infant Life (Preservation) Act, 1929, which covers the eventuality of deliberate destruction of the child before birth. In this respect, the Indian law is better placed and more appropriate that considers it to be a 'live birth', even if any part of the living child has been brought forth from the body of the mother. The other component is the capability to survive, which imports the concept of **viability**.

Establishing the particular duration of pregnancy is an important part of the complete medical burden. Unless the child has attained such degree of development as to be consistent with physical ability to survive, a charge of infanticide will not stand on any sound footing because the children whose age is less than this gestational period (period of viability) are usually presumed to be incapable of leading an independent existence owing to their immaturity. This age of viability may vary according to the condition of the particular foetus and the availability of medical facilities but under the English law, a period of 28 weeks of gestation was fixed for the onset of viability for the purposes of Infant Life (Preservation) Act, 1929. However, a foetus is usually considered to be viable at the age of 210 days (seven calendar months) as per Indian standards.

Proof of viability is relatively simple because most of the victims of infanticide and child destruction are mature, usually having attained a period of 36th week of gestation. **A combination of criteria as given below should be adopted to have a reliable opinion:**

- General condition of the body of the infant should be observed for any disease or malformation, etc. Weight, head circumference and crown heel length of the child need to be determined. It has been shown that there is reasonably close relationship between the age and weight of the foetus. However, when there are multiple births, the weight of each infant may be considerably less than that of single birth at the same stage of gestation. Due allowance may also be given to the sex since the female foetus is usually about 100 gm lighter than the male at the same stage of gestation.
- The crown heel length carries importance. According to the Haase Rule (1895), the length of a foetus up to 5th month (20th week) of gestation represents the square of its age in months. Thus, a foetus of about 4 months will have a length of 16 cm. Beyond 5th month, the length of the foetus measured in centimetres divided by 5 gives the age in months. Thus, a foetus of 35 cm length will be about 7th month of age. This is known as Morison Rule (1964).
- Radiological examination of the entire body may be carried out rather than conducting extensive dissection for the demonstration of the epiphyses. Though the time of appearance of the ossification centres is variable, yet their values cannot be overlooked (Fig. 7.1):

At the 28th week:	There are usually centres of ossification in the calcaneum, and talus.
At the 30th week:	(Usually accepted as age of viability) Ossification centres for all sacral vertebrae are usually present.
At the 36th week:	The centre of ossification in the lower end of the femur. This centre in the lower end of femur is most important because it is exceptional for this centre to be absent at maturity of the infant.

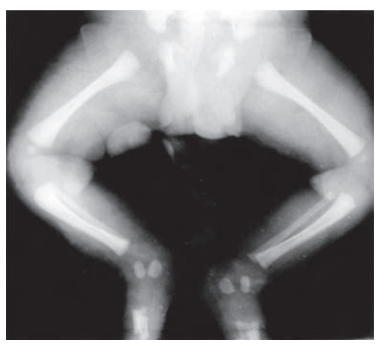


Fig. 7.1 X-ray of a full-term child showing centres of ossification.

The **second primary issue** is that the child was 'born alive' and had a separate existence.

The concept of **'separate existence'** must be understood unambiguously because the critical legal requirement resides in proving that the child was born alive and that it did have an independent existence. The law, therefore, lays stress on the differentiation of 'foetal life' and 'independent life' and the latter inevitably calls for presence of independent respiration or any other sign like independent circulation. This is something where the law runs parallel with the views of Barcroft, who said, 'Breathing is living: the onset of respiration is the beginning of (extra-uterine/independent) life'. It may be made more clear by citing an example, i.e. if an injury inflicted upon the mother who is quick with the child, results in death of the mother as well as the child, it will obviously be homicide, but if it causes the death of the unborn quick child only, it may be treated under Section 316 of IPC dealing with 'causing death of unborn quick child by an act amounting to culpable homicide'. However, an injury inflicted upon the unborn child that necessitates the process of delivery and causes death of the child when the child is fully born amounts to felony of homicide.

Before proceeding to a detailed discussion about 'live birth', it is better to have some idea about the conditions like stillborn or deadborn, etc. so that one is able to differentiate them easily. As already described, a **stillborn child** is one who has issued forth from its mother after 28th weeks of pregnancy and which did not, at any time after being completely expelled from its mother, breathe or show any other sign of life. A **deadborn child** is one who died in utero before the birth process began and may show one of the following signs after it is completely born:

- **Signs of maceration:** Maceration is a process of aseptic autolysis that occurs when the dead child remains in the uterus for some period surrounded with liquor amnii, but with the exclusion of air. Hence, if the child died in utero about 12 hours before it was born, the signs of maceration may not be seen and in such cases it would be difficult to say whether the child died in utero or during the birth.

  The earliest sign of maceration is skin slippage, which may be seen in 12 hours after the death of the child in utero (Fig. 7.2). The body of the macerated foetus is soft, flaccid and flattened and emits a sweetish disagreeable smell, which is quite different from that of putrefaction. The skin shows red or purple colouration but never greenish as in putrefaction. Large blebs containing serous or serosanguineous fluid are raised upon the surface of the skin, and epidermis is easily peeled off leaving moist greasy areas. The tissues are generally oedematous and turbid reddish fluid collects in the serous cavities. The bones become flexible and readily detached from the soft parts. All the viscera are oedematous and lose their morphology but lungs and uterus may remain unaffected for a longer period. The umbilical cord is red, smooth, softened and thickened. Loss of alignment and over-riding of the bones of cranial vault occurs due to shrinkage of the brain after death. This is known as **Spalding's sign**. It may be detected within a few days of death of the foetus in utero.
- **Signs of mummification:** May be seen when the foetus dries up from the deficient supply of blood and scanty liquor amnii, but with the exclusion of air. If the air gains entrance due to rupture of the membranes, the foetus undergoes putrefaction instead of maceration.



Fig. 7.2 Flattened body of a full-term deadborn child showing signs of maceration.

Proof of live birth/separate existence: This particularly implies the achievement of breathing, as already remarked, **"Breathing is living: the onset of respiration is the beginning of (extra-uterine/independent) life" (Barcroft).**

In the civil cases, the cry of the child, feeling, seeing or hearing of the heart beat or slight muscular movements such as twitching of the eyelids or a pulsating cord may be taken as sufficient to establish respiration. However, it may be kept in mind that it is possible for the child to cry while the head is still in the uterus (vagitus uterinus) or in the vagina (vagitus vaginalis) and to die before it is completely born. This can occur only if the membranes have ruptured and air has gained entrance into the uterus. It may also be possible that the child may not utter a cry, particularly when immature, and even then may be born alive. The law presumes that every newborn child found dead was born dead until the contrary is proved. Therefore, in **criminal cases**, signs of live birth have to be demonstrated by postmortem examination, and obviously the air passages and digestive tract will afford the strong evidence—whether the respiration has taken place or not.

Examination of the respiratory system (Table 7.1) includes the following:

- **Shape of the chest:** Before respiration, the chest is flat but it becomes arched or drum shaped after respiration.

Table 7.1 Difference between Respired and Unrespired Lungs

Features	Respired lung *(changes will vary quantitatively depending upon the length of time and the depth to which breaths have taken place)*	Unrespired lung
Chest	Arched or drum shaped. Circumference greater than that of abdomen. Intercostal spaces wider	Flat. Circumference less than that of abdomen. Intercostal spaces narrow
Diaphragm	Level of sixth or seventh rib	Level of fourth or fifth rib
Lungs		
Position	Fill the thoracic cavity, overlap heart, with taut covering pleura	Lying at the back of thoracic cavity behind the heart. Covering pleura wrinkled and loose
Volume	Voluminous	Small
Edges/margins	Rounded to varying extent (weak/feable or brief respiration ordinarily affects the margins, whilst more robust or prolonged breathing leads to total expansion)	Clearly defined
Colour	Mottled pink	Uniformly reddish brown or bluish red
Appearance	Marbled due to expanded air vesicles and development of circulation	Smooth and not marbled
Consistency	Spongy, elastic and crepitant	Dense, firm, liver-like and noncrepitant
Alveoli	Expanded, rising above the surface	Not so
Cut-section	Exude frothy blood	Exude little blood but no froth unless decomposed
Weight:body weight	Something of the order of 1/35th of body weight	Something of the order of 1/70th of body weight
Hydrostatic test and microscopy	'Limited value' (see discussion in the text)	—
Medicolegal importance	Significant of 'live birth'	Indicates still birth or dead birth

- **Position of the diaphragm:** The abdomen should be opened before the thorax, and highest point of the diaphragm should be noted that is found at the level of fourth or fifth rib if respiration has not taken place. The arch becomes flattened and depressed and descends to the level of sixth or seventh rib after the establishment of the respiration. The position of the diaphragm may be affected by the pressure of the gases of the decomposition.
- **Changes in the lungs:** These may be considered with reference to the following:
  - **Volume:** Before respiration, the lungs are small with sharp margins, covered by wrinkled loose pleural membranes lying in the back part of the chest on either side of vertebral column. After complete respiration, the lungs increase considerably in volume, covered by thin, tense pleura, have rounded margins and occupy the thoracic cavity, the left lung covering more or less the thymus and the heart. Glistening bullae may appear along the margins when there has been a struggle to breathe due to any natural or unnatural obstruction to the breathing.
  - **Consistency:** Before respiration, lungs are dense, firm, noncrepitant and liver like. After respiration, they are spongy, elastic and crepitant.
  - **Colour:** Before respiration, the colour of the lungs is uniformly reddish-brown like that of liver. The surface of the lobules is marked with shallow furrow. On section, little frothless blood exudes on pressing the cut surface. After respiration, the collapsed air cells first become distended with air, usually on the edges and concave surface of the upper lobe of the right lung and then on the remaining portions of the lungs. They are more or less mottled or marbled in appearance as the blood becomes aerated in the expanded area. On section, frothy blood exudes from the surface on application of pressure. The foetal lungs may assume more or less rosy colour on exposure to air after death but the air cells can never be distended by simple passive entrance

of air into the lungs. This condition may be simulated by artificially inflating the lungs, giving similar appearance in volume and colour but mottling is mostly absent.

- **Weight:** The weight of the lungs is almost doubled after the aeration. Before respiration, the lungs usually weigh about 30–40 gm and after respiration about 60–70 gm. The increase in weight is due to filling up of pulmonary blood vessels with the blood. The weight may increase from 1/70th to 1/35th of body weight after respiration. This is called the **Ploucquet's test**. But these factors like increase in the weight of the lungs in relation to the total weight of the body vary greatly and have little medicolegal bearing.

- **Presence of extraneous material in the lungs:** Presence of extraneous material in the respiratory passage is an important finding suggesting live birth, particularly in the distal respiratory passage because extraneous material can enter the air passage even after death but up to a limited distance and not into the intrapulmonary bronchi where its entry may be resisted by the air in the lungs. Hence, demonstration of extraneous material in the secondary bronchi and beyond is strongly indicative of its inhalation.
- **Hydrostatic test:** The test was first noted by Scheyer in 1683. With due recognition of its limitations, the test is helpful and may be performed. It is based on the principle that the specific gravity of the unrespired lungs varies from 1.04 to 1.05 and that of respired lungs is 0.94, because of increase in volume due to inhalation of air. The fetal lungs therefore sink in water and those that have respired float.

  Remove the lungs as far as the trachea along with the larynx by tying at the laryngeal end and place them in a jar of water and note for their floatation. The lungs are then separated and each is tested separately for the presence or absence of floatation. Finally, each lung is then cut into fragments that are again tested for floatation. If in all such events, floatation is present the test is positive (provided putrefaction is absent). If there is some decomposition, then the fragments should be compressed to remove the tidal air. The fragments are again placed in water. If they continue to float even after this compression due to presence of residual air, the test is positive and respiration has taken place. If some of the pieces sink and some float, it shows feeble respiration owing to the partial penetration of the air.

  - **False-positive findings** may be evoked under the following circumstances:
    - **Expanded lungs may sink from:**
      - Diseases like acute oedema of the lungs causing death of a newborn infant within a short time may be demonstrated histologically, if due precautions are taken to prevent draining of the amniotic fluid and the oedema fluid during preparation of the sections for histopathology. Bronchopneumonia should also be excluded. Further, it may not affect the entire lung uniformly and the unaffected portion may float in water.
      - Atelectasis (nonexpansion) may be due to obstruction by alveolar duct membrane or due to extreme feeble respiration or sometimes more air may be expelled from the lungs during expiration than it is inhaled during inspiration or air may not reach the alveoli but the aeration of blood is being maintained through the lining membrane of the trachea and bronchi.
    - **Unexpanded lungs may float from:**
      - Presence of putrefactive gases for which obviously other signs of putrefaction will also be evident.
      - **Artificial respiration:** The foetal lungs may be artificially inflated by blowing air through a tube or catheter or by mouth-to-mouth method or by Schultze's method. But lungs may be inflated partially and complete expansion is unlikely. In such cases, the stomach also usually contains air while it is airless in stillborn infants. The possibility of inflating the lungs artificially should be kept remote because it is difficult to conceive why a person who desires the death of the child should endeavour to resuscitate the child. However, an ultra-criminal mind may go in for such tactics (Fig. 7.3).
  - **Hydrostatic test is not necessary when:**
    - The foetus is born before 180 days of gestation.
    - The foetus is a monster and thereby incapable of leading a separate existence.



Fig. 7.3 X-ray photograph of a full-term dead-born child showing ground glass appearance of lungs except some air in the right middle zone. This was probably due to artificial respiration attempted at the time of delivery.

— The foetus shows signs of intrauterine maceration.
— The umbilical cord has separated and the umbilicus has cicatrised.
— The stomach contains milk showing active digestive function.

Other tests for separate existence include the following:

- **Air in the gastrointestinal tract:** Hajkis (1934) suggested that radiological demonstration of air in the stomach and intestines is a strong evidence of respiration. This is due to the fact that during the process of respiration, some air is likely to be swallowed, going into the stomach and further into the intestines due to peristalsis. According to Hirvonen et al., the air swallowed during crying can be seen in the stomach of the infant 5–15 minutes after the birth, in the small intestine after 1–2 hours and in the large intestine after 5–6 hours. Putrefaction and artificial respiration should, however, be excluded (Fig. 7.3). Test may be carried out by removing stomach and intestines after applying double ligatures at each end of the stomach, at the end of the duodenum and also some lower parts of the intestines. On placing them in water, they will float. Then they are tested separately for floatation. This is known as *Breslow's Second Life Test*. It is a corroborative test. On careful dissection under water, the stomach may show presence of mucus with air bubbles and saliva if respiration has taken place, and presence of only glairy mucus if respiration has not taken place.
- **Presence of milk in the stomach:** Presence of milk or farinaceous food in the stomach is a strong indication that the child was not only born but also lived for sometime after birth.

Concludingly, it has been forwarded that there are three schools of thought in general in considering 'proof of live birth', viz., *(i)* hydrostatic test, *(ii)* microscopy of the lungs and *(iii)* circumstances (whole case investigation plus examination).

Hydrostatic test is presently considered to be of limited value. It has been suggested that if the whole 'respiratory apparatus' floats, indication of breathing is invited and this may serve as a corroborative role of this test towards determining 'live birth'. However, difficulty in interpretation of findings arising out of even minimal degree of decomposition restricts its practicality. Difficulty is further confounded by considerations that so many potential infanticides are found hidden, buried, or submerged, thus evading performance of test in a large proportion of cases. Further in the present scenario, the advent of resuscitation attempts (like mouth-to-mouth breathing, external cardiac massage, and administration of oxygen, etc.) has made the evaluation of breathing more difficult. Hence, doubts better be resolved in the direction of 'no breathing' so as to avoid false sense of scientific validity.

Microscopy of the lungs involves looking for the evidence of alveolar aeration or pulmonary interstitial emphysema. For this purpose, thoracic contents should be removed intact up to the larynx by 'no touch' technique of Osborn, thus eliminating the artefacts likely to be produced by careless manipulation. Sections need be prepared from the whole lung after due fixation (it is worth mentioning here that even ordinary handling of the dead body has been incriminated for the entry of air into foetal lungs and apparently respired alveoli have been found in lung sections from a dead infant taken from the uterus of a dead mother). An added problem may arise in cases where there has been minimum respiration or in those infants who have succumbed to 'struggle to breathe' wherein blood may be drawn into the lungs but the process may finally not be successful, thought it may slightly expand the lungs or give a few subpleural spots and cause oedema in the lung tissue (such changes may also be found in an intentional attempt to prevent an infant from breathing but here, other external corroborative evidence of injury/pressure on the face or neck will be self-explanatory). Therefore, microscopy of the lungs too has been considered to be of dubious value.

Circumstances (whole case investigation plus examination): Investigators must try to obtain as much information as possible and to consider the findings in entirety. Such a view is amply supplemented in the advice of **Lester Adelson**, "Unless the pathologist has incontrovertible criteria of postnatal survival, e.g. well-expanded lungs, food in the stomach or vital reaction in the stump of the umbilical cord, he is legally bound not to diagnose live birth. Convictions for infanticide have been set aside where there was any doubt whatsoever that the child was born alive. Many courts have pushed this proposition to the extent that the state has been given the burden of proving that the baby was born alive beyond any possible doubt rather than beyond any reasonable doubt, the latter being the general level of proof required in a criminal prosecution."

Secondary Issues

Probable duration of life of the child, i.e. if born alive, how long did the child survive after the birth?

This issue is connected with the second secondary issue that requires to be proved that the mother has recently delivered and that the period of delivery coincides with the probable duration of the life of the infant or the degree of maturity of the infant. To determine the probable length of time the child has survived after its birth, the following changes may be helpful.

- **Changes in the skin:** The skin of the newborn infant is bright red and covered with vernix caseosa (a white cheesy substance made of sebaceous secretions and epithelial cells. Being sticky, it cannot be removed easily). The vernix caseosa is chiefly present in the flexures of the joints and neck folds. It is not easily removed and persists for a day or

two. After birth, the skin becomes darker on the second or third day and finally assumes its normal colour within a week. Physiological jaundice is evident between the third and sixth day. Exfoliation of the skin occurs during the first 3 days after birth.

- **Subgaleal haematoma, cephalohaematoma and caput succedaneum:** A subgaleal haematoma is a space occupying blood clot located between the periosteum of the skull and the galea aponeurotica. In most cases, it is located at the top of the head. It has been theorised that the negative forces imparted by traction applied on the top of the head can pull the aponeurosis from the cranium and injure the emissary veins connecting dural sinuses with the scalp veins. Cephalohaematoma is also a space occupying blood clot but situated beneath the periosteum of the skull and is caused by tearing of diploic veins due to mechanical trauma. They have been associated with higher parity, higher birth weight and instrumental delivery. Caput succedaneum is another entity needing differentiation. It is an area of transient congestion and oedema in the scalp tissues located over the presenting region of the head in cephalic presentation. Caput succedaneum disappears from 24 hours to 2–4 days after birth; cephalohaematoma, if present, will show the usual colour changes common with the bruises and disappears in about a fortnight.
- **Changes in the umbilical cord:** Changes in the umbilical cord begin to appear in the cut end to its base at the umbilicus soon after birth. Even when the putrefaction has rendered the evaluation of breathing extremely difficult, vital signs in the cord may be helpful in indicating live birth if there has been sufficient survival period. The portion of the cord attached to the child shrinks and dries within 12–24 hours and an inflammatory ring or a reddening ring appears at its base and the adjacent skin from 36 to 48 hours. By the second or third day, it shrivels up, mummifies and falls off on fifth or sixth day, leaving a raw area that heals and cicatrises within 10–12 days.
- **Circulatory changes:** Nucleated red blood cells begin to be formed in the yolk sac and mesothelial layers of placenta at about the third week of foetal development. At about 6 weeks, the liver begins to form blood cells, and in the third month, the spleen and other lymphoid tissues begin forming blood cells. From third month onwards, the bone marrow gradually becomes the chief source of the red blood cells as well as most white blood cells. Foetus contains foetal haemoglobin (haemoglobin F). Its structure is similar to that of adult haemoglobin except that the β chains are replaced by γ chains. The γ chains contain similar number of amino acid residues but some differ from those in the β chain of the adult haemoglobin. Foetal haemoglobin is normally replaced by adult haemoglobin shortly after birth. Oxygen content of foetal haemoglobin at a given PO_2 is greater than that of adult haemoglobin because the former binds 2,3-DPG (diphosphoglycerate) less avidly. This facilitates movement of oxygen from the maternal to the foetal circulation. Nucleated red blood cells usually disappear from the peripheral circulation within 24 hours or so.

At birth, following essential changes in circulation take place:

- The tremendous blood flow through the placenta is lost, which increases the aortic pressure as well as pressure in the left ventricle and left atrium.
- Pulmonary vascular resistance gets decreased considerably as a result of expansion of lungs. Due to expansion, the blood vessels are no longer compressed and the resistance to blood flow decreases. (In the unexpanded foetal lungs, blood vessels were compressed due to small volume of lungs.)

Depending upon the above changes, **closure of the foramen ovale** (the aperture in the septum secundum of the foetal heart that provides a communication between the atria, also called ovale foramen of foetal heart or ovale foramen of foetus) may be explained thus: high left atrial pressure that occurs secondarily to the above described changes at birth results in attempting to cause the flow of blood backwards through the foramen ovale. Therefore, blood tends to flow from the left atrium to right atrium, rather than in the other direction prevalent during foetal life. Consequently, the valve that lies over the foramen ovale on the left side of atrial septum closes over this opening. Further, **closure of the ductus arteriosus** (a foetal blood vessel connecting the left pulmonary artery directly to the descending aorta, also called arterial canal, duct of Botallo or pulmoaortic canal) occurs due to changes like (*i*) elevation of aortic pressure due to increased systemic resistance and (*ii*) reduction of pulmonary arterial pressure due to decreased pulmonary resistance. Consequently, after birth, blood begins to flow backwards from aorta into the pulmonary artery through the ductus arteriosus, rather than in the other direction as in foetal life. However, the muscular tissue of the ductus arteriosus starts constricting and within 1–8 days, the constriction becomes sufficient to stop the blood flow. During the next few months, the lumen of ductus arteriosus ordinarily becomes occluded by the growth of fibrous tissue. **Closure of ductus venosus** (major blood channel that develops through the embryonic liver from the umbilical vein to the inferior vena cava, also called canal or duct of Arantius, canal of Cuvier or ductus Arantic) occurs because of the fact that immediately after birth, blood flow through the umbilical vein ceases, but most of the portal blood still flows through the ductus venosus. However, within a few hours, the muscular tissue of the wall of ductus venosus starts constricting and ultimately closes. Consequently, portal venus pressure rises enough to force portal venous blood flow through the liver sinuses (Fig. 7.4).

- **Respiratory changes:** The most obvious effect of birth on the baby is the loss of placental connection with the mother and the most important immediate adjustment is the

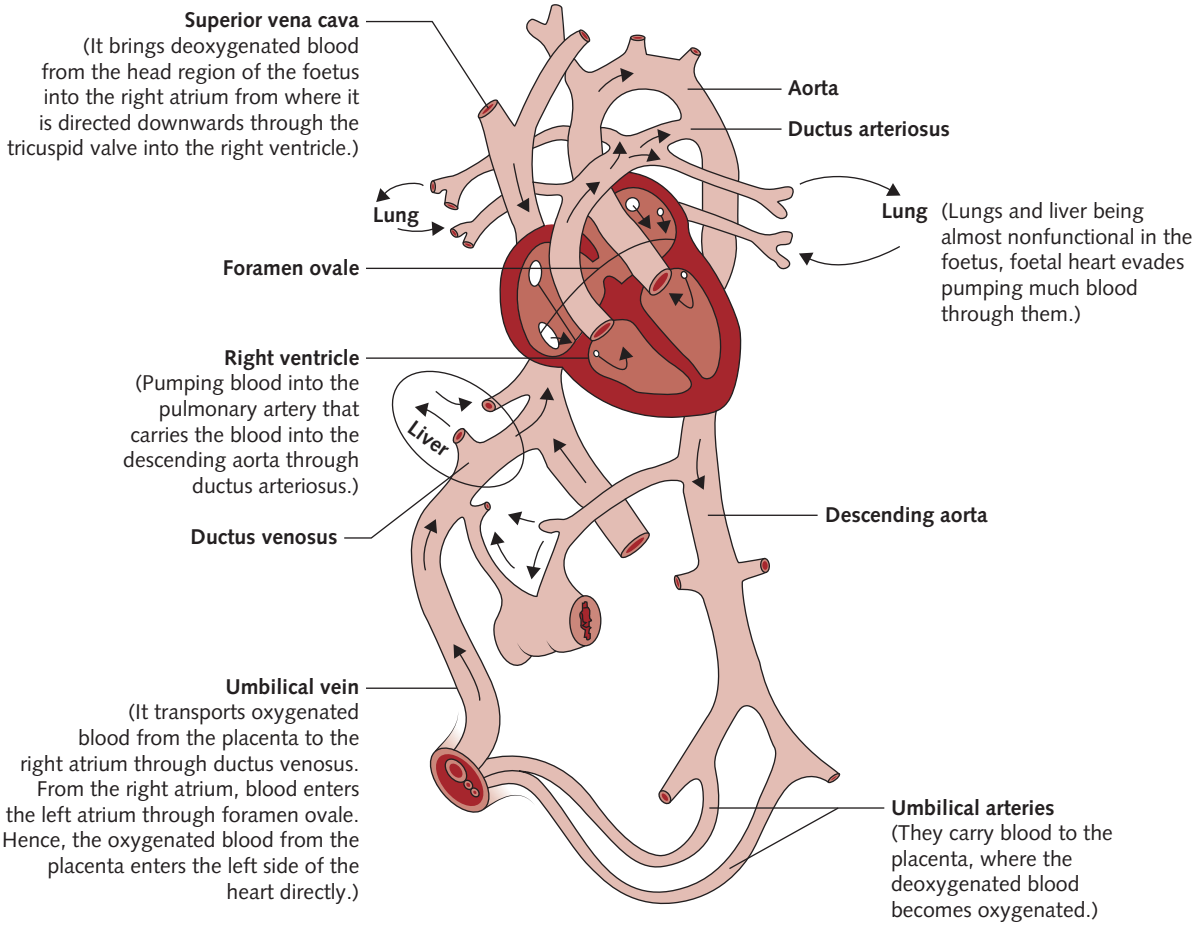


Fig. 7.4 Diagrammatic and descriptive presentation of foetal circulation (modified from Arey LB: Developmental Anatomy).

establishment of breathing. The child begins to breathe within seconds after birth. The process of breathing probably results from sensory impulses originating in the suddenly cooled skin and some asphyxiated state incidental to the birth process. This explains the delayed onset of respiration for several minutes if the mother had been instituted general anaesthesia during delivery. This can also occur in prolonged delivery of head trauma during delivery.

At birth, the walls of the alveoli are collapsed because of the surface tension of the viscid fluid contained in them. To overcome the effects of this surface tension and to open the alveoli, powerful inspiratory efforts are required. As reported, the initial inspirations of the normal neonate are powerful enough for creating as much as 50–60 mmHg negative pressure in the intrapleural space. Once the alveoli get opened, further respiration can be effected with relatively weak respiratory movements. The surfactant secreting cells (type II alveolar epithelial cells) do not begin to secrete surfactant until the last 1–3 months of gestation. Hence, many premature babies may be born without the capability to secrete sufficient surfactant leading to collapsed tendency of alveoli. (Surfactant is a substance normally secreted into the alveoli that decreases the surface tension of the alveolar fluid, thus allowing the alveoli to open easily during respiration) (Fig. 7.4).

Another secondary issue involves the establishment of connection between the identities of the child and the mother.

Newborn infants found dead may not necessarily be the victims of infanticide. The stillborn or dying naturally may be hidden or abandoned, for which the crime is 'concealment of birth'. A verdict of concealment of birth is an alternative to that of infanticide and in view of the problems for proving the main charge of infanticide, the person may be convicted of concealment of birth. Although the mother is the person usually convicted of concealment of birth, yet all who are concerned with the process of concealment stand as principals.

Identity of the child may be traced from blood grouping, which may help in eliminating or helping to confirm the consanguinity of any putative mother. DNA profiling is the latest achievement that helps in the establishment of identification.

AUTOPSY TO ESTABLISH CAUSE OF DEATH

As already emphasised, the circumstances leading to the death of an infant must be some act(s) of commission or omission and, consequently, the violence inflicted upon the child has to be differentiated from the injuries incidental to the birth, i.e. the accidental injuries connected to the birth trauma. The procedure for autopsy is almost same as in adults except certain deviation and need for specific attention to certain matters as detailed below:

- **Clothing and wrappings:** The coverings or wrappings and other articles associated with the infant need to be examined and retained. It may be torn clothing of the mother or newspaper or plastic bag or rags, etc. Any foreign material available should also be collected. All this would help in identification.
- **Measurements:** Estimation of weight, crown heel and crown rump lengths, head circumference, etc. is essential to know about the degree of maturity. For details, see the description at the end of this chapter.
- **Changes of decomposition:** It is vital to assess the changes of decomposition as these will help to ascertain the time since death and also make the doctor aware of the precautions to be observed while performing hydrostatic and other tests. Bodies of the newborn infants are normally sterile. When they breathe and swallow, micro-organisms gain entrance. Therefore, there may be differences in onset and degree of putrefaction in the stillborn and liveborn infants. Decomposition must be differentiated from intrauterine maceration, because the latter is a sure sign of deadborn foetus.
- **Presence/absence of vernix caseosa**, any injuries upon the body, particularly around the mouth, nose and upon the neck should be carefully examined. Presence of vernix caseosa is not as useful a sign as its absence, as the latter indicates that the child had been washed suggesting that it survived for sometime after birth. Foreign material/objects may be found in the mouth, nose or respiratory tract. Other orifices should also be examined at this stage. Presence or absence of caput succedaneum requires to be noted.
- **Umbilical cord and placenta:** If the cord and placenta are present, they constitute very important evidence and help in solving many issues. Placenta should be weighed to evaluate maturity, and any abnormality should also be observed. The changes in the cord are an important indicator of separate existence. The severed end of the cord deserves special attention as it may help in concluding whether the cord has been actually cut or broken because in the latter case, the defence of the mother that the death of the child occurred due to falling upon the ground in the course of precipitate labour may succeed. In such cases, length of the cord (if placental segment is available) should be measured so that the compatibility with the defence offered by the accused mother could be assessed. Morris and Hunt conducted experiments on the cords and determined that they could easily be broken with the traction of the hands. They described the appearance of ends of the cord by different modes of severance. The cut ends with a sharp instrument like scissors or knife will appear clean-cut but occasionally may appear ragged if the instrument is relatively blunt.
- Any evidence for malformations or birth injuries should be meticulously searched for, which may reveal obvious incompatibility with the continuation of life.

Internal Examination

The examination must follow a certain routine and should be complete.

Head

The scalp is opened by the usual incision from ear to ear and the flaps reflected. The skull is opened by cutting with scissors anteroposteriorly and across, and reflected as four flaps. Observation is made regarding injuries to fontanelles (especially punctured wounds through anterior fontanelle), tears of meninges, tentorial tears (common in forceps delivery), haemorrhages, contusions and lacerations of brain.

Neck

This is examined for internal injuries, and trachea for foreign body, froth, mucus, amniotic fluid, etc. The region of the nape of neck deserves special attention.

Thorax

The shape of the chest is observed. Before opening the thorax, the abdomen is opened and the position of the diaphragm noted by passing a finger up to its concave arch.

The lungs are examined for their volume, colour, consistency, weight and the presence of petechial haemorrhages. Hydrostatic test may be performed after taking due precautions as stressed at various points.

The chambers of the heart are opened to see the difference in colour of blood and whether it is normal. Observation is also made regarding the patency of foramen ovale and ductus arteriosus.

Abdomen

Stomach is removed by ligating both ends and tested for floatation. The contents are examined for presence of milk, poison, blood, amniotic fluid, mucus, etc.

The intestines are examined for presence of air and for presence of meconium and its location, which will help to fix the intra-uterine age of the foetus.

Other Viscera

These are examined for their development, any malformations, asphyxial signs and injuries.

Genitals

These are examined for any malformations. The position of the testes—whether descended or where located is noted.

Limbs and Sternum

These should be examined for presence of ossific centres to fix the age of the foetus. Centre of ossification for the calcaneum appears by the fifth month, first division of sternum by the sixth month, talus by the seventh month and lower end of femur by the ninth month. At birth, a centre of ossification is usually present in the cuboid and upper end of tibia.

Centres of ossification may be demonstrated as follows: For the ossific centres in the various divisions of the sternum, the bone is placed on a wooden board and sectioned in its long axis with a cartilage knife, which exposes centres of ossification in the various divisions of sternum. For the ossific centre in the lower end of the femur and the upper end of tibia, the leg is flexed against the thigh and a horizontal incision made across and into the knee joint. A number of cross-sections are made through the epiphysis starting from the articular surface and continuing until the largest cross-section of the ossification centre is reached. In the lower end of the femur, this is seen as brownish-red nucleus that is surrounded by a bluish-white cartilage. The centre appears about the 36th week. A centre of ossification in the upper end of tibia is found in some cases, but in others, it appears after birth. To expose the ossific centres in the bones of the foot, the heel of the foot is placed on a sponge and firmly held by one hand, and with the other hand an incision is made through the inter space between the third and fourth toes and carried downwards through the sole of the foot and heel. Centres in calcaneum and talus, which usually appear towards the end of the fifth and seventh month respectively of intrauterine life, are exposed.

Cause of Death

Once again, to repeat, the concern here is to determine whether the death was due to violence; if due to violence, the injuries incidental to birth trauma or other accidental injuries occurring during or after birth must be excluded.

ACTS OF COMMISSION

Dejected mothers may resort to a variety of ways but some modes of infanticide are more common; depending upon the frequency, the modes may include the following:

Smothering

It is a simple and convenient mode and extremely difficult to prove as it may not leave any evidence, particularly when exercised by pressing the face into a pillow or by closing the nose and mouth by a soft cloth. But the application of more force than usually necessary is likely to leave some pressure marks on the lips and face, especially the bruising of the inner surface of the lips. Sometimes, an area of pallor in an otherwise suffused face may be delineated. Petechial haemorrhages are rarely seen. At occasions, even mucus and squamous respiratory epithelium from the victim may be found in the smothering material.

Strangulation

If strangulation has been effected by a ligature, the ligature material may be found upon the neck but its antemortem nature, i.e. it was applied before death, must be ascertained. Sometimes an explanation that the infant was strangled accidentally by the umbilical cord may be forthcoming from the mother. Examination of the cord may reveal evidence of rough handling in the form of displacement of Wharton's jelly; moreover, there may be some evidence of injuries upon the neck of the infant.

Manual strangulation will present only relatively insignificant bruising or scratches upon the skin surface, though on dissection they may be more prominent. Traditional features like cyanosis, oedema and petechial haemorrhages may be present.

Head Injuries

Head injuries are relatively common. The mother may throw the child to the ground or dash its head against a wall or press under the leg of a *charpai* or sometimes may swing the child by holding legs. Depressed or comminuted fractures of the skull bones with cerebral contusions and/or lacerations, with or without lacerations of the scalp, may be noticed in such cases.

The usual defence may be that the child fell on the ground or the fracture occurred as a result of precipitate labour while the mother was standing erect. Here comes the usefulness of measuring the length of the umbilical cord. The usual length being 50 cm is likely to check a violent fall and further, the labour does not usually result in a forcible and rapid expulsion of the foetus. Cords can get broken during the precipitate birth and that is why it has repeatedly been stressed to examine the severed ends of the cord carefully. Even if the infant happened to fall on the ground, the force is usually insufficient to result in a fracture; none appeared in 183 precipitate labours in the Klein's series.

Precipitate labour is likely to occur in multiparous women with an old laceration of perineum or the woman may show recent rupture of perineum but it may be ruptured in the primipara even if the delivery was normal. Moreover, in a case of precipitate labour, the foetal head will not show caput succedaneum or moulding and the fracture, if it occurs, will usually be fissured and limited to the parietal bones and may radiate to the frontal and squamous portions of the temporal bone.

Fractures of the skull that occur during and as a result of the process of labour usually exhibit certain characteristics, namely, they are not associated with the lacerations of the scalp, usually involve the parietal bones and run downwards at right angles to the sagittal suture and are fissured fractures.

Fractures produced by the forceps may be associated with the lacerations of the scalp but lie at points normally gripped by the instrument and are usually 'gutter' or 'pond' fractures.

Multiple Injuries

Stabbings and cuttings are not common. Stab wounds may be inflicted by using an easily accessible weapon like a pair of scissors, domestic knife, etc. Rarely, weapons like needles and pins may be employed, and careful search for the puncture wound must be carried out before proceeding for the dissection, as the wounds may be concealed puncture wounds inflicted through the fontanelles, inner canthus of the eyes, through nostrils and nape of the neck and therefore might escape notice. The position and nature of wound will obviously be inconsistent with an accidental injury. Incised wounds are again rare, but cases have occurred when instruments such as razor blades have been used. A distinctive feature of such wounds may be that these are well-arranged and parallel because the child can easily be immobilised. These suggest the intention to kill. In such cases, the kind of instrument and nature and extent of injury are important. An extensive incised wound of the throat is highly unlikely to be of accidental origin. Infanticide by decapitation was described by Amoroso (1935). Cutting of the umbilical cord so as to cause exsanguination of the infant could be another mode of infanticide.

Drowning

Infanticide by drowning is unusual. Submersion, however, may be the mode of disposal of the stillborn infants. Most often, it is the household receptacle such as a bowl, bucket or bath but the infant may be taken out and disposed of in any open source of water where the likelihood of delay in the recovery of the infant will add putrefactive changes leading to the difficulties as described at each step. The mother may put the infant into a closet and allege that she gave birth while using it. In such cases, the possibility of precipitate labour (usually seen in multipara with roomy pelvis) and the demonstration of fluid in the respiratory passages and in the digestive tract, resembling the contents of the closet, may be evoked. The usual practice is to kill the infant by suffocation or strangulation and then to throw the body into a cesspool, well, tank or river with a view of concealing the crime. A living body may be similarly thrown, in which case the signs of drowning will be evident.

Poisoning

Presently, it is not a common mode of infanticide. In the olden times, tincture of opium, arsenic, antimony, acids and yellow phosphorus obtained from the matches have been used. Occasionally, coal gas may be used by the mother to include her child in the suicidal pact, as reported in Western countries. Poisoning is a premeditated crime to which the defence of accident or mental imbalance may not stand boldly.

Live Burial

It is extremely rare, though instances have been reported. The case of Berardinelli (1935) is an example. The author cited instances of live burial of infants. In her opinion, the common modes of infanticide in order of frequency were smothering, violence (especially to the head), strangulation, drowning and exposure. Live burial was common in the continent than elsewhere.

ACTS OF OMISSION

The law presumes that a woman who is about to be confined should take ordinary precautions to save her child after it is born. She is guilty of criminal negligence if she fails to do so. Deaths from omission are often rare and almost outside the purview of the doctor. Proof of lack of care immediately after birth will be a matter of witnesses and clinical observations. The circumstances may include the following:

- Omission to make the necessary preparation for the birth of the child (e.g., arrangement for medical aid).
- Omission to tie the cord after dividing it.
- Omission to remove the child from the mother's discharge.
- Omission to protect the child from exposure to cold or heat.
- Omission to supply proper food (deliberate starvation).

A woman must make necessary arrangement for the birth of her child. As soon as she gets labour pains, she must arrange for medical aid. Evidence to the effect that no provision of any kind had been made suggests that she had the intention of doing away with the child.

ABANDONING OF CHILD

Section 317 prohibits the exposure and abandonment of a child less than 12 years of age by the parents or person entrusted with the care of the child. The 'exposure' contemplated under the Section must be one by which some danger to the life of the child may ensue, and the child must have been exposed or left in any place with the intention of wholly abandoning it. The explanation attached to the Section makes it clear that the child must be alive when he/she is exposed or left at any place. If the child dies in consequence, the offender may be tried for culpable homicide or murder as the circumstances may warrant.

Section 318 deals with the concealment of corpus delicti. One way of preventing the detection of the crime of infanticide, and probably the most effective, is the concealment of corpus delicti (i.e. here, concealing the body of the child). This Section punishes secret disposal of the dead body of a child with the intention of concealing the birth of such child. Question may arise that what constitutes a child? Ordinarily, the term has been applied to an infant who has attained certain degree of maturity/development, so that it ceases to be a foetus and becomes capable of independent

existence in the event of its birth. The question is of the facts and may be left for the jury in each case. However, expert opinion is of significance. Here, the words spoken as early as 1869 need mention, "No specific limit can be assigned to the period when the chance of life begins, but it may perhaps be safely assumed that before 7 months the great probability is that the child would not be born alive" (Berriman, 6 Cox, 388 followed in proceedings dated 06.08.1869).

Development of the Foetus

The term developing ovum is used for the first 7–10 days after the conception until the implantation occurs. From 1 week to the end of the second month, it is called embryo and later foetus. Infant is the term applied to the foetus when it is completely born. Neonate is the term applied to the infant in the first 28 days of extra-uterine life. Intra-uterine developmental milestones are as follows (see Table 3.9):

- **At the end of the first month:** The length is about 1 cm. It weighs about 2.5 gm. The eyes are seen as two dark spots and the mouth as a cleft.
- **At the end of the second month:** The foetus is about 4 cm in length and 10 gm in weight. Eyes and nose are recognisable. The hands and feet are webbed. The anus is seen as a dark spot.
- **At the end of the third month:** The length is about 9 cm, and the weight about 30 gm. Nails begin to appear in the form of thin membranes on the fingers and toes. The sex is not yet distinguishable. Placenta is formed and differentiated. The eyes are closed and pupillary membranes appear.
- **At the end of the fourth month:** The length is about 16 cm, and the weight is about 120 gm. Sex is easily recognised. Lanugo is visible on the body. The pupillary membrane is visible. Meconium is seen in the upper part of small intestines (mixture of bile, mucus, and shedded off mucosa).
- **At the end of the fifth month:** The length is about 25 cm, and the weight is about 400 gm. Vernix caseosa appears on the body. It is supposed to protect the foetal skin from amniotic fluid. Fine hair on the scalp are visible. Lanugo is quite distinct. Meconium is seen at the beginning of the large intestine. Centre for the calcaneum usually present.
- **At the end of the sixth month:** The length is about 30 cm, and the weight is about 700 gm. Hair appear on the head. The eyebrows and eyelashes are beginning to form, the eyelids are adherent and the pupillary membrane is still present. The skin is red and wrinkled for want of fat. The testicles lie close to the kidneys, and the scrotum is empty. Meconium is seen in the upper part of the large intestine. Centre for the manubrium and first segment of mesosternum usually present.
- **At the end of the seventh month:** The length is about 35 cm, and the weight is about 1 kg. Subcutaneous fat begins to be deposited. The nails are thick but do not extend to the tips of fingers and toes. The eyelids are open. The pupillary membrane has almost disappeared. The testicles may be found in the external inguinal ring. Meconium is seen in the whole of large intestine. Centre of ossification for talus has appeared.
- **At the end of the eighth month:** The length is about 40 cm, and the weight is about 1.5 kg. Scalp hair is thicker. The skin is red, but not wrinkled, and covered with soft hair. Lanugo has disappeared from the face. The nails reach near to the end of the fingers and toes. The left testicle has descended to the scrotum.
- **At the end of the ninth month (or just before birth):** The length is about 45 cm, and the weight is about 2–2.5 kg. Scalp is covered with dark hair. Lanugo is seen only on the shoulders. Vernix caseosa is present over the flexures of joints and neck folds. Nails have grown over the tips of the fingers and toes. Both the testicles have descended to the scrotum. Meconium is seen at the end of the large intestine. Ossific centre appears at the lower end of femur.
- **At the end of the tenth month (appearance of a full-term mature infant):** The length is about 45–50 cm. The weight is about 3–3.5 kg. The head is about 28 cm in circumference and well-covered with hair. The lanugo is seen only on the shoulders. The skin is covered with vernix caseosa, which is readily seen in the flexures of the joints and neck folds. Vernix caseosa is a white cheesy substance, made up of sebaceous secretion and epithelial cells. Being sticky, it cannot be easily removed. It protects the foetal skin against maceration while in liquor amnii. The pupillary membrane is absent. The nails project beyond the finger tips and to the end of the toes. The umbilicus is situated midway between the pubis and the ensiform cartilage. The umbilical cord is fleshy, with a normal spiral twist and a glistening surface about 45–50 cm in length. Both the testicles have descended into the scrotum. Meconium is present in the large intestine. It is generally expelled in a day or two after delivery. The lower end of the femur shows a centre of ossification. A centre of ossification may be present in the cuboid and the upper end of tibia. Placenta is about 22 cm in diameter and about 700 gm in weight.

Foeticide

In view of the declining sex ratio because of female foeticide, it was considered necessary to bring out a legislation to regulate the use of and to provide deterrent punishment to stop the misuse of diagnostic techniques. Ultimately, the **Prenatal Diagnostic Techniques (Regulation and Prevention of Misuse) Act** was passed by both Houses of the Parliament

Table 7.2 Penalties as Provided under the Preconception and Prenatal Diagnostic Techniques (Prohibition of Sex Selection) Act

Concerned personnel	Punishment
Breach by any service provider like unit owner(s), medical professional(s), employee, etc.—Section 23(1)	Imprisonment that may extend to 3 years and fine that may extend to 10,000/- AND (in case of subsequent conviction) imprisonment that may extend to 5 years and fine that may extend to 50,000/-
Medical professional (RMP)—Section 23(2)	Appropriate authority to report the name of the RMP to the State Medical Council for taking necessary action including: • suspension of registration if charges have been framed by the court and till the case is disposed off • removal of the name from the register for a period of 5 years in the event of conviction for the first offence AND permanently for the subsequent offense
Persons seeking the aid of any unit for sex selection/sex determination on any pregnant woman—Section 23(3)	Imprisonment that may extend to 3 years and fine that may extend to 50,000/- AND (in case of subsequent offence) imprisonment that may extend to 5 years and fine that may extend to 1,00,000/-
Contravention of the provisions of the Act or the Rules for which no specific punishment is provided elsewhere in the Act—Section 25	Imprisonment that may extend to 3 months or fine that may extend to 1000/- or both AND (in case of continuing contravention) additional fine that may extend to 500/- for every day during which such contravention continues

and received the assent of the President on 20th September, 1994. Later, by amendment during 2002, the nomenclature of the Act was amended and now it stands as **The Preconception and Prenatal Diagnostic Techniques (Prohibition of Sex Selection) Act, 1994**. (This necessity emerged out of the fact that in the present set-up, sperms can be processed in the laboratory to effect separation of X and Y chromosomes, thus ensuring the birth of a male child, i.e. sex selection takes place even before conception. Hence, the amendment in the nomenclature of the Act was carried out.)

Law's Difficulty in Dealing with Foetus

The law has difficulty in dealing with the foetus. This is because of the controversy of focussing the evidence upon 'conception' or upon 'live birth' or upon the interim point at which the foetus becomes 'viable'. Furthermore, literature purports to indicate that conception is a 'process' overtime, rather than an event, and introduction of new medical techniques like menstrual extraction, implantation of embryos, artificial insemination and even artificial wombs has made the issues much more complicated. There has been a tendency in the past that in cases where the abortion was caused by the action of another person, to compensate the mother or the family in limited way, i.e. they were used to be compensated for their mental and physical anguish and any impairment of health of the mother occasioned by the miscarriage but not for the productivity of the aborted foetus as if it were a potential wage earner. However, on 5th March 2007 Maharashtra State Commission in the case of Kanta Mohan Lal Kotecha vs. United India Insurance Company ruled that the claim in respect of the unborn child was maintainable provided certain requirements were satisfied. The forum laid stress upon the concept of 'viability', i.e. whether the foetus had reached the stage of 'viability' at the time of the accident and thereby attained the status of a 'potential person' who could be able to live outside the mother's womb albeit with artificial aid. Penalties as provided under the Act are summarised in the Table 7.2 so as to apprise the readers of gravity of situation in the event of violation of the Act.

8 CHAPTER

Thermal Deaths

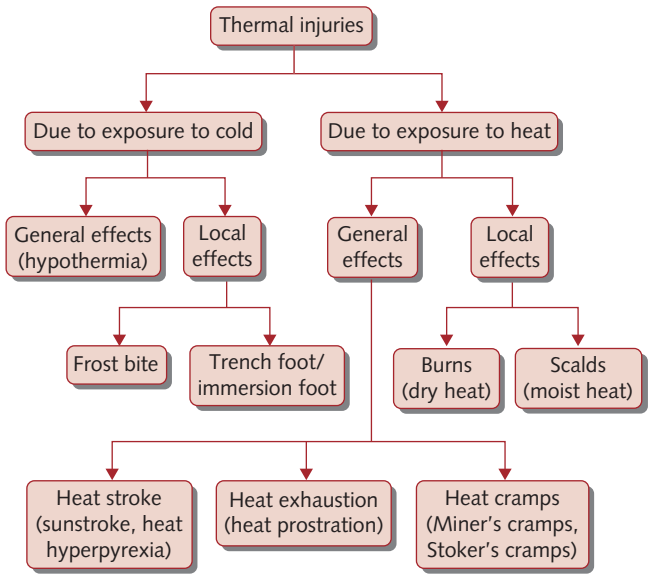
After going through this chapter, the reader will be able to describe: Heat regulation, systemic hyperthermia (heat cramps, heat exhaustion and heat stroke) | Character of burns produced by various agents | Different classification of burns | Rule of nine | Causes of death in burn | Nature of burn injury in the absence of death | Age of burn injury | Autopsy findings | Medicolegal aspects of death due to burns | Antemortem and postmortem differentiation of burns | Some legal provisions in relation to dowry death | Scalds and their medicolegal aspects | Differentiation of injuries due to dry heat, moist heat and chemicals | Pathophysiology of hypothermia | Circumstances of injury due to cold | Autopsy findings in death due to cold

Thermal deaths are those that result from the systemic and/or localised exposure to excessive heat or cold. The main factors determining the deleterious effects of heat are the temperature (i.e., the intensity of heat applied) and the duration for which it is applied. This is amply clear from the observations of Moritz and Henriques, who found that the lowest temperature that would produce damage was 44° C, though the time required to produce burn was of the order of about 5 hours, whereas if the object was at 60° C, it required only 3 seconds to cause burn.

Systemic Hyperthermia (Flowchart 8.1)

Human body is far more susceptible to elevation than to diminution of temperature. Neurons in both the pre-optic anterior hypothalamus and posterior hypothalamus receive two types of signals—one from peripheral nerves that reflect warmth/cold receptors and the other from the temperature of the blood bathing the region. These two types of signals are integrated by the thermoregulatory centre of the hypothalamus to maintain normal temperature. The normal body temperature is maintained despite environmental variations because the thermoregulatory centre balances the excess heat production derived from metabolic activities in the muscle and liver with the heat dissipation from the skin and lungs, etc. [**Endogenous/febrile hyperthermia** implies an elevation of body temperature that exceeds normal daily variation and occurs in conjunction with an increase in the hypothalamic 'set point'. (This set point has been considered as 37.1° C. When the temperature goes above or below this point, drastic changes occur in the rates of heat loss or production so that the body temperature re-approaches 37.1° C.) **Exogenous/Nonfebrile hyperthermia**, on the other hand, implies an unchanged setting of the thermoregulatory centre in conjunction with an uncontrolled increase in the body temperature that exceeds the body's ability to lose heat. For example, work or exercise in hot humid atmosphere can produce heat faster than peripheral mechanisms can lose it. Hyperthermia is often diagnosed on the basis of the events immediately preceding the elevation of body temperature.]

Depending upon the progressive order of severity, effects of excessive heat may be divided into heat cramps, heat exhaustion and heat stroke. Ultimately all result from the loss of equilibrium between heat load (metabolic and climatic) and



Flowchart 8.1 Categorisation of thermal injuries.

the capacity of the body to eliminate/dissipate heat. Conditions with differentiating features are given in Table 8.1.

Burns

The term **'burn'** denotes a variety of conditions of which the local effects of dry heat are the classical examples. There are differences in the circumstances and the resulting destruction of tissues and therefore some separate account for several kinds of burns is being furnished before we march on to the 'classical burns' because the conclusion as to the agent producing the burns may be of importance as derived from Sections 324 and 326 of the Indian Penal Code wherein it is laid that causing of 'hurt' or 'grievous hurt' by some specified means is punishable more severely than when such means have not been employed. Among the means are also included means like 'fire or any heated substance' or any 'corrosive substance' or 'explosive substance', etc. under these Sections.

BURNS BY X-RAYS AND ULTRAVIOLET RAYS

Burns resulting from X-rays are usually from fault exposure and vary from mere redness of the skin to dermatitis with shedding of the hair and epidermis and pigmentation of the surrounding skin. Severe exposure may produce vesicles and/or pustules, which often form sloughing ulcers on bursting and

Table 8.1 Summary of Differentiating Features of Various Manifestations of Hyperthermia

Heat cramps/miner's cramps/stoker's cramps	Heath exhaustion/heat prostration/heat collapse	Heat stroke/sun stroke/heat hyperpyrexia/thermic fever
Painful spasm of voluntary muscles, especially those of the extremities and the abdominal wall. Usually occurs in persons who do heavy muscular work in high temperature and humidity.	Prodromal symptoms usually comprise of increasing thirst, nausea, vomiting, headache, irritability with feeling of exhaustion. Usually occurs in persons after several days of hard work in closed environment and under moderately high temperature.	Often rapid in onset. May be preceded by headache, nausea, weakness, myalgia, confusion, disorientation, locomotor disturbances, etc. Usually occurs in persons undergoing strenuous work in the open fields under conditions of high temperature with lack of acclimatisation.
	Skin is moist and clammy. Pale face. Pulse is thready. Low blood pressure. Breathing is sighing. Temperature normal or sub-normal. Fainting and manifestations of peripheral vascular collapse may ensue.	Skin is dry and hot. Flushed face. Pulse is fast, thready. Hypotension. Breathing is rapid, deep. Very high fever (may reach 105°–107° F). Varying manifestations of cerebral deficit usually constitute an important feature.
Cause is stated to be loss of sodium and chlorides from the body.	Cause is stated to be water and/or salt loss from excessive sweating that is inadequately replaced. May be similar in presentation to insulin shock, alcohol/drug abuse or hypovolaemia from occult blood loss.	Cause is stated to be failure of thermo-regulatory systems resulting in severe hyperthermia and multisystem damage in the form of neurological deficit, DIC, rhabdomyolysis, renal and hepatic failure, etc. First 24 hours are most critical. Autopsy findings lack specificity. Postmortem caloricity may be a distinct feature. Rigor mortis appears early and passes off quickly. Increased body temperature causes diminished peripheral resistance leading to increased venous return, and thus triggering high output cardiac failure. However, ultimate manifestations attending death are considered to be deprivation of oxygen to the tissues rather than the direct effect of increased heat load.
Management consists in maintaining an adequate salt intake in food or in saline drinks. Source needs be removed.	Management consists in making the patient lie in cool area. Fluids and salts need be replenished. If needed, intravenous fluid may be administered.	Management consists in shifting the patient to the cooler place and hospitalisation. Maintaining airways and circulation. Intravenous fluids and electrolyte therapy.

Note: Death from general effects of heat seldom becomes subject of medicolegal enquiry/investigation. In cases of sun stroke, the victim may pass into the stage of suspended animation, inviting caution in pronouncing death. Unfavourable working conditions at industrial unit/working place leading to hyperthermia in an employee can attract provisions of Workmen's Compensation Act.

usually take a long time to heal. Ulcers on healing form radiate scars, and the surrounding skin shows pigmentation. However, these are rare these days because of improvements in the equipment, use of protective screens and the precautions taken by the operator. Occasionally, late reaction may be after months or even years can occur that presents like a bruise, followed by extensive deep sloughing of the tissues and delayed healing. Burns by radium are similar to those produced by X-rays. Improper use of ultraviolet lamps can cause ill effects. A short exposure of parts normally covered by clothing may be sufficient to produce hyperaemia and irritation, which may subside within a few hours. Overdosage with ultraviolet light can sometimes lead to severe and persistent dermatitis. Same thing holds good with the exposure to infrared rays. Similarly, the sunrays can also cause dermatitis, particularly in sensitive and fair complexioned individuals.

BURNS BY ELECTRICITY AND LIGHTNING

Electric burns may be caused either due to contact with an electric circuit (i.e., the electric mark) or burns produced by a flash, which usually accompanies a short circuit (i.e., thermal burns or flash burns). The latter are essentially the same as burns produced by a flame, and their gravity is evaluated in terms of first, second or third degree. In case of the former, i.e. the 'electric mark', the lesion appears as a round or oval shallow crater, bordered by ridge of skin having raised margins. The skin looks distinctly pale as it is produced by conversion of electricity into heat within the tissues. That is why they are sometimes termed as 'endogenous burns' to distinguish them from the flash burns or 'exogenous burns'. Burns from lightning may appear in the form of 'arborescent markings' on the surface of the skin, looking like branches of a tree.

BURNS BY CORROSIVES

Burns produced by corrosive acids and alkalies are usually similar in character. 'Corrosion' means to destroy something by chemical action. The lesions often show distinctive stains and result in eschars, which are soft, moist and readily slough away. In these burns, the red line of demarcation is absent, hair are not scorched nor are any vesicles formed. In these chemical burns, the characteristic appearance along with stains, stains on clothing and chemical analysis of the tissue and clothing will establish the diagnosis. These burns usually may not cause death yet they may cause permanent disfiguration of head and/or face leading to grievous hurt. Involvement of eyes may further add to the problem.

BURNS BY DRY HEAT (FLOWCHART 8.1)

They are usually the result of contact with a naked flame or the heated elements of an electric wire or contact with hot metals or glass. These substances outwardly may appear to be harmless but may be at dangerous temperatures, which the victim realises only after the mishap. The worst burns in the domestic circumstances are those that usually occur following the ignition of clothing that have come in contact with the coal, gas or an oil stove. The lower part of the clothing may catch fire and within a few seconds the entire garment is usually affected. The burns will usually commence on the side of the leg immediately opposite to the part of the garment that first caught fire. Wilson's classification is widely acclaimed and therefore some details are given below (see Table 8.2).

Epidermal

Erythema (reddening) and blistering without involvement of dermis are the characteristics of the epidermal burns. There is capillary dilatation and transudation of fluid into the tissues resulting in swelling. The blister thus formed contains albuminous fluid and is covered by avascular whitened epidermis and surrounded by a zone of hyperaemia. If small, the blister may resorb due to absorption of the fluid and the raised dead epidermis is later on shedded to be replaced by the new growth from the periphery of the burn area. Such blisters are painful and heal without scar formation.

Dermo-epidermal

Here, the burns involve full thickness of skin, including hair follicles and sweat and sebaceous glands. Hence, they are extremely painful as they expose and affect the sensory nerve endings. They heal with scar formation as they damage the full thickness of skin.

Table 8.2 Different Classifications of Burns

Degree of damage	Dupuytren's	Hebra's	Wilson's
Erythema/redness	1st degree	1st degree	Epidermal
Vesication	2nd degree	1st degree	
Damage to superficial skin	3rd degree	2nd degree	Dermo-epidermal
Damage to whole skin	4th degree	2nd degree	
Damage to muscles	5th degree	3rd degree	Deep
Damage to the deeper tissues including bone(s), etc.	6th degree	3rd degree	

Deep

These involve destruction of the deeper tissues below the skin. Therefore, these can be of any severity, varying from damage to the subcutaneous tissues to the involvement of muscle, bone, etc. The burnt parts may even be completely charred. These burns are relatively painless as the nerve endings are completely destroyed.

RULE OF NINES

The body surface involved is traditionally determined by the surgical 'Rule of Nines', though for the purposes of autopsy reports a detailed description of the burnt areas is essential (Table 8.3). According to this rule, the percentage of area attributed to the different parts of the body surface is as described in Table 8.3.

'Rule of Nines' was advocated by Wallace (1951). However, explanation furnished by Lund and Browder (1944) needs to be used for more accurate estimate, particularly in children, whose proportions are quite different from those of adults (Fig. 8.1 and Table 8.4).

The prognosis of the burn injuries depends more upon the extent of body surface involved than upon the degree/depth of burns. A third degree burn of a limb, though may result in a great disability but may not prove fatal, whereas a first degree burn involving 40–50% of body surface is nearly always incompatible with survival. However, infants, very young children and old people appear to be more vulnerable to lethal complications. Older children and adults can withstand burn injury better. Women are more susceptible than men. A useful rule of thumb for estimating total surface area involved by a scattered burn injury is the 'palm of the hand rule', viz., 'the surface area of the patient's palm is roughly 1% of the total body surface area.'

Table 8.3 Percentage of Burn Surface According to 'Rule of Nines'

Head and neck	– 9% of the whole body surface
Front of chest	– 9% of the whole body surface
Back of chest	– 9% of the whole body surface
Front of abdomen	– 9% of the whole body surface
Back of abdomen	– 9% of the whole body surface
Right upper limb	– 9% of the whole body surface
Left upper limb	– 9% of the whole body surface
Front of right lower limb	– 9% of the whole body surface
Back of right lower limb	– 9% of the whole body surface
Front of left lower limb	– 9% of the whole body surface
Back of left lower limb	– 9% of the whole body surface
Pudendal area	– 1% of the whole body surface
Total	=100%

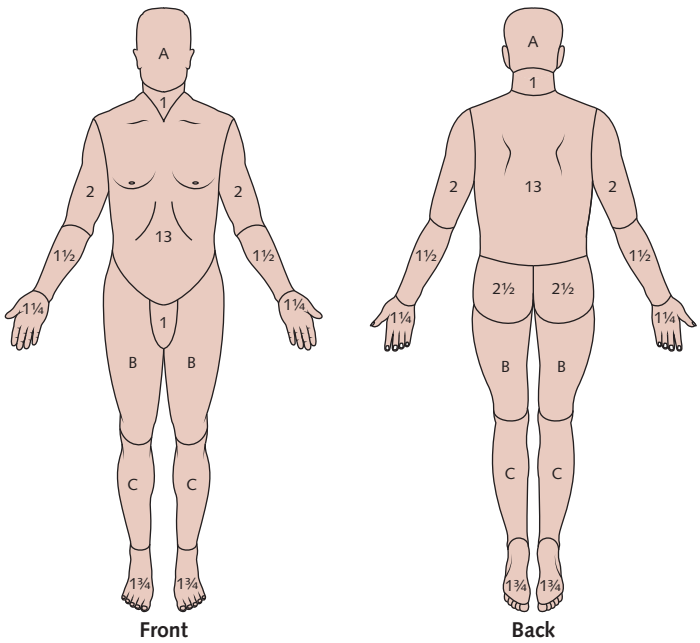


Fig. 8.1 Relative percentage of body surface area affected by growth.

Table 8.4 Lund and Browder Chart for Estimating Size of the Burn as a Percentage of Body Surface Area

Area	Age (yr)					Adult
	0	1	5	10	15	
A=1/2 of head	9½	8½	6½	5½	4½	3½
B=1/2 of one thigh	2¾	3¼	4	4½	4½	4¾
C=1/2 of one leg	2½	2½	3¾	3	3¼	3½

Causes of Death in Burns

IMMEDIATE CAUSES OF DEATH

Shock

Death may occur within a few hours due to primary or neurogenic shock or it may occur within 24–48 hours due to secondary or hypovolemic shock, which is principally due to loss of fluid from the burned surfaces due to increased capillary permeability. Shock can also occur merely from fright or extreme fear before the individual is affected by burns if the heart is debilitated or weak.

Suffocation

Victims removed from houses destroyed by fire or from any other building destroyed by fire are often found dead from suffocation due to inhalation of smoke, carbon monoxide or any other irrespirable gases. Toxic inhalation of combustion products

of synthetic materials may accentuate the toxic effects of the carbon monoxide. Burning of plastic materials may produce toxic and potentially lethal gases like hydrogen cyanide and oxides of nitrogen. Burning of wool or silk may yield ammonia, hydrogen cyanide, hydrogen sulphide and oxides of sulphur. Death may also occur due to mechanical asphyxiation, i.e. traumatic asphyxia in case of a collapse of a house or building resulting from outbreak of fire.

Accident/Injuries

Death may result from an accident occurring in an attempt to escape from a burning building or by injuries sustained from the falling of walls or other structures upon the body.

DELAYED CAUSES OF DEATH

Toxaemia

It occurs from absorption of toxic products from the burned areas. Death usually occurs in such cases after 4 or 5 days or sometimes even later. Death occurring on 3rd or 4th day is usually due to irreversible hypotension and renal failure due to acute tubular necrosis.

Inflammatory Complications

Inflammatory complications of serous membranes and internal organs such as meningitis, bronchopneumonia, pleurisy, peritonitis, pericarditis and so on can be responsible for delayed deaths. A less common complication is the duodenal ulcer, also known as Curling ulcer. They may occur in association with head injuries too. Gastric ulcers occurring due to burns have been named as Dupuytren ulcers. Gastric ulcers may occur within a day or two of burning, but duodenal ulcers appear late. Various factors attributed to their production may be local ischaemia, infection and effects of stress on adrenocortical function.

Fatal Period

Most of the deaths from burn injuries occur from shock within about 24–48 hours. Death due to toxaemia occurs usually within 4–5 days. The first week happens to be the most critical period. In suppurative cases, death may ensue after a few weeks.

NATURE OF BURN INJURIES IN THE ABSENCE OF DEATH (Fig. 8.2)

In a case where death has not occurred, burns may constitute simple or grievous hurt as the case may be. Burns of the first degree, if not extensive, constitute simple injury. Burns constitute grievous hurt if they result in permanent disfiguration of head or face or permanent privation of sight of either eye or permanent impairment of powers of any member or joint, etc.



Fig. 8.2 The victim received the burn injuries while saving his wife and children from the burning *jhuggi*. Medication by some antibiotic cream is evident.

Lastly, they may also fall under the category of last clause of grievous hurt, i.e. if the individual has suffered 'shock' so as to endanger life or if he/she has been in severe bodily pain or bedridden and thereby incapacitated to follow ordinary pursuits of life for a period of 20 days. The kind of weapon in such cases will obviously be 'dry heat'.

Age of the Burn Injury

Sometimes the question may arise as to the time of infliction of burn injuries and further, in case of several burns on the same individual, the issue may be that whether they were inflicted simultaneously or at different times. Different stages of the repairative process may provide the answer.

Erythema (redness) appears immediately after the burn. Vesication usually develops within about 2–3 hours. The exudate begins to dry in 12–24 hours, and within 48–72 hours, it forms a dry brown crust. The red inflammatory zone disappears in 36–72 hours. If infection happens to occur, pus is formed in 2–3 days but not before 36 hours. Superficial sloughs usually separate out from the 4th to 6th day and deeper sloughs within about 2 weeks. After this period, granulation tissue begins to cover the area and a scar is formed after several weeks or even months depending upon the amount of suppuration, sloughing and depth and extent of the burn.

Autopsy Findings

The remnants of clothing, particularly the portions that were in rigid contact with the body and those that got pressed against the body surface during the process of burning should

be looked for and removed carefully. They should be examined for presence of any characteristic smell like that of kerosene, petrol or some other combustible substance. It is possible to recover and identify hydrocarbons from clothing, even when they are severely burned. Clothing should, therefore, be preserved and transmitted to a Forensic Science Laboratory/ chemical examiner through the police, after sealing them in glass containers and not in plastic bags.

EXTERNAL FINDINGS

Before opening the body, a careful record, supplemented by photographs (if possible), should be made of the **distribution of burns**. If the body is severely burnt, then all the surface of the skin might have been damaged, making it impossible to determine the antemortem or postmortem nature of the burns from the external examination as no skin surface is available to observe for the presence or absence of vital reaction. However, if some undamaged skin is present at places adjacent to the damaged areas, the appearance of vital reaction in the form of reddened borders adjoining the burnt areas is an important finding towards antemortem nature of burns. Burns inflicted after death present a distinctive appearance, usually showing no red margins and dried scorching of the skin surface. The scalp hair and other body hair may be singed. There may be blackening of the extensive body surface. In lesser degree burns, ends of the hair may be 'clubbed'. Here the keratin melts at the distal end nearing the heat and resolidifies on cooling forming a terminal knob on the shaft and thus imparting 'clubbed' appearance. The burns caused by kerosene oil are usually very severe and are identified from its characteristic odour and the sooty blackening of the parts. Blood-tinged froth may be seen at the mouth and nostrils owing to pulmonary oedema provoked by heat irritation of the respiratory passages and the lungs. The tongue usually protrudes and may be scorched.

When the body has been exposed to substantial heat, it will almost always assume the appearance known as **'pugilistic or boxer's attitude'**. This occurs due to coagulation of the muscle proteins resulting in contraction of the muscle fibres. As the flexors are bulkier than the extensors, they contract more and force the body to assume such an attitude. However, this attitude does not speak anything about the fact that whether the victim was alive when the fire started or not (Fig. 8.3).



Fig. 8.3 Photograph of a victim with burn injuries showing 'pugilistic attitude'. Also note blackening and singeing of the hair. Appreciate the areas showing remnants of clothing usually available, which need be removed and sent to FSL.

Another finding of importance and needing careful evaluation is the appearance of **heat ruptures**, usually seen over the area of severe burning and over fleshy areas like calves and thighs, etc. These are the splits occurring in the skin due to contraction of the heated and coagulated tissue and the resultant breaches may simulate incised or lacerated wounds. They can be differentiated from the antemortem lacerations by their appearance, distribution as written above and by associated findings like bleeding into the deeper tissues. In such heat ruptures, absence of blood clot or absence of infiltration of blood into the cellular spaces and the presence of intact blood vessels and nerves stretching across the floor of the ruptures will be sufficient to indicate their spurious nature. Sometimes, the charred and brittle skin due to effect of heat may undergo cracks while the body is being removed from the house or building destroyed by fire or during transportation of the body to the mortuary or within the mortuary, if handled carelessly.

INTERNAL FINDINGS

On opening the body, the depth of heat coagulation of the tissues should be observed. It is generally considered that a badly charred body with no heat changes beyond the subcutaneous tissues indicates a short exposure to intense heat, whereas heat coagulation of deeper muscle proteins without severe external charring suggests that the body was exposed to moderate heat over a long period. However, there is no reliable method to evaluate the duration of burning as it depends upon multitude of factors and the opinion must be given with due reservations.

Internal **organs are usually congested** but may be coagulated, firm and pallid and occasionally the body cavities might have given way by partial destruction of their walls. The blood is usually bright pink in colour if death has occurred due to inhalation of carbon monoxide (see discussion under Antemortem and Postmortem Nature of Burns). The mucosa of stomach and duodenum is frequently reddened and may show ulcers as described earlier.

Internally, **the finding of 'heat haematoma', if revealed,** deserves special evaluation. This condition has an appearance of extradural haemorrhage but actually is an artefact, occurring only in the circumstances where the head has been exposed to intense heat and, obviously, will be maximum in amount opposite to the site of greatest external damage to the skull. Therefore, the usual sites of this haematoma will be frontal or occipital regions. Other characters of such a haematoma may include chocolate brown colour of the blood, the clot is soft, friable and presents a honeycomb appearance due

to presence of bubbles of steam produced due to boiling of blood by external heat. The mechanism of its production is obscure. Possible sources may be that either it arises from venous sinuses or the blood may be squeezed out of the diploic space through emissary venous channels. It is a post-mortem phenomenon or may be produced agonally when the victim is already unconscious either due to inhalation of carbon monoxide or shock due to severe burns (see discussion under Antemortem and Postmortem Nature of Burns).

Most important findings are to be observed in the respiratory passage and blood, which will be extremely clinching towards the fact that whether the victim was alive when the fire commenced. The tongue, fauces, larynx, trachea and bronchi are usually inflamed and contain soot often intimately mixed with mucus. If the deceased had inhaled very hot gases or fumes or rarely the flame itself, then the mucosa over the tongue and larynx may be oedematous and exhibit blistering or shredding. Sometimes, some vomitus (presumably due to bouts of coughing) may also be present in the respiratory passages. Carbon impregnated mucus may be swallowed and found in the oesophagus and/or in the stomach.

Together with the soot, the inhaled smoke usually contains some carbon monoxide, which is therefore absorbed by the blood. The **presence of carbon monoxide in the blood** is often obvious from the bright pink appearance of the blood, the muscles and even the cut surfaces of the organs (see discussion under Antemortem and Postmortem Nature of Burns.)

MEDICOLEGAL CONSIDERATIONS

The death investigation of a person exposed to excessive heat may warrant the following medicolegal considerations.

Identity of the Deceased

Identification of the deceased is done on the usual lines and may not be a problem in routine cases when the burn injury has left the body in its usual configuration. However, when different parts of the body are charred including the face, it poses a problem. The internal examination may contribute significantly, especially in cases where the deceased has been shown to have had diseases like myocardial infarction, pulmonary tuberculosis or some surgery such as appendectomy, hysterectomy, nephrectomy, etc. The detection of pacemaker, valve implant, etc. may be especially helpful towards identification. The subject of identification is lengthy and only certain points, peculiar to the burns, will be discussed.

One must look for any **remnant clothing or any other article** near or on the body. Even on mostly charred body, fragments of clothing may be found in the folds of the skin or in the flexures of armpits or inguinal regions or some traces of jewellery may be found.

X-raying the entire body carries unique importance. It will help to demonstrate any old bony deformity or presence of any foreign body like bullet or any calcified object. Comparison of the X-rays of the corpse with previous X-rays, taken sometimes back in the hospital, may prove helpful in establishing identity. If the face is badly charred by fire, the front teeth may also be damaged but the premolars and molars lying farther back under the cheeks may be saved and can be examined. **In a badly charred victim**, dental identification is the best hope as the teeth are relatively resistant to fire. X-rays of the teeth may reveal the evidence of some root canal fillings or some other treatment. When heat contractures have locked the jaw rigidly and visual examination is precluded then proper dissection may be carried out and the upper jaw may be excised. (Webster was not able to destroy the evidence of the identity of his victim when he attempted to dispose of the body of Dr. Parkman in a laboratory furnace. The dentures of the victim were sufficiently preserved to establish the identity.) A fire in the open, as in a field, may not usually permit complete combustion of the human body as was observed in case of attempted disposal of Hitler's body at the end of World War II. Though the gasoline was used as an accelerant, yet in spite of widespread charring, positive dental identification could be made. (Bezymenski L. *The Death of Adolf Hitler,* New York, Harbrace World, 1968.)

In occasional cases (in case of an edentulous body with extremely burnt extremities), the identification may remain presumptive and may pose medicolegal problems. However, one must keep in mind that regardless of the surface destruction by fire making the sight recognition of the body unreliable, internal organs (particularly uterus and prostate due to their thick musculature and relatively well-protected pelvic location) may be available and if not, then bones, especially teeth, dentures/parts of dentures may hold the key for solving the issue.

CASE: Burns vis-á-vis Identification—Gravity towards Criminality thereof (Vide Communication from Dr. Manpreet Kaul et al.)

On 14.02.2002, a lady R lodged a complaint to the police alleging that her daughter (aged about 17 years) who as usual had gone to do work in the house of some H Singh on 13.02.2002 had not returned back. When R visited H Singh's house to ask about the whereabouts of her daughter, he told R that the girl had left his place. R getting panicky, curiously raised queries about one dead body lying in the verandah. To this, H Singh satisfied her by telling that the dead body was of his wife who had died of burns while working in the kitchen. However, R asserted that she was able to recognise the body from the spared hand (carrying specific colour of the nail polish) and from the teeth, etc. (Such structures being relatively resistant to fire due to their hard composition provide best hope for identification. This has been duly stressed in the text of the chapter too.) These developments made her to inform the police. The police recorded the FIR under Section 302/201/34 IPC and took the body in custody from the cremation ground. The

postmortem was conducted. In the columns of 'name' and 'age' of the deceased, the Inquest Papers showed:

- Name of the deceased as Jagdish Kaur/Kiran', i.e. the wife of H Singh/daughter of R.
- Age of the deceased as 'about 27 years/17 years', i.e. the age of the wife of H Singh/daughter of R.

Various issues were resolved by exercising due precautions as follows:

- Dead body was subjected to 'radiography' before proceeding for autopsy (considering radiological and dental findings, the age of the deceased was estimated to be about 17 years).
- Videography of the entire procedure was got conducted so as to escape any element of bias, which may creep up sooner or later.
- Further evidence of age was afforded by findings of the genital organs, viz. intact hymen, vaginal rugosities and uterine size being $3^2 \times 2^2 \times 1^2$ with convex walls and triangular cavity.
- Vaginal swabs sent to the Chemical Examiner showed negative results.

Antemortem and Postmortem Nature of Burns

Indications that the victim was alive at the time of fire are discussed below (Table 8.5).

PRESENCE OF SMOKE IN THE AIR PASSAGES

Presence of greyish-black or black amorphous material embedded/adherent to the mucosae of larynx, trachea and bronchi indicates that the victim inhaled smoke and hence was breathing during the fire. Finding of soot/smoke in the superficial areas like mouth and/or nose etc. does not serve any meaningful purpose towards the diagnosis. The quantity of soot/smoke in the air passages is influenced by the type of fuel and fire, amount of smoke produced depending upon the degree of combustion, the nature of articles burnt and the period of survival of the victim. Adherence of soot to the oesophageal and gastric mucosae indicates that it was swallowed and accordingly shows that the life existed at the time of fire. Absence of this finding of smoke may suggest two possibilities, viz.:

- Either the victim died before the fire commenced or
- Death was so rapid that it prevented any inhalation of smoke by the victim. Such a situation may occur in extremely hot fires or rapid flash fires or explosions.

EVIDENCE OF THERMAL INJURY OF THE RESPIRATORY TRACT BY FUMES/HOT GASES

Death may sometimes be occasioned by inhalation of fumes or hot gases, causing thermal damage to the respiratory mucosa and acute laryngeal oedema. If oedema develops rapidly as may occasionally be encountered when the victim happens to inhale the flame or superheated air, the laryngeal region may be damaged and sudden asphyxiation may ensue. Most of the burns in such rapid deaths will usually be of postmortem nature and severe postmortem burning will obliterate the few surface burns, which might have been sustained by the victim up to the moment of death. Under such circumstances where no antemortem surface burns are appreciable, it may be a logical conclusion to attribute death to asphyxia due to suffocation originating from the thermal damage to the respiratory passage.

ELEVATED BLOOD CARBOXYHAEMOGLOBIN LEVELS

Smokes differ in the composition depending upon the type of material undergoing combustion, the amount of available oxygen to supplement fire and the temperature attained by the fire. However, in most of the fires, particularly the house fires, most of the carbon of the organic material such as timber, furniture, etc. leads to production of carbon monoxide along with other gases. Slow smouldering fires are likely to produce more carbon monoxide as may be seen in the confined places than the open fires.

Saturation of haemoglobin with carbon monoxide varies from case to case, as it is dependant upon a number of factors like concentration of the carbon monoxide in the inhaled air as outlined above, the duration of exposure, rate and depth of respiration of the victim, haemoglobin content of the blood of the victim and the local variations in the draughts. Activity of the individual within an atmosphere containing carbon monoxide also increases the rate of absorption. Therefore, the interpretations of results of carbon monoxide blood analysis in the fire victims must be evaluated in concurrence with the circumstances of combustion, the anatomical findings and the factors attributable to the prior state of body of the victim.

More than 10% blood haemoglobin saturation with carbon monoxide in bodies recovered from fires usually indicates that the victim inhaled smoke and hence was alive at the time of fire. This level of more than 10% blood haemoglobin saturation with carbon monoxide indicating that the victim had inhaled smoke and therefore was alive should be particularly adhered to in case of **chain-smokers** because they could build up a level of 8–10% merely due to cigarette smoking. Therefore, **in a nonsmoker** even such a blood level of carbon monoxide may be considered as an evidence of smoke inhalation and consequent existence of life when the fire started. When death results solely from carbon monoxide, carboxyhaemoglobin levels may range as high as 50–60% haemoglobin saturation except in old and debilitated individuals where deaths have been reported at much lower saturations. When death occurs extremely rapidly from a violent explosion or flash fire or an intensely hot quickly

Table 8.5 Differences between Antemortem and Postmortem Burns

Features	Antemortem burns	Postmortem burns
Line of redness	Most often present around the injured burn area	Not so
Vesicles	Contain serous fluid with high proportion of albumen and chlorides. Base of the blister is red and inflamed	Contain air mostly; if any fluid present, it comprises of very little albumen, no chlorides. Base of the blister is dull, dry, hard and yellow
Evidence of inflammation	Inflammatory oedema with signs of repairative and repair processes (depending upon the survival period—leucocytic infiltration has been reported to occur at 6 hours)	No such evidence of reaction is ever noticed
Presence of carbonaceous deposits/soot in the respiratory tract	Indicative of death from suffocation following antemortem burns	Not present
Presence of carboxyhaemoglobin in blood	When present with other features of antemortem burns, it is highly suggestive and diagnostic (it has been reported that there is no significant difference in the blood carboxyhaemoglobin levels drawn from peripheral versus heart sites)	It will be absent since artificial elevation of carboxyhaemoglobin saturation level in a dead person simply by being in or near a fire is unlikely as the gas cannot diffuse through the skin or otherwise be absorbed by a dead body
Enzymatic activity	Increased enzyme reaction in the periphery of antemortem burn (it is time-related, viz.: tissue cathepsin—immediate, leucine aminopeptidase—2 hr, acid phosphatase—3 hr, nonspecific esterase—3/4 hr and alkaline phosphatase—4 hr)	No such activity/reaction will ever be noticed

engulfing blaze, little or no carboxyhaemoglobin may be present in the blood of the victim because respiration ceased too rapidly to allow inhalation of any appreciable quantity of the gas.

A high percentage of carboxyhaemoglobin in the blood may explain why the victim was not able to escape. As written already, the activity of the individual within the atmosphere containing carbon monoxide increases the rate of absorption and may cause immediate loss of consciousness even when the levels are well below the lethal level. Hence, one may encounter persons lying dead in the immediate vicinity of fires, who may have none or only a few surface burns but the blood revealing a lethal saturation of carboxyhaemoglobin.

Another remarkable advantage of estimation of carbon monoxide in the blood may be seen in the circumstances where the autopsy reveals injuries or haemorrhage. Here, estimating the quantity of carbon monoxide in the blood composing the haemorrhage or surrounding the injury and comparing it to that of its concentrations in the circulating blood may lead to disclosure of the circumstances of death. Thus, if finding of 'extradural haematoma' is encountered at autopsy, its origin (i.e., whether it is a true traumatic lesion that occurred before the fire began or a fake heat haematoma formed from the blood that has been squeezed out of the venous sinuses or emerged out of the diploic space due to exposure of the head to intense heat) may be determined by demonstrating presence of carbon monoxide in the blood. In the former case, the blood from the haematoma will be free from carbon monoxide, whereas in the latter case, the haematoma will show the presence of carboxyhaemoglobin if the victim had absorbed this gas during the process of death in the fire. Hence, blood carbon monoxide levels must be evaluated in all victims known, alleged or suspected of having succumbed to death in a fire. Fluid blood may be obtained from chambers of the heart or major blood vessels and put in chemically cleaned, tightly stoppered containers and sent to a Forensic Science Laboratory through the police. The container must be filled with the blood or a layer of liquid paraffin be spread over it to prevent dissociation of the carboxyhaemoglobin—a process that readily takes place at low concentrations. If the blood is solidified, the coagulum should be sent.

PRESENCE OF OTHER TOXIC GASES IN THE BLOOD

Many fires produce noxious gases and fumes that can kill by many different mechanisms. Several toxic gases may be produced in the same fire depending upon the nature of substances undergoing combustion, as already mentioned. The gases may include hydrogen cyanide, ammonia, sulphur dioxide, hydrogen sulphide, nitrogen oxide and carbon dioxide, particularly when some plastic substances have been burnt in the fire. All of these are potentially toxic, but cyanide deserves special mention. Small concentrations of cyanide have been found in blood of victims dying in ordinary house fires. The

role of hydrogen cyanide in fire deaths is difficult to assess. However, a caution needs to be exercised while interpreting the presence of cyanide in the blood of the deceased, because cyanide is produced in significant quantities by the postmortem decomposition. It has been found to be generated in stored samples of blood, even when refrigerated for some days. [Curry AS, Price DE, Rutter ER. *Acta Pharmacol Toxical* 1967;25:339.] When such toxic gases are also present along with presence of carbon monoxide, it becomes difficult to assign the relative contribution of each and further, where there are antemortem burns too, the problem gets more complicated. However, where there are lethal levels of carbon monoxide in the blood, death may be assigned to that. Whetherell found both carbon monoxide and cyanide in 75% of fire victims that he analysed. The level of cyanide detected was less than the minimum lethal level of 300 μg per 100 ml.

CUTANEOUS REACTION TO HEAT AND FLAME

A dead body recovered from fire may exhibit skin burns sustained prior to or after death or at both the times. **The distinction whether the burns were sustained before or after from the surface appearance of the body** does not usually present much difficulty provided that the entire body surface has not been damaged. The points for differentiation include the following:

Presence of Vital Reaction (Red Flare/Red Line)

The intact skin adjacent to the burnt area will normally show a line of redness when the burns are sustained during life. This line even persists after death. It may take sometime to develop but may be absent in weak, debilitated subjects dying immediately from shock due to burns.

Vesication (Blisters)

Vesication caused by burns during life contains a serous fluid containing albumen and chlorides. The vesicles have red inflamed base and erythematous border. In contrast to it, the postmortem blisters (vesicles) are not bordered by red hyperaemic zone, may contain traces of albumen and chloride and more air. The base of such blister will be dry, hard, yellow and horny instead of being red and inflamed as seen in antemortem blisters.

Microscopic examination of the tissue from the burnt area and the sample should include skin from the intact area also beyond the margins of the burnt area. It will show congestion of the vessels and infiltration of polymorphonuclear leucocytes into the tissues and into the blister fluid depending upon the period of survival of the victim.

Histological methods of distinguishing antemortem and postmortem burns have been described by Mallik (1970) using burns inflicted experimentally on guinea pigs, burns of human skin obtained from autopsy examinations and burns inflicted experimentally on amputated human tissues [Mallik MOA. *J For Sci* 1970;5:489]. In case of human burns, the earliest histological change in antemortem burns was leucocytic infiltration at 6 hours after burning. Staining reactions for DNA and RNA at the margins of the burnt area increased at the same time, as for the enzymes alkaline phosphatase. An increase in the reaction for acid phosphatase was detected at 3 hours, for leucine amino peptidase at 2 hours and for nonspecific esterase at 3/4 hour. The histochemical reactions were not affected by surgical dressings or a lapse of time between death and postmortem examination up to 3 days. Raekallio has reviewed the application of histochemical methods to burns but states that no reports have been published on some extensive work on human material. [Raekallio J. *Z Rechtsmed* 1973;73:83.]

Suicide, Accident or Homicide

SUICIDAL BURNS

Suicides are not uncommon. In India, suicide by burning in domestic environment is much more common in females. Usually, some inflammable material like kerosene or petrol is used. A suicide note may sometimes be left.

Cases of self-immolation may be encountered as a means of indicating political dissent. In such circumstances, usually the clothes are soaked with some inflammable liquid like kerosene or petrol. At times, superficial burns may be inflicted over the accessible parts of the body for the purpose of false accusation against the enemy. Similarly, burns sustained accidentally may be falsely ascribed to have been inflicted by some particular person.

ACCIDENTAL BURNS

A vast majority of cases occur accidentally when the victims are trapped in the burning buildings or vehicles. Some accidents occur in the kitchen. In India, the victims are often women because their synthetic sarees or chunnies etc. easily catch fire while cooking, sitting or working near a stove or gas, or open lighted kerosene lamps. Children, epileptics, old, infirm, drunkards or otherwise incapacitated individuals may fall accidentally into the fire or vats of boiling water. Children or people engaged in show business or circus may get accidentally burnt in a variety of ways. Leakage of cooking gas, at times, may saturate the kitchen and accidental conflagration may occur when under such circumstances an attempt is made to light the gas. During Diwali days, accidental burn injuries are common.

HOMICIDAL BURNS

Homicidal burns, though not common, are known. Cases are on the record when lighted fire sticks, hot metals, boiling liquid or corrosives have been used with a criminal intent. Burns are occasionally caused by a mother-in-law on the body of her young daughter-in-law for trifling faults. At times, grown-up females may be punished by inflicting burn injuries over the

pudenda for adultery. A husband in a drunken state or in a fit of temper may push his wife or child into the fire. A suspicion of homicide may be entertained under two situations. **Firstly,** when it is evident that several distinct and separate parts/portions of the body have been involved and are not readily explainable by the accident under issue. **Secondly,** when there appear marks of homicidal violence. (However, the possibility of such marks being effaced by the burns may also be kept in mind and at the same time marks of physical injuries may be received prior to or at the time of accidental ignition.)

Some Legal Provisions in Relation to Dowry Death

304B: DOWRY DEATH

(1) Where the death of a woman is caused by any burn or bodily injury 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.

(2) Whoever commits dowry death shall be punished with imprisonment for a term that shall not be less than 7 years but that may extend to imprisonment for life.

498A: A HUSBAND OR RELATIVE OF HUSBAND OF A WOMAN SUBJECTING HER TO CRUELTY

Whoever being the husband or the relative of the husband of a woman subjects her to cruelty shall be punished with imprisonment for a term that may extend to 3 years and shall be liable to fine.

Explanation: For the purposes of this Section 'cruelty' means:

(1) Any willful conduct that is of such a nature as is likely to drive a woman to commit suicide or to cause grave injury or danger to her life, limb or health (whether mental or physical) or

(2) Harassment of a woman where such harassment is with a view to coercing her or any person related to her, to meet any unlawful demand for any property or valuable security or on account of failure by her or any person related to her to meet such demand.

113A: PRESUMPTION AS TO ABETMENT OF SUICIDE BY A MARRIED WOMAN

When the question is whether the commission of suicide by a woman had been abetted by her husband or any relative of her husband and it is shown that she had committed suicide within a period of 7 years from the date of her marriage and that her husband or such relative of her husband had subjected her to cruelty, the court may presume, having regard to all the other circumstances of the case, that such suicide had been abetted by her husband or by any relative of her husband.

Explanation: For the purposes of this Section, 'cruelty' shall have the same meaning as in Section 498A of the Indian Penal Code.

113B: PRESUMPTION AS TO DOWRY DEATH

When the question is whether a person has committed the dowry death of a woman and it is shown that soon before her death, such woman had been subjected by such person to cruelty or harassment for, or in connection with, any demand for dowry, the court shall presume that such person had caused the dowry death.

Explanation: For the purpose of this Section, 'dowry death' shall have the same meaning as in Section 304B of Indian Penal Code.

Burns by Moist Heat (Scalds) (Flowchart 8.1)

A scald is an injury resulting from the application of liquid above 60° C or from steam. Because of cooling of the liquid due to evaporation, the lesion due to scald is not very deep. (The protection afforded by the skin and the short duration of contact of the liquid prevent inward conduction of heat. Thus, generally superficial skin layers are affected.) However, the latent heat (heat retention capacity) of the sticky viscid liquids is high and hence, the penetration capacity of heat of such liquids is also more. Thus, an injury caused by hot tar, syrup, oil, etc. is comparatively deeper than that caused by hot water.

FEATURES OF SCALDING (Table 8.6)

- The liquid responsible for scalding may be seen on the clothes and body. Sometimes, its smell may be obvious. Scalding can occur through intact clothing.
- The injury is usually limited to the area of contact and is more severe at the point of initial contact.
- As the hot liquid gets cooled while being dispersed, scalds are severe at places where the hot liquid has come into initial contact with the skin. As the liquid runs down the body, the degree of scalding also progressively diminishes.
- Redness appears at once, and blistering (vesication) takes place within a few minutes. Vesicles are abundant along the course of the running liquid. There is usually a well-demarcated edge, corresponding to the limits of contact of the liquid. The blisters have a hyperaemic zone surrounding them. There is reddening and swelling of the papillae in the floor of the blister. If the blister skin is removed, the floor appears reddish with serosanguinous discharge. Postmortem blisters (as already

Table 8.6 Differences between Burns from Dry Heat, Moist Heat and Chemicals

Trait	Dry heat	Moist heat	Chemicals
Cause	Flame, heated solid substance or radiant heat	Steam or any liquid at or near boiling point	Corrosive acids and alkalies
Clothing	Burnt and may be adherent to the body	Usually wet but not burnt	Characteristic stains
Discolouration	Skin roasted, charred etc.	Skin bleached	Distinctive depending upon the action of chemical on the skin
Site	At and above the site of flame	At and below the site of contact	At and below the site of contact
Skin	Dry, shrivelled, charred	Sodden and bleached	Stained, corroded
Vesication	At the circumference of burnt area	Most marked over burnt area	Rarely found
Red line	Present	Present	Absent
Singeing	Present	Absent	Absent
Charring	Present	Absent	May be present in case of mineral acids
Trickled marks (splashing)	Absent	Present	Present
Ulceration	Absent (unless infected)	Absent (unless infected)	Present due to penetrating and devitalising effects of the agent
Scarring	Thick and causes disfigurement	Thin and causes less disfigurement	Keloid scar and much disfigurement

stressed under burns) contain mostly gas or may contain little fluid, which is scanty in proteins and chlorides. They do not show any antemortem reaction in and around them.

- Superheated steam soddens the skin, making the skin dirty white in colour. Occasionally, steam may be inhaled causing thermal injury to the respiratory tract, producing death by asphyxia due to obstruction of airway by the oedematous mucous membrane.
- Burning of clothes, singeing of hair, deposition of carbonaceous material and charring of tissues (common in burns) are not seen in scalding.

CIRCUMSTANCES OF SCALDING

- Scalds are usually accidental due to splashing of fluid from the cooking utensils or pouring hot water during bath or from bursting of boilers, etc.
- Children may upset the vessel containing hot water/milk/tea etc. or suck the spouts of kettles containing such hot liquids resulting in severe scalds of mouth and throat.
- Boiling water may be thrown with the intent to injure or annoy. Deliberate scalding by hot fluid is common in child abuse.
- Suicide by scalding is rare because it is very painful and moreover, there is no guarantee of death.

Hypothermia (Exposure to Cold) (Flowchart 8.1)

As in the case of heat, cold may affect the body in two ways:

- Through the generalised effects caused by lowering of body temperature as a whole.
- Through the local effects of cold producing lesions on the extremities, such as frostbite or immersion foot.

PATHOPHYSIOLOGY OF HYPOTHERMIA

The temperature of peripheral parts of body may vary depending upon the environmental and other factors, but the central (core) part of the interior of the body is usually kept out at a constant temperature. There may be slight diurnal variations of about 1.5°F. The internal temperature of the body is regulated by heat production or heat loss by the body, the former through metabolism controlled by thyroid hormone and muscular activity and the

latter by increasing blood flow through the skin or by sweating. Clothing, obviously, plays a part. Central receptors, usually considered to be situated in the hypothalamic region of the brain and also near the carotid sinus, regulate the body temperature. Peripheral temperature receptors are also known to exist.

Skin and lungs are the two most affected systems of the body. The colder temperature of the atmosphere results in the loss of body heat from the skin surface. To conserve body heat, blood vessels of the skin contract thereby lowering the surface temperature and causing a feeling of chill. Continued exposure to cold, therefore, results in injury to the superficial surface as well as loss of the body heat.

However, the major source of heat exchange is the lung surface. The air we breathe gets equilibrated with the body temperature almost immediately after it passes through the respiratory tract. Therefore, continuous breathing of cold air results in a massive loss of body heat, lowering the temperature of inner core of the body. Furthermore, since the moisture content of the cold air is rather scanty, the problem gets compounded due to inhalation of dry air.

The local effects of cold upon the tissues may result from the following factors:

- Vasoconstriction, which is usually a protective mechanism for the maintenance of body temperature.
- Injury to the small blood vessels with the formation of agglutinative thrombi in them, caused by stagnation of blood.
- An additional factor may be subsequent overheating, which elevates the metabolism beyond the level that the damaged blood supply can support.

Three phases of hypothermia as recognised by Duguid et al. (1961) are as under:

- **First phase**, which has no clinical significance, is that where rectal temperature is between 98.4° and 90° F. There is a feeling of being cold and shivering. It promptly responds to simple measures.
- **Second phase**, where rectal temperature is between 90° and 75° F, the subject is depressed and there is progressive fall in pulse, respiration and blood pressure. Shivering ceases at about 90°–85° F.
- **Third phase**, where rectal temperature falls below 75° F. Here, the temperature regulating centre usually ceases to function and there occurs progressive cooling of the body until it attains the level of atmospheric temperature. Survival from this phase is rare.

CIRCUMSTANCES OF HYPOTHERMIA

Hypothermia may be due to exogenous or endogenous causes or due to both. Exogenous causes are almost always environmental. Air temperature below 10° C is probably low enough to lead to hypothermia in vulnerable individuals but air movements like draughts will also affect by increasing the rate of body cooling. Damp conditions will also aid cooling from latent heat of evaporation. The elderly with waning defences and the children with poorly developed thermoregulatory mechanisms are likely to suffer the onslaught of cold weather. Similar ill effects are likely to ensue in individuals whose thermoregulatory mechanisms are already impaired, such as hypothyroid patients and those who are drunk.

Sips of brandy or other forms of **alcohol** are usually consumed in an attempt to ward off ill effects of cold. However, they are counterproductive and tend to aggravate the lowering of body temperature rather than raising the same (the heat generated by alcohol is due to internal combustion, which therefore lowers the temperature of inner core of the body). Further, the tendency to consume more and more alcohol to keep oneself warm impairs judgement and sense of the risk.

Endogenous causes may involve the following:

- Some disease process or drugs etc. that modify the normal physiological temperature regulating mechanism. Diseases of endocrine glands like hypopituitarism and hypothyroidism figure in many reported series. Drugs like barbiturates, phenothiazines, tranquillisers, diazepam and alcohol have all been recognised to cause increased susceptibility to cold.
- Age and physique: There are usually three groups of persons who are liable to suffer from accidental hypothermia, namely, newly born babies, elderly persons and persons engaged in hazardous outdoor activities such as mountaineering, pot-holing and sailing, etc. In general, individuals whose vitality has been lowered by fatigue, alcohol or some other factor are less able to withstand the effects of cold. Owing to greater deposit of subcutaneous fat—a nonconducting material—women can endure cold longer and better than men.
- Lack of food, adequate clothing, etc.: These are the social or financial factors often related to the depressed, apathetic state of many old people, particularly those having atherosclerosis.

MECHANISM OF DEATH

The temperature regulating mechanism gets disturbed as described earlier. As the body temperature falls, progressive decrease in the dissociation of oxyhaemoglobin occurs and, therefore, there is less supply of oxygen to the tissues. Most affected is the nervous tissue. Also, the utilising capacity of the tissues is reduced at lower temperatures. All these depress the oxidative process in the tissues and there results stagnation of blood, leading to tissue hypoxia. Therefore, the immediate cause of death is the circulatory failure.

AUTOPSY FINDINGS

Hypothermia, like some situations of drowning, epilepsy, electrocution, etc., may present difficulty as there may not be any

specific finding diagnostic of death due to cold exposure. This is particularly so when the victim of hypothermia has been admitted to the hospital and death may ensue while he is being 'warmed up'. Hence, in cases of deaths due to hypothermia, circumstances of death may be all important to establish the diagnosis with a degree of certainty.

Obviously, the extent of findings will depend upon the intensity of cold and duration of exposure. A typical case of death due to hypothermia may reveal the following findings.

External Findings

The body surface is usually pale in appearance with irregular dusky-red patches of frost erythemas, particularly on the exposed parts, large joints and extensor surfaces. Hypostasis appears pink. The colour is obviously due to the persistence of oxyhaemoglobin in the skin capillaries as discussed under mechanism of death. Rigor mortis is slow to appear and lasts longer. Extremities may be cyanosed or white. The exposed parts such as ears, nose, fingers and toes may show localised effects, **known as frostbite**, which occurs primarily by impaired local circulation. The lesions here may be superficial involving skin and subcutaneous tissues showing blisters or may also involve muscles, bones, etc. Prolonged exposure of extremities to cold sea water or cold tranches (nonfreezing temperature) for many hours produces a condition known as **'Trench Foot or Immersion Foot'** as may be seen in shipwreck survivors or soldiers. The condition of frostbite is indicative of excessive exposure to intense cold (freezing temperature of the range of –8° to –10° C) and suggests the presence of vital reaction and hence the existence of life at the time of exposure to cold.

Internal Findings

The internal appearances are not characteristic. Signs of some preexisting disease may be seen. The subcutaneous tissues are relatively avascular. The blood is often of bright red colour due to retention of oxygen by the haemoglobin at low temperatures. **More specific changes are to be found in the digestive tract, pancreas, parotid glands and brain.** Acute small submucosal gastric and duodenal haemorrhages, appearing dark brown due to the presence of altered blood, may be present. If a period of survival follows the hypothermic state, the mucosa over these haemorrhages may slough leaving shallow ulcers. Perivascular haemorrhages in the region of the third ventricle with chromatolysis of the ganglion cells may occur. A variable degree of fat necrosis in the pancreas, related to the high serum amylase levels, is probably the most striking finding. It varies from occasional patches of fat necrosis to a frank nonhaemorrhagic pancreatitis with fat necrosis in the adjacent mesentery. Multiple visceral infarcts caused by stagnation of blood by packed red cells may be the other feature. Venous thrombosis may also be found.

From a series of six fatalities and a review of the literature, Mant (1964) suggested that the criteria for the diagnosis of death due to hypothermia may appear to depend upon the presence of all or most of the following changes:

- The body may not be cyanosed owing to the lack of dissociation of the oxyhaemoglobin.
- Large, irregular, erythematous patches on the trunk and limbs due to packing or sludging of the corpuscles in the superficial capillaries.
- A relatively avascular state of the subcutaneous tissues with congestion of the internal organs associated with packing of the blood cells in the small capillaries.
- A variable degree of fat necrosis along the pancreas. This is the most constant finding and varies from occasional patches of fat necrosis to a frank nonhaemorrhagic pancreatitis with fat necrosis in the adjacent mesentery.
- Small submucosal gastric and duodenal haemorrhages, brownish-black due to the presence of altered blood. If a period of survival follows the hypothermic state, the mucosa over these haemorrhages sloughs, leaving shallow ulcers; these ulcers only rarely become deep and perforate into the peritoneal cavity.
- Excellent histological preservation of tissue.

9 CHAPTER

Starvation and Neglect

After going through this chapter, the reader will be able to describe: Starvation and its types | Autopsy findings | Circumstances of death | Malnutrition

Starvation and neglect are not synonymous but are usually considered together owing to their close association. Extremes of life are the usual victims because they are dependent upon other family members for the necessities of life. That is why certain countries have passed some legal provisions in this direction to check this menace. **In Britain**, the Children and Young Persons Act places the responsibility upon the parents/guardians/community to care for the children. **In India**, Sections 317 and 318 dealing with 'abandoning of infants' and 'concealment of birth', respectively are the steps to curb this evil. Presently, 'child abuse' (physical, sexual and mental) is inviting more attention than starvation and neglect.

Starvation may result from complete or partial deprivation of regular and constant supply of food. It is regarded as **acute** when the necessary food and water are suddenly and completely withheld as, for example, in mines or landslides, in entombment in pits, willful withholding of food and also willful refusal to take food. **Chronic starvation** occurs when there is gradual deficient supply of food, as in famines and camp conditions. The minimum food requirement for an adult depends upon his ideal weight (not current weight) and his normal work and daily activities. For an ideal weight of 60 kg, the usual requirement would be 1800 calories per day. **Life is threatened** when more than 40% of the original body weight has been lost, though the speed of loss also matters.

Fatal Period

Death usually occurs in 10–12 days if both water and food are totally withdrawn. If food alone is withdrawn, death may occur in 6–8 weeks or even more. The period, however, is influenced by a number of factors like age, sex, condition and environment of the body. A 50-year-old Jain woman successfully completed a 108-day religious fast. She used to have only boiled water during the fast, as reported.

Autopsy Findings

In both children and adults, the major problem remains the connection between the cause and effect, especially when some disease is also present. It is always advisable to perform radiography prior to autopsy so as **to exclude the physical abuse,** which is often associated with neglect. Photography should also be performed.

EXTERNAL APPEARANCES

Size and weight should be recorded. In case of children, crown heel and crown rump lengths and diameter of head are essential to be noted down. Fontanelles may be depressed owing to loss of pressure of cerebrospinal fluid (CSF). Hair may be dry and brittle, sometimes becoming depigmented. Then the comparison may be made with the standard paediatric growth charts. Diameter of the limbs at recognised landmarks should be measured. The body usually appears greatly emaciated showing dry shrivelled skin. The skin of the face is stretched tightly across the cheek bones, the cheeks are sunken and the eyes are also deeply sunken due to loss of orbital fat and an element of dehydration. The skin surface, in general, may present differing appearances varying with the length of time of the nutritional loss and qualitative deficiencies in the diet, particularly of some vitamins. Skin may be pale, lustrous and semi-translucent or coarse and rough. Pigmentation, either diffuse or localised, may sometimes be seen. Dehydration is a usual feature, and there is almost complete loss of subcutaneous fat. Pressure sores may occur on buttocks, heels and spinal region, or other frictional areas like elbows, knees, shoulders or occiput.

Prominence of ribs is conspicuously observed on the trunk, with concavities in the intercostal spaces and sunken supraclavicular fossae. The abdomen is of typical 'scaphoid shape', and the limbs are almost skeletonised from loss of fat and muscular tissue. In chronic starvation, hypoproteinaemia can lead to oedema.

INTERNAL APPEARANCES

The loss of adipose tissue will also be evident internally in the internal fat depots like omentum, mesentery and perirenal fat. Organs will be small and contracted. **More specific internal signs include** small, contracted empty stomach with bile-stained mucosa. It may contain undigested food if it had been given shortly before death to avert suspicion of willful starvation. Intestines show atrophy of all the coats showing extensive thinning out and translucency of the walls, thereby indicating that no food had passed the stomach down to the intestines for a considerable period. The lower portion of the large intestines may sometimes contain hard, scybalous faecal matter. The gall-bladder is usually distended and contains dark inspissated bile.

Circumstances of Death

It is preferable to exercise caution in expressing opinion regarding death by acute or chronic starvation, and diseases like malignancy, progressive muscular atrophy, Addison disease, tuberculosis, pernicious anaemia, etc. must be excluded. It is often difficult to assess precisely the roles of starvation, neglect and cruelty. Occasionally, it may be impossible to determine cause of death, especially when the victim is newborn. In some cases, natural disease, wholly or partially, may be responsible for death.

Suicide as a result of starvation is rare because the person cannot usually resist the intolerable thirst or quest for food. However, it may be seen in lunatics or prisoners, who may go on hunger strike. Voluntary starvation for political and religious reasons is also well-known. In this context, it may be remembered that forcible feeding of prisoners, when they refuse to take food, is not an assault but is lawful because the prisoners are under the care of the State, which has duty to protect them. The declaration of Tokyo (1975), which lays down guidelines for the doctors concerning torture and cruel treatment or other inhuman and degrading punishment, may be referred to when a prisoner refuses food. However, the doctor may be confronted with two conflicting ethical issues—his duty to preserve life and his obligation to respect the rights of a patient/prisoner to refuse treatment/nourishment. This may be resolved depending upon the circumstances of each case. Loss of weight and acidosis are the two criteria to advise forced feeding.

Homicidal starvation may be encountered in old, infirm, helpless or feeble-minded persons and illegitimate children and infants, who may be done to death by deliberate withholding of food and also exposure to cold. In big cities, small children may be kidnapped, starved, maimed and forced to beg and the earnings taken by the kidnapper.

In accidental starvation, the circumstances are self-explanatory such as famine, cyclone or earthquake, shipwreck, persons entombed in collapsed mines or the wreckage of bomb explosion. Diseases like anorexia nervosa, ankylosis of jaw, stricture or carcinoma of oesophagus and stomach, etc. may also lead to inanition. Signs of neglect and emaciation may also be seen in drug addicts where the desire for the drug surpasses the desire for food.

Malnutrition

It results from partial deprivation of food, either qualitatively or quantitatively for some period. This may lead to deficiency of bodily constituents like proteins, carbohydrates, vitamins/minerals, etc. Body weight is gradually decreased due to loss of carbohydrates, fats and proteins. The body fats and muscles are slowly wasted and the body gets emaciated. Other features may be the same as described before. Death may be due to various intercurrent infections or malnutrition syndromes. The words **'cachexia'**, **'emaciation'** and **'marasmus'** are synonymous, though 'marasmus' is usually confined to the description of infants. In cases of children, wasting and marasmus may occur from many metabolic disorders, and therefore their diagnosis may require elaborate biochemical studies and the exclusion of each and every condition is hardly expected from the autopsy alone.

Although the cause of starvation may not be capable of determination, yet the question of neglect on the part of parents or guardians may become the issue of criminal significance. Neglect implies lack of care or attention, or to leave undone what one ought to do, or failure/forgetfulness to do something.

10 CHAPTER

Death by Electrocution

After going through this chapter, the reader will be able to describe: Types of fatal electrocution | Factors influencing effects of electricity | Mechanism of death | Autopsy findings in 'medium-tension' and 'high-tension' currents | Joule burn (endogenous burn) | Circumstances of electrocution | Iatrogenic electrocution | Judicial electrocution | Lightning and mechanism of injury by it | Circumstances of lightning

The passage of electric current through human body is capable of producing a wide range of effects, varying from insignificant localised muscular spasm and little or no contact burns to instantaneous death with little or no burns or extremely severe burning.

Fatal electrocution may be divided into three groups, according to the voltage involved:

- **Domestic:** The voltage of domestic supply varies from country to country and within the country itself. Standard domestic voltages in the United States are 110 volts at 60 cycles and in Great Britain, usually 240 volts at 50 cycles whereas in India, it is usually 220–240 volts alternating current with 50 cycles per second.
- **Industrial:** Very high voltages are involved in driving heavy electrical machinery, and the voltages employed by different industries may vary. Voltages up to 400,000 volts may be employed in electric grid networks for the bulk transfer of power.
- **Lightning:** Benjamin Franklin (1706–1790) demonstrated in about 1750 that the lightning flashes were electrical discharges and not, as thought earlier, gaseous explosions. He succeeded in collecting electricity from the clouds by flying a kite during a storm and connecting the lower end of its string to the Leyden jar. This experiment led to the invention of lightning rods or conductors, which are presently employed in protecting buildings and other prominent structures (see details ahead under 'Lightning').

Factors Involved in Electrocution

As all electric supplies are potentially dangerous, some consideration of factors that are likely to lessen the hazards of electrocution is important. Some of these are related to the nature of electric supply, while others reside in the circumstances of the victim. An appraisal of these factors may not contribute in reaching the diagnosis at the time of autopsy but has a considered value in interpreting the findings and reconstructing the events surrounding the death.

FACTORS RELATED TO THE NATURE OF ELECTRIC SUPPLY

Voltage (Tension)

Volt is the unit of electromotive force. It is the force required to produce 1 ampere of intensity when passed through a conductor having the resistance of 1 ohm. **Low tensions (below 50 volts),** as used therapeutically, are usually not fatal. However, fatalities due to alternating current of low tension have been reported. Most fatalities follow shocks from currents at tensions of 220–250 volts, which is the usual range of household supply. At such voltages, the usual visible damage to the body occurs in the form of small 'electric marks' and death is owing to internal derangement of functions. **Medium voltages, i.e. under 500 volts,** predispose to prolonged contact due to induction of spasm of the muscles and therefore the victim grips and 'holds on' to the conductor. Under these conditions, a current whose momentary passage would merely cause a shock may become lethal. **At high voltages,** a person may be thrown clear of the source by the violent muscular contractions caused by the current or the body may be extensively damaged with severe and deep burns.

Amperage (Intensity)

This is probably the most important factor as far as the electricity itself is concerned. It is the unit of intensity of electric current and may be calculated in any given circuit by dividing the voltage by the resistance in ohms. On receiving a current of

1 mA, a person usually experiences tingling sensations and as the intensity of the current is increased, contraction of the muscles is greater and the current of about 8–20 mA is sufficient to prevent the victim letting go off the source of the current. At much higher currents, this factor of 'hold on' is not seen, and the victim may be thrown clear of the source of the current by violent muscular contractions and this may be responsible for secondary injuries to be sustained by the victim depending upon the circumstances. The intensity of 70–80 mA created by alternating current and 200–250 mA by direct current are **considered to be dangerous**. The danger increases as the amperage rises above 100 mA up to about 4 A and thereafter it decreases. Amperage above 4 A arrests ventricular fibrillation; **this is the principle of treatment with a defibrillator**. A current of high voltage with low amperage can be less dangerous than one with moderate voltage but high amperage. This again emphasises that the amperage is more important than the voltage.

Form of Current (Whether AC/DC)

Alternating current is one that reverses its direction at regular intervals. Standard commercial cycles are 25–60 per second. The direct current is one where the current flows constantly in the same direction. Alternating current is more dangerous than the direct current. In one series of 212 fatalities, only 8 were due to direct current (Boruttau, 1918, cited Jaffe, 1928). The body is much less susceptible to very rapidly alternating or slowly alternating currents, for instance, below 10 and above 1000 cycles per second. **The danger to the body exists when the rate lies between 30 and 150 cycles per second.** An increase in rate above this range decreases the danger. Prevost and Battelli (1899) found that heart was about 20 times more tolerant when the current was raised to 1720 cycles per second than to one at 150 cycles per second. The high frequency current used in the **diathermy**, which oscillates at one million cycles per second and carries 20,000–40,000 volts at 1–2 mA, is harmless as the effect of each impulse is to annul the effect of the preceding impulse.

FACTORS RELATED TO THE VICTIM

Resistance of the Body Tissues

It is well-known that the current flowing through the conductor is determined by the voltage divided by the resistance, i.e. I=V/R, where I is the current in amperes, V is the potential difference in volts and R is the resistance in ohms. Therefore, the resistance of the body tissues plays its role. **The major barrier to the electric current is the skin**, which exercises far greater resistance than the internal body tissues. Once the skin has been overpowered by the electric current, the vascular system filled with electrolyte rich fluid serves as a favourable medium for the passage of current. The resistance offered by the skin is further modified by the thickness (on soles and finger pads and palmar surfaces, the resistance is greater than the thin skin elsewhere) and the dryness or dampness of the skin. The dry skin of the palms offers the greatest resistance. In a labourer, it was estimated by Jellinek as from a million to two million ohms. Sweating appreciably reduces resistance. Jaffey stated that sweating could reduce the resistance of the skin from 30,000 to 2500 ohms. The resistance of the skin, as written earlier, varies from one region to the other, it being greatest in the palm and least on the inner side of the thighs. The average skin resistance is of the order of 500–10,000 ohms. Bone has a resistance of about 900,000 ohms. Vascular areas like cheeks, mucosae, etc. offer less resistance.

Area of Contact of the Body

This carries importance in two respects. **Firstly**, the smaller area of contact between the skin and the electric supply will exert more resistance than the larger area, e.g., the tip of the finger compared to the palm of the hand. Broad good contact usually reduces the resistance considerably (from 100,000 ohms to 1000 ohms as reported by Simonin, 1955). This may occur when one grasps a hot wire with a wet/sweated hand. Here, the entire skin surface being bathed in salt water (sweating) becomes a conductor and not enough current passes through any localised portion of hand to generate sufficient heat to burn the skin, which is the most efficient barrier against the passage of the current and hence electrocution can occur with no visible skin burning. Similarly, electrocution in a bath may occur without any external mark. **Secondly**, the part/site of the body and the route of current through the body have a considerable bearing. The passage through the region of the heart is most dangerous. Heart is usually involved when the path is from hand to hand or from left arm to the right leg. When the head of the worker may come in contact with the conductor, brain stem may be directly involved leading to paralysis of cardiac or respiratory centre.

Duration of Contact

It will obviously determine the amount of damage. The longer the contact, the greater will be the damage. Low tension currents may prove lethal if the contact is maintained for sufficiently long periods.

Earthing/Insulation

The pathway of the current will depend mainly upon the relative resistance of the various potential exit points. **It tends to follow the shortest route**, irrespective of the varying conductivity of the various internal tissues. The current enters at one point (most often at the hand as the hand is mostly used to hold, touch or to manipulate some electric appliances) and then leaves the body at some exit point, usually to the earth. The better the contact between the person and the earth at the time of sustaining the electric shock, the more dangerous will be its effects. A person, therefore, standing with dry shoes on a dry surface may scarcely notice a shock, which could prove fatal to someone

standing bare footed on a wet surface. Hence, stout rubber gloves and rubber boots provide considerable protection.

OTHER FACTORS

Personal idiosyncrasies of the human beings may also play a role. Jellinek considered that the personality and **physical condition of the individual** and the existence of bodily or mental distress at the time of sustaining shock influence the effects of the shock. Further, **awareness of the victim** towards the possibility of shock being sustained may make the victim withstand one which might otherwise be dangerous. Reported cases show that an individual taken by surprise may succumb to shocks that ordinarily produce no ill effects. An engine driver used to exhibit his skills by exposing himself to shocks from an electric lamp carrying a tension of 50 volts by catching hold of the lamp with both hands and letting it go as a bet for a glass of beer. He succeeded in doing so with impunity until one day he happened to have accidental contact with the lamp and died of an unexpected shock (Taylor, 1948). **Presence of any disease** in the victim, especially the cardiac disease, may predispose to death from currents of low tensions.

Mortality and Mechanism of Death by Electric Shock

In spite of almost universal use of electricity for domestic lighting and heating purposes and its extensive use in industry in the developed and developing countries, the fatal accident rate is meagre. Immediate and adequate resuscitation can decrease the mortality rate because quite often the victim may be in a state of suspended animation. Therefore, resuscitation needs to be continued for a sufficient period until unequivocal signs of death appear. There were 323 recoveries in the series of 479 cases studied by Mac Lachlan (1930), and he observed that artificial respiration was likely to succeed if initiated within 3 minutes of the sustaining of the electric shock. He reported a remarkable recovery of a victim who had received a shock from the current of 22,000 volts, when he was instituted resuscitation at once. **The following mechanisms may operate in causing death**.

VENTRICULAR FIBRILLATION

Most deaths from the electric shock are from cardiac arrhythmias, usually ventricular fibrillation terminating in arrest. This occurs when the current passes through the thorax, from hand to hand or from hand to leg routes. The critical level of current seems to be of the order of 100 mA. The most dangerous is from the left arm to the opposite leg; from arm to arm is about 60% as lethal. Lee observed that loss of consciousness needs not be immediate, and some may even be able to walk some distance before they die. The autopsy appearances under such circumstances will be nonspecific except the presence of external electric marks.

SPASM OF THE RESPIRATORY MUSCLES (TETANIC ASPHYXIA)

Electric current passing through the thorax may lead to tetanic contraction of the muscles of respiration and ultimately producing respiratory arrest. Here the mode of death obviously will be congestive hypoxia. These victims are likely to be cyanosed whereas in case of death due to ventricular fibrillation, they usually appear pale. The range of current that can induce tetanic contractions of the extrinsic muscles of respiration may be 20–30 mA.

PARALYSIS OF THE RESPIRATORY CENTRE

Paralysis of the respiratory centre occurs when the current passes through the head, which is a rare event (Fig. 10.1). The passage of several hundred milliamperes through the brain during the electroconvulsive therapy rarely results in suppression of respiratory centre, though a current of much less intensity would be sufficient if it passed through the centre. The heart may continue to beat and hence the importance of resuscitation, as already stressed.

SECONDARY CAUSES

Death, in some cases, may occur actually due to sustaining of mechanical injuries, secondary to the circumstances of

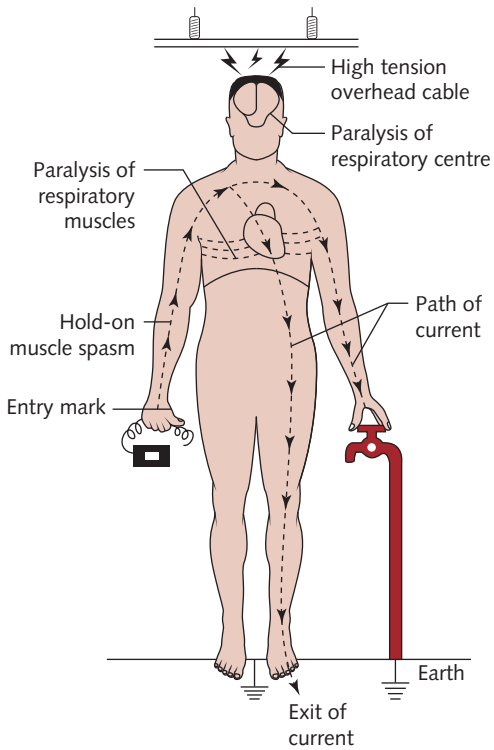


Fig. 10.1 Pathways of current in electrocution.

electrocution, as may happen when a worker working on a high voltage supply system gets electrocuted and falls from a height and receives head injury or some other injuries. Late deaths can occur in those who do not die immediately and sustain severe burns due to infection or haemorrhage because of damage to the blood vessels.

Autopsy Findings

Visible damage due to electrocution varies within wide limits from nil to gross. This depends mainly upon the strength of the current, and it is obvious that gross damage is usually observed in the high-tension currents, and prolonged contact with the low or medium currents can also lead to gross damage. Attention of the readers is again drawn to the opening sentence of this chapter that clearly stresses the variability of damage in deaths due to electric shock. Hence, the findings may be discussed depending upon the involvement of the victim in low or medium current and those involved in high-tension circuits.

FINDINGS IN DEATHS DUE TO LOW- OR MEDIUM-TENSION CURRENTS

The point where the current enters the body is usually characterised by the presence of an electric mark or electric burn **(Joule burn or endogenous burn)**. Another mark or marks may appear where the body is earthed or grounded. However, it must be stressed here again that fatal electrocution can occur with no visible skin mark, and the doctor may have to reach the diagnosis by exclusion of all the other possible causes and by attending to the circumstances of death. The examples, as already described, may be deaths occurring in the bath, when the entire body surface acts as a source of entry, and this coupled with the lowered resistance of the wet skin prevents the formation of any localised mark.

The electric mark, though specific of contact with the electricity, is not in itself a proof of electrocution because marks resembling those found on the victims of electrocution can be produced after death (excluding a zone of hyperaemia) as reported by Polson and Gee. It is also possible to produce changes in the skin resembling an electric mark by applying a glowing or intense hot wire to the skin. Distinction between electric mark and thermal burn may be made by acro-reaction and by scanning electron microscopy (SEM), as explained below. However, they do raise strong presumption of death by electrocution and together with the study of circumstances, diagnosis can conveniently be achieved. **Some important features of the mark may include the following:**

- If the contact of the skin with the conductor is good and firm, the passage of current heats up the tissue fluid and the skin offering resistance gets split and blister may be raised. The blister so formed may get ruptured if the contact continues or the area involved is relatively large. The shape and size of the mark may correspond to the shape and size of the source of the current in such cases (Fig. 10.2A).
- If the contact is not good or is less firm, the current jumps the gap between the source and the skin in the form of a spark and causes the outer skin keratin to melt over a small area. On cooling, the keratin gets condensed into a hard brownish nodule and this is termed as the so-called spark lesion (Fig. 10.2B).
- Combined lesions are encountered in many electric burns due to the movement of the hand or the body against the conductor.

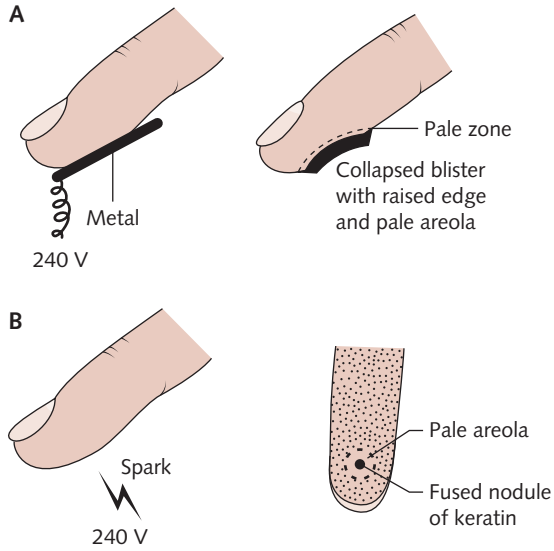


Fig. 10.2 Effect of contact of the skin with the conductor: **(A)** firm contact—electrical marks; **(B)** less firm contact—spark burn.

Characteristically, a well-developed electric mark is a round or oval, shallow crater, bordered by raised areola of blanched skin around a part or whole of its circumference (Fig. 10.2). This pallor (blanched) border is possibly due to arteriolar spasm caused by direct effects of the current on the vessel walls. The pallor survives after death and is a useful indicator of electric damage. There may be mild hyperaemia of the intact skin immediately beyond the blanching. The floor of the crater is lined by a pale flattened skin, and the ridge pattern may or may not be preserved. These electric marks are produced by conversion of electricity into heat within the tissues and, therefore, they might be termed as **'endogenous burns'** to distinguish them from **'flash or exogenous burns'**. If the contact is with the long axis of the wire, a linear mark or groove may be caused but contact with the end of the wire may produce a hole that may go deep into the tissues involving even muscles and bone.

It may be kept in mind that these lesions usually have greater depth than the insignificant surface appearance. Therefore, it is essential to examine the flexor surfaces of the fingers by forcible breaking of the rigor and if need be, flexor

tendons may be cut to release the rigor. Occasionally, the mark may be present in the mouth, especially in case of children; therefore, mouth must be inspected and if need be, proper dissection of the mouth may be carried out.

The exit mark(s) are variable in appearance but usually have some of the features of entrance marks. There may be more disruption of tissues, and they are often seen as splits in the skin or sometimes even lacerations. Burns and perforations of the clothing or shoes may be seen over the site of exit. If the current enters and leaves the body over a wide area of low resistance as provided by water (wet hands or wet body surface) and good grounding (wet soil), neither current marks nor burns may be found. Hence, absence of electric marks does not exclude death from electrocution. In such cases, exclusion of other possible causes and a study of the circumstances will help to resolve the issue.

HISTOLOGICAL APPEARANCES

The epidermis may appear flattened due to distortion of the cells, caused by the passage of current. Epidermis may get separated from the corium to form blisters. The cells of the epidermis are often elongated and the nuclei of the lower layers get stretched. An important feature is the occurrence of spaces of varying sizes and shapes in the corium and epidermis to impart a honeycomb appearance.

Hassin (1933, 1937) found histological changes in the brain even when death was instantaneous. He stressed upon the changes like tearing of the nervous tissue and its shrinkage around the smaller blood vessels. Changes in the brain have been described even when the cause of death was cardiac in origin.

Scanning electron microscopy may be promising in distinguishing electrical damage from that of thermal damage. It possibly provides the chemical analysis by electron microprobes and helps in identifying the metallic deposits.

FINDINGS IN DEATHS DUE TO HIGH-TENSION CURRENTS (EXOGENOUS BURNS)

Injury by high-tension electric currents occurs either by direct contact or by indirect results of arcing or flashover. The high-tension injuries are usually seen in linemen working on the grid systems and occasionally in thieves stealing wires from high-voltage overhead lines. With extremely high voltages, there may be actual arcing of the current over several centimetres, without occurring actual contact. The man may be hurled (knocked down) from the vicinity due to sudden and appreciable increase in the local atmospheric pressure and sustain secondary non-electrical trauma. An interesting case has been cited by Polson and Gee of a man who climbed a pylon with the intention of committing suicide, suffered a short circuit and flashover and consequently was knocked down with cloths catching fire. He fell to the ground and died of multiple injuries (secondary trauma) as well as burns due to clothing catching fire. Electricity played only indirect role (Leeds City Coroner, No. 432/37).

Actual charring of the tissues with carbonisation is common but depending upon the degree, there may be:

- Brownish discolouration of the large areas of the skin apart from actual burning.
- Arborescent pattern resembling lightning burns.
- Crocodile skin effect comprising of confluent multiple spark burns over large areas of the skin.

INTERNAL FINDINGS

Gross findings in the internal organs may be absent because the tissues are mainly aqueous and contain electrolytes thus providing a diffuse pathway for the passage of the current and preventing any thermal damage. Mostly the death is due to cardiac arrhythmias leading to ventricular fibrillation and arrest. In such cases, findings in the internal tissues are negligible except for external skin marks.

- In the event of death due to tetanic asphyxia, cyanosis of the face, petechial haemorrhages in the skin of face and beneath the pleura and epicardium may be seen. There may be congestion of viscera and oedema of lungs, etc.
- In the event of the brain in the route of the current, some macroscopic and microscopic changes may be noticed as described earlier.

Circumstances of Electrocution

Deaths due to electrocution need to be thoroughly investigated and documented for reasons of compensation and for instituting future measures in relation to safety and prevention. A worker who drops dead while working on some electric line should be suspected to have died of electrocution. Close attention must be paid to the hands and the mouth as these are the usual involved sites. Blood and viscera should be analysed to assess whether the victim was impaired at the time of accident. Secondary injuries due to fall or 'knock down' need to be described in detail. The allegedly defective appliance should also be got examined by the investigating officer from some competent expert. The investigating officer must prepare a detailed report of the scene along with sketches and photographs. Clothing of the victim should also be described and preserved properly.

ACCIDENTAL ELECTROCUTION

Majority of the fatalities usually result from the accidental contact with the low voltage currents (normally 220–240 volts). Accident may occur from a faulty line, while working on an electric cooking heater, room heater or inside the bathroom from a heating electric coil that, if defective, may charge the water in the bath tub or bucket with electricity. Accidents may occur while repairing high-tensions overhead wire connections. It may occur due to short circuit in temporary wiring in a tent.

Accidental ventricular fibrillation has been recorded in cases of intracardiac catheterisation and from the site of pacemaker. Accidents may also be seen with the use of electric blankets, and the hazards created by these blankets may include electric shock, burns as well as fire. When out of use, the blankets should be stored flat to prevent damage to the wiring by folding. Accidents due to contact with high-voltage supplies are usually seen in industries. Outside the industry, it may be encountered when an individual disregards warning signs or ignores the presence of high-voltage cables while moving some ladder or otherwise engaged in some activity in the vicinity of such cables or systems.

The danger of flying kites in the vicinity of overhead electric supply lines has been reported. While flying a kite with the ordinary string, a boy happened to touch a live electric wire with his kite, and was burnt badly and rendered unconscious. He succumbed eventually to his injuries. On the day of the occurrence, the ground was wet with rain and the string moistened by contact with it. Indirect contact with high voltage was also reported when a boy urinated on an electrified rail, the current travelled upwards through the urinary stream. Figure 10.3 represent a case of accidental electrocution of two children.

SUICIDAL ELECTROCUTION

Electrocution is an infrequent mode of suicide. The victim usually winds wire round the wrists or other parts of the body, makes their connection with the wall socket and switches it on. Normally, the apparatus is found in situation when the body is examined at the scene. A case was reported in the *Times of India* (14th April, 1962), narrating suicide by a college lecturer by wrapping his wrists with naked wires and connecting them to the electric plug.

HOMICIDAL ELECTROCUTION

Homicide by electrocution, though extremely rare, is not unknown. In R vs. Whybrow, Chelmsford Assizes case (1951), the husband connected the soap dish in the bathroom used by his wife to a source of electric current in such a manner that while operating the switch in his bedroom, he could cause his wife to receive shock whenever she happened to touch the soap dish. The wife received shocks during baths and eventually called for the electrician, who was able to locate the design. The husband was convicted for attempted murder by electrocution and sentenced to 10 years imprisonment.

Erection of electrified wires to protect property or to attach a live wire to door knobs, gates, railings, etc. to prevent theft and burglary may cause death of the intruder. At times, the victim may be murdered by other means and a case of electrocution by producing electric burns on the fingers may be presented.

IATROGENIC ELECTROCUTION

Accidents have occurred in the course of investigations or treatment of patients with electrical equipment. Such iatrogenic electrocutions have occurred in the course of electroconvulsive therapy. Electrical hazards are increasing in the intensive care units, operating theatre, X-ray room or during direct electrical connection to the heart, e.g. when cardiac catheters are used for pressure monitoring, injection of radiographic contrast media or passage of pacemaker electrodes. Since there is always a risk, the electrical instruments should be of safe design and regularly serviced to keep them in good working order. It is worth mentioning that micro-ampere electrocution and natural death cannot be distinguished at autopsy. In cardiac defibrillation, though the current flow is enormous, it is for such a brief period that it stops the fibrillating heart. The discharge must be delivered at an appropriate time during the cardiac cycle to prevent induction of fibrillation or standstill.

JUDICIAL ELECTROCUTION

In some of the states in USA, the death sentence is carried out in an electric chair. The condemned person is strapped to a



Fig. 10.3 Effect of accidental electrocution on two children. **(A)** This child got struck against her neck by a wire hanging from an electric pole and died on the spot. **(B)** The elder child in an attempt to save the younger caught the same wire and got electrocuted.

wooden chair and a metal electrode is placed over his shaved scalp and the other metal electrode around one leg. An alternative current of 1700 volts and 7.5 amperes is passed through the body for a minute or two. The current is passed through the body a second time and repeated till life is extinct. In such deaths by electrocution, it is recorded that the brain is heated up to 60° C and that vacuolation around the vessels occurs.

Lightning

Although deaths from lightning are quite rare, yet their effects are so capricious and unpredictable that they demand recognition by a medicolegal expert.

MECHANISM OF INJURY BY LIGHTNING

As already described under Electrocution, it was Benjamin Franklin (1706–1790) who discovered that lightning flashes were electrical discharges and not gaseous explosions. In lightning, the discharge may be from cloud to cloud or from cloud to the earth through some object, usually the tallest object in contact with the earth. Lightning chooses the easiest path (not the shortest), i.e. the path of least electrical resistance. It liberates terrific amount of energy. The electric current is direct, of about 20,000 amperes and about a million volts operating over an average period of 30 microseconds. The effects of lightning are due to:

- Passage of very high potential electricity that liberates tremendous energy in the form of heat, responsible for producing various burns that are usually superficial owing to the very short duration of the flash.
- Blast effects of the rapidly expanding air that may tear the clothing and impart suspicion of foul play.

The burns produced by lightning may be studied under the following groups:

- **Surface burns,** which are tissue burns and usually travel through metallic objects worn or carried by the victim. Even metals with very high melting points, such as gold (melting point over 1000° C), may volatilise and the metal object becomes molten or heated up producing surface burns of varying depth and intensity. Metallic articles like wrist watches, bracelets, metal hooks or zip and so on may be the source of attraction for the lightning discharge. Some melted metal may be implanted into the skin, producing distinctive colouring.
- **Linear burns** may be found where the area of the skin offers lesser resistance, i.e. moist creases and folds of the skin.
- **Arborescent or filigree burns or feathering,** which usually appears like that of the branches of a tree or the fronds of a fern, is due to rupture of smaller blood vessels at several places giving rise to ecchymoses with arborescent pattern. Arborescent pattern, when present, is quite distinctive but it fades within a few hours if the victim survives.

The primary burn is usually on the head and may be diffuse or the injury may be simply in the form of bruising usually at the back of the head, which may be attributed to 'sledge hammer blow' dealt by the compressed air pushed before the current (Spencer). The passage of the charge over the body may be traced by skin burns and damage to the clothing. A burn mark may also be present at the point of discharge from the body to the earth. This may be more intense than the skin markings between it and the point of entry or it may appear as a deep burn.

BLAST EFFECTS

They may be observed in the form of tearing of clothing or shoes or the effects may also be observed on the trees showing areas of scorched leaves and vegetation in the vicinity of the scene of death. Metallic objects in the area may get fused or become magnetised. Nylon underclothing may melt. Objects at a distance of 100 feet or more may be struck. A dramatic example was reported by Skan from Africa. He was struck by lightning inside his hut and the injuries included tearing away of left shoulder, a large hole in the left side of the neck and fractures of the skull and left humerus (Skan DA. *Br Med J* 1949;i:666). Mant described an interesting case of a girl riding a horse, who was killed by lightning that struck her through a metal stud on the top of her hat and travelled through her body, melting her nylon panties and tearing her jodhpuris. (Mant AK. 1968. In: Gradwohl's *Legal Medicine,* 2nd ed., Bristol: John Wright.)

DIAGNOSIS OF DEATH BY LIGHTNING STROKE

As doubts may be raised of foul play because of bizarre extent and distribution of injuries and torn clothing, the diagnosis of death may be achieved by carefully considering the following:

- History of thunderstorm in the locality.
- Evidence of the effects of lightning in the vicinity of the scene of death, i.e. damage to the houses or trees or the animals, fusion or magnetisation of metallic substances in the nearby place, etc.
- Bursting open of the clothing is characteristic and the tears may be scorched and impart smell of singeing. Boots and belt can also burst open.
- Characteristic nature and distribution of burns, which are usually superficial due to very brief duration of flash.
- Sudden death may be attributed to the direct involvement of the central nervous system causing death from paralysis of cardiac and/or respiratory centre. High-voltage electric current may cause spasmodic contraction of the cerebral vessels leading to cerebral anaemia, which in turn may be responsible for stoppage of respiration and circulation. However, contraction of the heart muscle may also be due

to direct effect of current on the heart. In such cases, no specific findings are to be expected and the diagnosis may rest upon the factors enumerated above.

CIRCUMSTANCES OF LIGHTNING STROKE

Ordinarily, lightning deaths do not pose any problem except in occasional cases when the dead body may be recovered from the open with no marks upon it, but usually the history and evaluation of the scene of death resolves the issue. Lightning tends to pass along the surface of the conductor rather than through it, so that the persons in the building are rarely affected, if the building is struck. Occasionally, persons in the building may be affected when the lightning strikes a chimney or television aerial and passes down through a living room.

Most fatalities occur in open. Persons sheltering under trees in the thunderstorm make up quite a percentage of the victims. Others may be struck in the open fields, particularly if they are carrying or wearing something that may attract lightning.

Spencer explained the apparent immunity of motor vehicles from the lightning stroke. He opined that it might be due to the insulation provided by the tyres. A car, however, may be indirectly struck by a 'splash' from some other object nearby. According to Dr. AS Curry, this immunity of motor vehicles may be due to the fact that it behaves like an empty metal box having no circuit within it. Immunity may be lost as and when some portion (a fist or hand, etc.) is put outside the vehicle.

Once again, it may be projected that the effects of lightning are extremely bizarre and unpredictable. Imagine two persons standing side by side during a stroke. One may be struck and killed, whereas the other remains unharmed. An example, as cited in Gradwohl's *Legal Medicine,* is quite instructive:

Two persons with unfurled umbrellas were walking together across a field during a thunderstorm. One was struck through the umbrella he was holding. The charge passed down the umbrella and entered his body through the right side of his head and the right lower chest and abdomen, and was earthed through both legs. His umbrella disintegrated, his clothes were torn into small pieces, and the shoes also disintegrated along the soles. He died in hospital the next day from coronary thrombosis and rupture of a gangrenous caecum due to local thrombosis. His companion was uninjured.

Converse may also be true, i.e. when two or more persons are close together during a thunderstorm, all or several of them may be killed or severely injured although only one of them suffers a direct stroke. For example, 23 climbers in the age group of 20–25 years were caught in a thunderstorm while climbing up a step mountain in Japan. Only one of them was struck but all were killed (Goldie and Lee, 1976).

11 CHAPTER

Deaths Associated with Surgery, Anaesthesia and Blood Transfusion

After going through this chapter, this reader will be able to describe: Surgical intervention | Respiratory embarrassment | Cardiac embarrassment | Regional and spinal anaesthesia | Instruments and instrumentation | Unforeseeable problems | Precautions for autopsy | The autopsy | Medicolegal considerations | Blood transfusion—hazards and risks | Periprocedural complications, etc.

Informative and sociologic awareness of the public about their perceived rights and prerogatives is evident from the spate of malpractice actions against the medical profession. No wonder, therefore, that deaths occurring during or within a short period after surgical, invasive diagnostic procedure or administration of an anaesthetic may become the subject of a medicolegal investigation. To be effective, such investigations must include autopsy. Simpson reviewed 500 deaths during anaesthesia and calculated that 56% were occasioned by the disease for which the operation was conducted, 30% were the outcome of shock and inevitable risks of the operation itself, 8% were caused by the risks and complications of anaesthesia, while 6% could be pinned upon overdosage, maladministration or bad choice of anaesthetic.

American Society of Anesthesiologists (ASA) has classified patients into a number of grades according to their clinical condition for assessing their fitness to undergo anaesthesia. The classification is following: I—A normal healthy patient. II—A patient with mild systemic disease. III—A patient with severe systemic disease that limits activity, but is not incapacitating. IV—A patient with incapacitating systemic disease that is a constant threat to life. V—A moribund patient not expected to survive 24 hours with or without operation. The ASA scheme is the most comprehensive system, but it does not embrace all aspects of anaesthetic risk, as there is no allowance for inclusion of many criteria such as age or smoking history or obesity or pregnancy or the risk of asymptomatic patient who may have severe coronary artery disease. It also disregards the inherent risks of a particular operation. Deaths associated with surgical intervention or invasive diagnostic procedures may necessitate following considerations:

- Where the disease or injury was in an advanced stage and a heroic effort was taken in the hope of saving the life of the patient, there must have been some chance of success, or even mitigation of symptoms foreseeable, otherwise the intervention would not have been justified. However, the effort could not bring fruits and death is ordinarily attributed to the disease or injury for which the intervention was necessitated.
- Where the death can be attributed solely or partly to disease or disability other than for which the intervention had been carried out. This calls for careful preoperative assessment/evaluation of the case and drawing a risk:benefit ratio. Modification of operative and anaesthetic procedures may be adopted after taking an account of the known adverse conditions. Issue of negligence may creep up when some occult disease process remains undetected. However, the test for liability would remain the same, i.e. whether a doctor of reasonable skill and competence could have/could not have diagnosed the same under similar conditions.
- Occasionally, some unusually difficult surgical procedure may land into a failure. This may happen inadvertently from a true accident, from some anatomical abnormality or from failure of equipment. In the event of failure of equipment, examination and advice by the concerned expert carry utmost importance. Injury to some large blood vessel or leaving of some pack/swab/instrument in the body may directly be covered under the concept of 'error' or 'incompetence'.

As far as deaths associated with anaesthesia are concerned, an interesting approach to the problem was made by the publication 'Communications in Anesthesiology', which queried its readership about their views concerning anaesthetic complications. The replies showed that the anaesthetists were most concerned with (*i*) respiratory and circulatory problems, (*ii*) problems related to local or regional anaesthesia and

(*iii*) problems related to the mistakes or errors in the utilisation of the apparatus. Each needs brief discussion, which is as follows.

Respiratory Embarrassment

Compromised oxygen supply to the tissues is the ultimate matter of great concern. Intra-operative problems are likely to effect oxygenation even in an otherwise normal lung. Some important factors may include the following:

- Some of the iatrogenic precipitating factors for hypoventilation may include oversedation, prolonged anaesthesia, chest fixation, misplaced tracheal intubation, too generous use of oxygen in a patient with chronic respiratory insufficiency and inadequate reversal of muscle relaxation, etc.
- Improper or excessive premedication can also lead to hypoventilation. It is well-known that morphine, tranquillisers and barbiturates cause central respiratory depression and decrease the depth and volume of respiration. Hypoventilation may occur in deep planes of anaesthesia with intercostal paralysis and diaphragmatic impairment.
- Packs and retractors may press upon the diaphragm and impede movement of the lungs. Patients with neuromuscular weakness or chronic obstructive lung disease are particularly prone to hypoventilation. Aged patient with fixed thoracic cage, depressed ciliary activity and suppression of the coughing reflex are the other sufferers.
- Hyperventilation also needs to be checked. However, it has been advocated that it is still safer to err on the side of hyperventilation rather than risk the possibility of hypoventilation.
- A paralysed patient cannot vomit, but regurgitation takes place easily. Hence, contamination of tracheobronchial tree may take place in many ways, viz., secretions, gastric contents, food, blood, etc. may be aspirated into the respiratory tract. Aspiration of pus may lead to multiple abscesses of lungs. With aspirated gastric contents, chemical irritation may ensue that may ultimately lead to pulmonary oedema and chemical pneumonitis. Though tracheal intubation is instituted to prevent dissemination of vomitus, pus or regurgitated matter, yet prevention may not be absolute because material may leak past the inflated cuff. (At the autopsy, finding of gastric contents in the airways needs careful scrutiny. It needs some evidence of its clinical/antemortem supplementation because such contents can be present in some part of the air passages due to agonal or even postmortem spillage. This has been amply discussed and illustrated under 'Death due to Choking'.)
- Obstruction by solid particles/objects may lead to atelectasis. Misplacement of the tracheal tube into one of the bronchi may also lead to atelectasis.
- Massive transfusion may lead to pulmonary oedema. It is usually the result of relatively excessive amounts of fluids, plasma expanders or blood. The inhalation of toxic agents is a less likely cause. Another possibility may be the effect of airway negative pressure, which increases the gradient between capillary pressure and alveolar pressure, producing transudation.
- Rupture of an emphysematous bleb during operative procedures from inhalational anaesthesia may result in pneumothorax. It is also one of the complications of subclavian vein catheterisation or supraclavicular brachial plexus block. If accumulation of air in the pleural cavity continues and intrapleural pressure rises, tension pneumothorax may develop, which is dangerous because of the displacement of the mediastinum and resulting bad effects on the heart and great vessels.

Cardiac Embarrassment

Normal cardiac function depends upon the balanced action of the two mutually antagonistic divisions of the autonomic nervous system, viz., the sympathetic and parasympathetic. **The former** stimulates while **the latter** depresses the heart. Fear, severe pain, extremes of temperature and decrease in the tension of the carotid sinus cause the stimulation of the sympathetic system. This stimulation increases myocardial irritability and leads to premature ventricular beats, ventricular tachycardia and ventricular fibrillation. The stimulation of peripheral fibres, the stimulation of respiratory tract, pleura, peritoneum, abdominal viscera or pulling on their attachments and increase in the tension of the carotid sinus lead to parasympathetic stimulation. This stimulation leads to the sudden slowing of the heart, hypotension and even cardiac asystole.

Hypoxia acts as a contributory factor in producing cardiac embarrassment in two ways:

(i) Hypoxia, acidosis and hypotension from pain, fear and other causes sensitise the myocardium to the action of catecholamines, eventually leading to ventricular fibrillation, and drugs (such as chloroform) or diseases (such as coronary sclerosis) also sensitise the myocardium to catecholamines and reflex nervous stimulation.

(ii) Oxygen starvation to the brain and heart may occur owing to hypotensive episodes during the operation, and the effect of such episodes is cumulative. The heart, after beating under hypoxic conditions for some period, progressively builds up greater and greater debt and finally gives way. Furthermore, heart and brain that are already burdened with drugs (premedication and anaesthetic agents) are more likely to succumb to insignificant deprivations.

Hypovolaemia and Other Factors

Unrecognised or inadequately managed hypovolaemia is quite common cause of anaesthesia related death attributed to the cardiovascular system. Deaths due to hypovolaemia may arise in a number of ways, namely:

(i) Failure to recognise or to make adequate provision for preoperative hypovolaemia.
(ii) Improper dosage of anaesthetic agent or spinal or epidural anaesthesia converting a compensated hypovolaemia into an uncompensated lethal form.
(iii) Inadequate volume replacement for intra-operative losses, etc.

Conversely, rapid infusion of large quantities of blood may lead to deficiencies in clotting factors. It has been documented that administration of more than 8–10 units of blood in 24 hours will lead to a fall in the level of labile coagulation factor in the recipient's serum leading to bleeding called 'dilution coagulopathy' [factor V (proaccelerin) is an unstable protein substance, also known as 'labile factor']. Overenthusiastic fluid therapy may also contribute to anaesthesia related mortality from pulmonary oedema or cardiac failure.

Regional and Spinal Anaesthesia

Local anaesthetics exert their action by preventing impulses arising from and passing through nervous tissue. These agents are given along with some vasoconstrictor drug to ensure localisation of the agent and prevent its rapid absorption. Large doses depress the central nervous system and myocardium leading to hypotension, unconsciousness and circulatory collapse. Some important complications associated with their use may be enumerated as follows:

- Bacterial contamination at the site of the block may cause infection or cellulitis.
- Chemical contamination may cause necrosis or sterile abscess.
- Needle trauma to a blood vessel may cause a haematoma and gangrene.
- Air embolism may occur from any injection adjacent to cervical veins.
- Pneumothorax may result from brachial plexus or stellate ganglion block.
- A broken needle or catheter constitutes a foreign body and necessitates removal.

Systemic reactions/toxicity can also be encountered. These reactions may be considered as those adverse effects of the absorbed drug that affect the body as a whole. Although allergy is often cited as the cause, it is involved in rare instances only. Such reactions may range from anaphylactic shock to dermatitis.

Aetiological factors concerning toxic reactions may include total amount and concentration of the drug injected, the rate and route of injection, the vascularity of the area, use of vasoconstrictors and spreading agents, relative toxicity of the agent to the individual and the possibility of an inadvertent injection into some blood vessel or into a particularly vascular area. Patient's physical status, presence of any disease and the particular climatic conditions may be other factors. In a small percentage of cases, hypersensitivity reaction may occur. The test for skin sensitivity is of doubtful value. Often, slip in prompt evaluation of the reaction/situation, non-availability or poor availability of immediate resuscitatory measures and antidotes extend contribution towards fatal outcome.

Spinal anaesthesia is a relatively safe type of anaesthesia in skilful hands. However, complications may arise due to faulty technique. Hypotension due to paralysis of sympathetic outflow is a common complication, and patient with coronary insufficiency may develop myocardial infarction. In high spinal anaesthesia, the involvement of sympathetic fibres to the heart may lead to cardiac inhibition from unopposed activity of parasympathetic fibres. Damage to spinal roots may cause weakness of muscles, which is manifested not immediately but about a couple of weeks afterwards. Chronic adhesive arachnoiditis is a common complication. It is slow in onset, and development progresses in a cephalad direction. Injury to the spinal ligaments and annulus fibrosis may occur and occasionally, osteomyelitis of the vertebra may occur.

Instruments and Instrumentation

Equipment failure is a relatively minor cause of anaesthetic mishap these days. Cooper, Newbower and Kitz (1978) reported that equipment failure accounted for only 11% of all critical incidents, and that only 4% of the incidents involving equipment failure presented substantive negative outcomes. Human error was responsible for 82% of the deaths. Other factors included inadequate communication amongst staff, haste and distraction. Cooper et al. described the following three types of human errors:

- **Technical:** In which the action taken is not the action intended. This may arise from deficiencies of technical skill or from poor design of the equipment.
- **Judgemental:** In which the action represents a bad decision, arising from lapses in the training or poorly developed decision-making skills.
- **Monitoring and vigilance failure:** In which there lies an essential failure to recognise or act upon an observation/finding requiring a response.

Circumstances may include the following:

- The endotracheal tube may get dislodged, kinked or obstructed by mucus, blood or growth. Further, it may injure pharyngeal

or oesophageal mucosa or the postmembranous part of the trachea, especially in elderly.
- The separated cuff may block the larynx at some occasions.
- Manoeuvring of the head and neck associated with laryngoscopy may cause glottic oedema and injury to teeth and cervical vertebrae, especially in elderly.
- Veins are likely to get injured in the procedures of the neck resulting in air embolism.
- The malfunction of anaesthetic machines may lead to hypoxia, anoxia and anaesthetic over- or underdosage (Eger and Epstein, 1964).

Unforeseeable Problems

- **Haemoglobinopathies:** Victims of haemoglobinopathies, especially sickle cell anaemia, are unduly susceptible to low oxygen tension in blood, and this may pose a hazard to the unaware surgeon or anaesthetist.
- **Thrombosis:** Coronary thrombosis may supervene in a patient operated upon for injuries.
- **Transfusion infection:** Transfusion hepatitis is not unknown. AIDS infection through transfusion is another possibility.
- **Muscle weakness:** Competitive neuromuscular blocking agents can cause severe muscle weakness in patients with myasthenia gravis and myopathies.
- **Hyperthermia:** Halothane and/or succinylcholine may produce 'malignant hyperthermia' occasionally. This is supposed to occur in patients with an inherited muscular defect and is ascribed to increased muscular metabolism.
- **Anaesthetic gas ignition:** Inflammable mixtures of anaesthetic gases with air or oxygen may get ignited through sparks from electric appliances, diathermy apparatus, naked flames and cause explosions and fires. Any electrical apparatus is potentially dangerous and defective cauteries, defibrillators and diathermy equipment have been reported to have caused death. Explosions from inflammable gases and vapours, such as cyclopropane and ether, have been catastrophic at occasions.

Precautions for Autopsy

Before discussing anything regarding the autopsy, it must be made clear that most deaths concerning anaesthesia are unlikely to be discernible at the postmortem examination. Moreover, as described earlier, oxygen starvation to brain and heart may occur due to hypotensive episodes during the operation, which may have a cumulative effect and one slip in the technique can trigger a chain reaction ultimately leading to death. 'Such slips' are undetectable at autopsy, whereas surgical mistakes being anatomical may be observable at the postmortem and anaesthetic mistakes being physiological are no longer appreciable after death except where overdose with specific drug is involved. Therefore, an autopsy surgeon is usually left to discover or exclude some natural disease or mechanical obstruction as the pharmacological aspects usually remain beyond reach.

Another aspect is the sensitivity of the task from the point of view of the profession. Therefore, it is preferable to refer autopsy to some forensic expert from outside the hospital, thus making way for the expert of the hospital concerned to escape much possible embarrassment. It must, however, be realised that the findings of the autopsy surgeon alone will not be sufficient to explain death; therefore, it is advisable to hold a discussion across the autopsy table involving forensic expert/autopsy surgeon, anaesthetist and the surgeon/clinician concerned.

Autopsy

It may pose certain problems due to surgical intervention and its sequelae, especially in abdominal and thoracic procedures. Introduction of some surgical and anaesthetic devices like airways, indwelling catheters/needles, intravenous cannulae, wound drains, chest tubes, etc. during and subsequent to surgery are likely to interfere with the findings. Their proper placement and patency should be assessed. A full range of specimens for histological, toxicological, bacteriological examinations and those required to exclude hazards associated with blood or fluid transfusions must be collected.

FOR HISTOLOGICAL PURPOSES

Specimens should be taken particularly to exclude any cardiovascular disorder including occult conditions like myocarditis as well as specimens for assessing the severity of disease for which the operation was carried out. Histological examination of the brain is imperative, which is primarily intended to demonstrate the effects of hypoxia, particularly in the region of Sommer's area of the hippocampal gyrus and the cerebellum, where changes are expected even if the victim suffers hypoxia for a short period. Plum found morphological changes in the brains of victims who suffered hypoxia for a short period but survived for long periods after an anaesthetic. His findings included diffuse, severe leucoencephalopathy of cerebral hemispheres with sparing of the immediate subcortical connecting fibres. Demyelination and obliteration of axons was also observed and at times, infarction of the basal ganglia. Damage appeared limited to the white matter which Plum explained on the basis of greater glycolysis in the white matter during hypoxia as compared with the grey matter. (Plum F, Posner JB. *Diagnosis of Stupor and Coma,* 3rd ed., Philadelphia, F Davis and Co 1984, pp 218–19.)

FOR THE PURPOSES OF TOXICOLOGICAL EXAMINATION

- If death due to inhalational agents is suspected (e.g. suspected huffing deaths, deaths from freon, helium, or other gaseous agents, etc.), toxicological specimens should be placed in gas-tight containers. Lung tissue needs be collected in small sealed metal cans.
- To avoid loss of gases due to exposure of the tissues to the air, it may be necessary to obtain samples by the biopsy techniques prior to autopsy and immediately frozen.
- Some fat from the mesentery, some skeletal muscular tissue, some portion of the brain, some portion of the liver, half of each kidney may be retained.
- Blood should be collected under liquid paraffin. Alternatively, glass tubes with aluminium foil or Teflon-lined caps need to be used (volatile substances will otherwise escape through the rubber caps).
- Urine should also be collected, if available.
- Adequate blood, urine and other body fluids may have to be collected for bacteriological and other related issues.

(Specimens should be collected in the containers with as little head space as possible, sealed and immediately refrigerated or frozen as the case may be.)

EXTRANEOUS SPECIMENS

Like residual solutions, medication containers, samples of gases used in the anaesthesia and samples of the operating room air may have to be collected in occasional cases.

Medicolegal Considerations

All deaths occurring during the course of anaesthesia and surgery or within reasonable time afterwards should be reported to the police (CrPC 1973 Section 39). They cannot be regarded as natural deaths. There is a usual tendency on the part of the relatives and/or their counsels to raise a finger of accusation towards the doctor because of death being so closely related in time to the intervention. Hence, the public and private interest would be best served by displaying independence and reporting the issue to the police. The circumstances need not be hushed up just to avoid embarrassment of a colleague of the institution. All too often the tragedy may be due to a combination of errors in varying proportions rather than one particular mistake. However, any such death believed to be caused, or contributed to, by any of these procedures may be adequately investigated both from the point of view of the satisfaction of the relatives of the deceased and instituting future safety/preventive measures.

It is a general rule that those who deprive the patient of his protective reflexes are responsible for any injury that befalls because these reflexes were obtunded. Hence, following precautions are suggested:

- Keep the patient in the recovery room till his condition gets stabilised and protective reflexes return to normal.
- Issue clear and adequate instructions to the nursing staff of the recovery room and also for any specific problem to look for and to guard against.
- Keep the patient under personal supervision at least until all monitoring, mechanical ventilation, drainage of all secretions and the like are properly and efficiently attended to. [A **related case cited in** *The Lancet* 1964;2:971 "Medicine and the law" is of significance: A 13-year-old boy died 9 days after an operation with considerable brain damage at the Coventry and Warwickshire Hospital. During investigations, the anaesthetist said that while he was out of theatre having a coffee break, the supply of oxygen failed. Birmingham Regional Hospital Board appointed a committee of enquiry, which recommended that (*i*) it is anaesthetist's responsibility to see that anaesthetic apparatus is working efficiently throughout an operation and (*ii*) since a prime duty of an anaesthetist is to care for his patient under anaesthesia, he must regard this as a continuing responsibility. Before leaving the theatre, therefore, he must satisfy himself that the patient is under the charge of a competent person, who is familiar with the apparatus and is able to care for an unconscious patient. He should also let the surgeon know that he intends to leave the theatre.]

Apportioning relative contribution between the anaesthetist and the surgeon is extremely difficult and both are required to exercise due care and skill. Each one is responsible for negligent acts of oneself and not of the other as there exists no master–servant relationship between them, though they may be answerable to the hospital administration. As the surgeon possesses absolute control over the staff who assist him in the operation, he will be liable for the negligent acts of his assistants but the surgeon, however, has no absolute control over the activities of the anaesthetist and the connected staff. Under exceptional situations, however, one may become liable for the wrongful acts of the other, which one observes or should have observed and permits to pass unheeded without inviting the attention of the other. In teaching hospitals, apart from the nursing staff and the assistants, interns, residents and fellows, etc. are also considered to be the agents of the institution in which they work. The specific determination of this agency role is based upon the 'issue of control' (control is manifested by the right to hire and fire and give specific directions as to clinical functions and related matters).

Blood Transfusion—Hazards and Risks

Although 'hazard' and 'risk' are interchangeable terms to the general public, but they carry different meanings. 'Hazard' is an

intrinsic property of a substance or situation that in particular circumstances could lead to harm/damage. 'Risk' differs from hazard, as it involves a consideration of the probability/likelihood of a consequence occurring as well as what the consequence might be. The Royal Society Study Group (1983) defined risk as 'the probability that a particular adverse event occurs during a specified period of time or results from a particular challenge'. As a simple illustration, the ability of scalpels, needles, etc. to cause an injury represents a hazard, whereas the likelihood of acquiring HIV or hepatitis B from such an injury represents a risk.

Blood seems to have gained a singular status of being simultaneously feared and revered. Developed countries have come to demand absolute freedom from transfusion-transmitted infection, while at the same time conceding that zero-risk transfusion is unlikely to be achieved ever. By and large, the 'protective/safeguard' system has proved effective. Glynn et al. measured markers of viral exposure in 1.9 million volunteer blood donors at five regional blood centres between 1991 and 1996 and reported an extremely low risk for major transfusion-transmitted viruses.

Today, because of sensitive screening tests being employed, transfusion reactions are extremely rare. However, these tests are usually confined to ABO cross-matching and therefore, possibility of reaction or sensitisation due to incompatibilities in systems other than ABO systems may exist. (Individuals with type AB blood are 'universal recipients' because they have no circulating agglutinins and can be given blood of any type without developing a transfusion reaction due to ABO incompatibility. Type O individuals are 'universal donors' because they lack A and B antigens, and type O blood can be given to anyone without producing a transfusion reaction due to ABO incompatibility.) Approximately 2% of patients who receive blood or blood components develop reactions of some type or other. Complications associated with blood transfusion and the introduction of other fluids into circulation may be broadly grouped into following categories.

IMMUNOLOGICAL COMPLICATIONS

These may be further of two sub-types:

(i) **Intravascular haemolysis** usually takes place due to errors in grouping and cross-matching. The antigens of ABO system commonly produce transfusion reactions since naturally occurring antibodies (anti-A and anti-B) are capable of fixing complement (a group of proteins in the blood that influences the inflammatory process and serve as the primary mediator in the antigen–antibody reactions. It gets activated whenever tissue injury occurs, specifically by neutrophil aggregation, antigen–antibody contact, bacterial endotoxin, and the release of blood coagulation proteins). Simultaneous administration of blood and intravenous fluids of different pH and tonicity may cause haemolysis. Occasionally, transfusion of group O donor blood containing high titre of antibodies, especially from previous transfusion or incompatible pregnancy, may cause lysis of recipient red cells. Haemoglobinaemia, haemoglobinuria, shock and renal failure from acute tubular necrosis are the usual findings.

(ii) **Extravascular haemolysis** is more often due to the presence of antibodies against Rh antigens, and rarely, against antigens of Kell, Duffy, and Kidd systems. In such cases, haemolysis occurs in reticuloendothelial systems mostly in the liver and the spleen. Shock and renal complications are rare. [Rhesus (Rh) blood group system was first discovered on human red cells by the use of antisera prepared by immunising rabbits with red cells from Rhesus monkey. The Rh allelic genes are C or c, D or d, and E or e, located on chromosome 1. One set of three genes is inherited from each parent giving rise to various complex combinations. The corresponding antigens are similarly named as Cc, Ee, and only D, since no d antigen exists. In practice, Rh grouping is performed with anti-D antiserum since D antigen is most strongly immunogenic. Individuals who are D-positive are referred to as Rh+ve, and those who lack D antigen are termed as Rh −ve. Practically, there are no naturally occurring Rh antibodies. Rh antibodies in Rh −ve individuals are acquired from immunisation such as transfusion and during pregnancy, resulting in fatal haemolytic transfusion reaction and haemolytic disease of the newborn.]

(iii) **Other allergic reactions** may include the following:
   (a) **Febrile reaction** is usually attributed to immunologic reaction against white blood cells, platelets or, IgA immunoglobulins.
   (b) **Anaphylactic reaction** may be seen in patients with antibodies against IgA molecule on transfusion of blood from other human subjects.
   (c) Transfusion related graft-versus-host disease mediated by donor T lymphocytes may occur, etc.

NONIMMUNOLOGICAL COMPLICATIONS

These may include the following:

(i) **Circulatory overload:** It may occur in patient with renal or cardiac insufficiency after rapid blood transfusion. The reaction can be prevented by transfusing in sitting position and with well monitoring.

(ii) **Coagulation defect:** Infusion of more than 8–10 units of blood in 24 hours will lead to a fall in the level of labile coagulation factors in the recipient's serum leading to bleeding called 'dilution coagulopathy'.

(iii) **Hyperkalaemia:** Potassium ions leave stored red cells causing a steep rise in the potassium ion concentration in the plasma. Improper handling of stored blood during transportation causes lysis of red cells. Transfusion of such blood poses threat to life in the patient of renal

insufficiency and especially in patients with burns and crush syndrome.

(iv) **Haemosiderosis:** Patients with severe anaemia who have had multiple transfusions may develop haemosiderosis (a condition characterised by the deposition, especially in the liver and spleen, of haemosiderin).

(v) **Air embolism:** If blood is administered rapidly under pressure, air may get into the circulation and cause embolism. This complication can be prevented by using a plastic container instead of usual glass bottle and applying a sphygmomanometer cuff around the container to increase the pressure within the container at will. A debilitated person may develop symptomatic air embolism even if small volume (10–40 ml) makes its way into the circulation, while a healthy individual is at lesser risk.

(vi) **Transmission of infection:** Blood transfusion is at times responsible for transmitting diseases such as AIDS (HIV infection), homologous serum hepatitis, cytomegalovirus disease, infectious mononucleosis, syphilis, malaria, toxoplasmosis and brucellosis, etc. Blood may also get infected during collection and preservation with Gram-negative bacilli, which grow at refrigeration temperature with the release of endotoxin. Introduction of contaminated fluid, even when the organisms are non-pathogenic or dead, can cause severe pyrogenic reactions. Screening of blood has been made mandatory for HIV, HBV, HCV, syphilis and for malarial parasite. The incubation period of viral hepatitis is lengthy (about 3 months) and has been associated with 'pooled serum' or 'plasma'.

INVESTIGATION OF TRANSFUSION REACTIONS

Deaths following blood transfusion are of crucial medicolegal importance, as the relatives of the deceased may level charges of criminal negligence against the doctor. An occasional patient who survives the mishap may claim damages. Investigation should be aimed at finding out whether death or disability was the result of transfusion reactions.

Haematological Examination

The presence of haemoglobin and methemalbumin in the serum of a post-transfusion sample of blood from the patient would indicate intravascular haemolysis (samples of blood obtained postmortem are not good enough for this type of examination due to postmortem lysis of red cells). In extravascular haemolysis, there is an increase in the quantity of unconjugated bilirubin.

Urine Examination

Urine will contain haemoglobin in intravascular haemolysis. Urobilin and urobilinogen may be present. Sediment from centrifuged specimen may show red cell casts.

Serological Examination

A 2% red cell suspension in saline of the post-transfusion sample of blood may show agglutinates. The absence of agglutinates, however, does not rule out haemolysis. In reactions of sensitivity to donor leucocytes, platelets and plasma factor IgA, specific antibodies can be demonstrated.

Bacteriological Examination

If bacterial contamination is suspected, a sample of residual donor blood is collected and centrifuged lightly. A drop of supernatant plasma is smeared, fixed by heating, and stained by Gram's method. Demonstration of several organisms in oil immersion fields will be indicative of bacterial contamination. Blood culture will confirm the diagnosis. *Autopsy* (wherever warranted): In acute intravascular haemolytic reactions, the kidney will show haemoglobinuric nephrosis. Acute tubular necrosis and casts of haemoglobin in the tubules will be seen—the so-called 'transfusion kidney'.

The kidney damage, which can begin within a few minutes to few hours and continue until the person dies of renal failure, seems to result from the factors such as (*i*) release of toxic substances from the haemolysing blood that cause powerful renal vasoconstriction, (*ii*) loss of circulating red cells along with production of toxic substances from haemolysed cells and from the immune reaction, usually leads to circulatory shock and (*iii*) greater amount of free haemoglobin in the circulating blood and therefore, the excess leaks through the glomerular membranes into the kidney tubules causing the tubular haemoglobin concentration to rise, which ultimately precipitates and blocks many of the tubules. Pulmonary oedema will be evident if death occurred from circulatory overload. Subendocardial haemorrhages on the interventricular septum will be demonstrable in deaths from severe shock following the transfusion of infected/contaminated blood. In death from air embolism, the presence of air in the right ventricle of the heart can be demonstrated by underwater dissection.

Death due to Periprocedural Complication

Death due to periprocedural complication may be described as "a death that is known or suspected as having resulted in whole or in part from diagnostic, therapeutic or anaesthetic procedures". These generally fall into two groups: **(*i*) Expected risk** (risk is considered as a chance of encountering harm/injury or loss). Depending upon the experience and concerned data available, certain risks have been recognised. **(*ii*) Unexpected risk** (usually include the circumstances viewed as therapeutic accident or misadventure). **An accident** may be considered as a condition/situation/reaction occurring

unexpectedly without any ill intention or design. The term **misadventure** may be analysed through its construction, i.e. to 'venture' literally implies undertaking hazardous activity/incurring risk/to expose to chance, etc. Prefix 'ad' conveys increase/intensification and prefix 'mis' conveys bad, wrong, improper or negative, etc. Therefore, 'misadventure' may be considered to imply an unfortunate mischance arising out of an act/situation/circumstance. In other words, it may be considered to convey 'an inevitable accident when the avoidance of it would have required a degree of care, exceeding the standard demanded by the law'. Hence, it may commonly form a good defence under the given scenario/situation/circumstance. The term **iatrogenic** is often used in medical field and needs explanation at this juncture—'iatro' is from the Greek, meaning physician and 'genic' is also from Greek, meaning 'generation/producing'. However, in the broader sense, it may be considered to indicate any adverse condition/reaction induced in a patient as a result of diagnostic or therapeutic procedure, medication, etc. This term is usually used in the sense that such adverse condition/reaction could have been avoided by proper and judicious care on the part of the physician, surgeon, or technician, etc.

There has been common public misperception that doctors and the medical arts are infallible. Given the difficulties inherent in the profession itself, medical errors are always possible and are often the end result of a chain of factors. Diversity of human reaction to drugs makes it an intellectual impossibility to establish definite scientific rules governing the use of therapeutic medications. In other words, it may be put like, "Drug pharmacology is not an exact science but rather a scientific humanism". Moreover, in the present time, patients are increasingly cared by a healthcare team rather than by a single doctor. Fragmentation of diagnostic activities through 'medical team approach' and the shift towards an extended responsibility has profound implications. Still further, the efficiency of the team is regulated by the availability of resources on one hand and external agencies influencing the healthcare team on the other, for example, third party payers, fourth party auditors, etc. The situation is often fueled by the mass media that sensationalises the single cases of real or alleged iatrogenic injury in an attempt to gain larger audience shares.

The medicolegal expert has a duty to perform in such cases especially in contributing to the final diagnosis as to the manner of death, i.e. whether it is 'natural' or whether 'unnatural' (accident, misadventure, suicide, homicide, or occasionally, undetermined). Consultation/opinion of requisite experts and critical evaluation of the circumstances would usually provide answers to the related issues.

12
CHAPTER

Custody Related Torture and/or Death

After going through this chapter, the reader will be able to describe: Meaning of custody and torture | Methods of torture | Circumstances of death | Related cases | Role of autopsy surgeon | Incisions at autopsy

Since time immemorial man has been attempting to subjugate his fellow human beings. Over the centuries, with the growth of civilisation, there has been an increased use of violence and torture to twist and turn people around. Custodial torture is a matter of great concern to the Champions of Human Rights because torture, in some way or the other, is aimed at not only to silence the tortured but also to others who may be frightened into passivity. Due to public awareness, Human Rights Commissions have been constituted. The latest developments necessitating reporting of custodial death within 24 hours and conducting a postmortem by a board of doctors including video filming of the procedure speak of the gravity of concern of the Commission.

During the Australian Royal Commission into Aboriginal Deaths in Custody (1991), the issue that whether some deaths actually occurred in custody surfaced glaringly. The Royal Commission recommended that a 'death in custody' should, at least, include the following circumstances of death:

- Those occurring in prison or while in police custody.
- Those caused or contributed to by traumatic injuries sustained, or due to lack of proper care, while in custody or detention.
- Those in which the fatal injury resulted from police or prison authorities attempting to detain a person.
- Those in which a fatal injury resulted on a person escaping or attempting to escape from police custody or juvenile detention.

Custody—Meaning Thereof

The word 'custody' has been derived from Latin 'custos odis' meaning guardian. Black's Law Dictionary describes it as 'the care and control of ownership'/'responsibility for protection and preservation of the thing/person in custody', etc. 'Police custody' does not necessarily mean formal arrest. It also includes some form of police surveillance and restriction on the movements of the person(s). 'Police detention', however, requires that person has been formally arrested and detained. The word 'arrest' when used in the ordinary and natural sense implies the apprehension or restraint or the deprivation of one's personal liberty. When used in the legal sense in the procedure connected with the criminal offences, an arrest consists in taking into custody of another person under authority empowered by law, for the purpose of holding or detaining him to answer a criminal charge or of preventing the commission of a criminal offence. 'Detention' does not mean 'imprisonment'. The word 'imprisonment' is always used in the sense of punishing a person, whereas 'detention' does not denote any punishment.

Torture

Medical profession and human rights are intricately woven in the sense that the doctors can alleviate violations of human rights being among the first to become aware of these violations, particularly in the field of 'Torture'. The definition of 'torture' as we use today is the definition from the UN Convention against Torture and other Cruel, Inhuman or Degrading Treatment or Punishment of December 10, 1984, which entered into force in June, 1987. As per this Convention, **torture is defined as** "Any act by which severe pain or suffering, whether physical or mental, is intentionally inflicted on a person for such purposes as obtaining from him or third person information or a confession, punishing him for an act he or a third person has committed or is suspected of having committed, or intimidating or coercing him or a third person, or for any reason based on discrimination of any kind, when such pain or suffering is inflicted by or at the instigation of or with the consent or acquiescence of a public official or other person acting in an official capacity. It does not include pain or suffering arising from, inherent in or incidental to lawful sanctions".

Methods of Torture

From didactic point of view, torture methods have been divided into **physical, psychological and sexual**. Physical methods of torture challenge any possible classification because of increasing number and variety of methods. Most of the time, torture is selectively tailored to the characteristics of the victims. Despite the fact that there seems to be ongoing research into refining torture methods so as to leave little or no trace of injury, many of the methods used are simple (Thomson et al., 1984).

PHYSICAL TORTURE

The methods for physical torture are those that inflict pain, discomfort and dysfunction in different parts of the body. The torturer also takes care that the torture inflicted upon the victim remains undetected by an ordinary examination. However, despite all precautions, physical torture almost always leaves a trail that eventually leads to its discovery. Methods may include the following.

Beating and Severe Beating

Beating with a variety of objects is very common and will result in usual signs, i.e. bruising, abrasions and/or lacerations. Particular weapons/objects may leave particular pattern, for example, a blow/strike from a rod or thong will commonly result in parallel tramline bruises. Where the skin gets lacerated, the scar left will provide evidence of the site of injury. Susceptible persons may develop keloid scarring. The sites of beating are again variable. Blows to the head, back, buttocks, perineum and soles of feet (falanga) are favourite sites for beating. Weapons/objects employed may include sticks, whips, cables, chains, belts, or other instruments. Similarly, punching with fists and kicking, etc. are also employed for beating. Simple beating means slapping on less sensitive and less delicate parts of the body that does not cause significant external and internal damage. (**Baton** is a short thin stick used by the conductor of a band to direct an orchestra. In athletes, it implies a short stick carried and handed on in a relay race. In relation to police, it implies a short stick that indicates a certain rank and being used to drive a crowd back.) **Poking** the victim with a baton, rod or any other similar object is common. Any part of the body may be poked. The police personnel using baton must be aware of the relationship between the choice of target and the extent of injury that may be produced from the amount of force going to be applied.

Falanga

Severe beating on the soles of the feet is known as falanga. It is one of the most common types of systematic torture used in many countries around the world. Presence of thick skin and dense fascia at this site prevents appearance of any appreciable surface injuries even though deep tenderness may be long lasting after the initial pain and swelling have subsided. Other kinds of systematic beating can be beating on both ears, beating on previous fracture sites or punches in the stomach, e.g. on pregnant women to provoke miscarriage.

Ear Torture

The victim's ear may be twisted or pulled to such an extent that the external ear gets torn. One victim may be asked to torture another in this way. Producing impairment in hearing by beating both ears simultaneously may also be used, which is known as **telephono**. This may lead to rupture of tympanic membrane causing extreme pain, bleeding or hearing loss.

Finger Torture

Pencil or a similar object is put in between two fingers, which are then pressed hard together against the objects. Similarly, fingers may be twisted to cause severe pain.

Hair Torture

The victim is dragged by the hair. The hair may be cut short or the head shaven altogether. Hair may also be pulled out forcibly.

Suspension

The victim is suspended by legs or arms or by hair. Suspension is usually combined with other forms of torture like severe beating, electric shock, falanga, heat or cold torture, etc. (hanging from the feet or ankles while using a rod as the suspending platform is more universal than the 'Parrots perch', a type of hanging that is more frequent in Brazil and Ethiopia).

Forced Position

The victim may be forced to remain in an abnormal or strained position for hours together and may also be exposed to kicks, blows, etc. In some cases, the victim may be tied down in many ways and then kept in this position for several hours. He may be suspended in this position and also beaten.

Electric Torture

This is excruciatingly painful and is commonly used because it leaves little permanent signs. The points of contact of electrodes may leave small lesions in varying stage of healing or scar marks but in general, the residual effects of electricity are almost undetectable in the living. If death occurs during or shortly after electric current, careful histological examination of the sites of electrocution can be helpful in determining the recent electrical injuries.

The electrodes are placed on the most sensitive areas of the body like ears, tongue, gums, fingertips, toes, genitals and nipples. It may be applied inside the mouth, which is quite painful and difficult to detect later. Sometimes the victim is soaked

with water before applying the electricity so that the magnitude of the shock is greatly enhanced and production of burn being prevented.

Suffocation

It is known especially in the form of **wet submarine**. The head of the victim is forced under water polluted with excrement, urine, vomit or blood. The head is forced below the surface until the stage of suffocation, or until the physical reflexes cause aspiration of the contaminated fluid (autopsies of the victims who have died following this type of torture have reportedly shown faeces in the lungs). In **dry submarine**, the victim's head and face are tightly covered with plastic bag or similar article. The victim may also be suffocated by the closing of his mouth and nose with some object or even with bare hands once the victim is tied down in such a way that he cannot exercise resist ance.

Burning or Heat Torture

Burns may be inflicted from anything that will either burn or can be heated, for example, cigarette stubs, cigarette lighters, hot irons, branding irons or molten rubber. The residual lesions will depend upon the site and severity of the burns. Cigarette burn is the most common type of heat torture reported. Sometimes, the victims may be forced to stand for hours in the sun in an atmospheric temperature of more than 30° C. He/she may also be forced to work hard under the scorching heat.

Burns can also be caused by acid and caustic materials, the favourite being sulphuric acid in the form of 'battery fluid'. The fluid nature of the agent can often be understandable by the presence of the typical trickle marks. Scarring will depend upon the depth of the lesion.

Cold Torture

The victim is subjected to varying degree of cold in different ways. He may be forced to sleep on a damp floor, may be forced to stay naked in extremely cold weather.

PSYCHOLOGICAL TORTURE

This type of torture may include several categories, which are as follows.

Deprivation Techniques

Victims are deprived of various necessities so that they are mentally tortured. These techniques may include **sensory deprivation**, where the victim is deprived of various sensory stimuli such as light, sound, etc. The victim may be blindfolded, hooded, kept in a dark room, etc. **Perceptual deprivation**, where victims are deprived of perceptions so that they become disoriented and confused, for example, frequent transfer of victim from one place to another while blindfolded, frequent disturbance of sleep, etc. **Social deprivation**, where victims are deprived of seeing visitors or confined to an isolated cell. **Deprivation of basic needs**, where victims are deprived of basic needs like food, water, medical facilities, clothes, comforts, communication, etc.

Witness Torture

The victims are forced to witness the torture of another prisoner or of family members. In the words of Salvadorian torture survivor, "what happened to me is nothing compared to being forced to witness the torturing to death of other comrades".

Threats and Humiliation

The torturer may perform humiliating acts such as urinating upon victim. Conversely, victim may be forced to perform humiliating acts. It is obvious that humiliation is an important part of sexual torture. Sometimes, the victims are threatened with death. These threats are extended to their family and occasionally they experience **sham executions**. (In this procedure, the perpetrators blindfold the victim and place him before a wall. The victim is told that a vehicle is going to run over him/her and that he/she is going to die. The victim then hears an engine starting and coming towards him/her at full speed. However, as it gets really close to the victim it screeches to a halt.)

Pharmacological Techniques

Various drugs may be used to torture the victim, to facilitate torture, to mask the effects of torture and also as a means of torture, for example, use of drugs to induce self-disclosure, use of muscle relaxants, pain-inducing drugs or psycho-pharmacological drugs, etc. Thomson et al. (1984) cite direct fatal poisoning in a Middle Eastern Country where political prisoners were given a drink of orange juice before their release. It was later found to have contained thallium.

SEXUAL TORTURE

Most investigators, lately, have defined sexual torture as follows:

- Violence against sexual organs such as electric torture in genital areas, the introduction of foreign bodies into the genitalia or rectum.
- Physical sexual assault, such as rape by torturer or other victims, forced masturbation, fellatio and oral coitus.
- Mental sexual assault, such as forced nakedness, sexual humiliation, sexual threats and forced witness of sexual torture.
- Any mixture of these.

Circumstances of Death

Deaths in custody may take place due to varied reasons, which may include inadequate medical facilities, inadequate safety

measures of inmates and negligent behaviour of the prison authorities. However, cases of custodial violence/torture are also being reported and thereby focussing public concern.

A retrospective study of 277 deaths over the 6-year period (January 1990–December 1996) was conducted by Police Research Group (PRG) in England and Wales. This paper categorised deaths into three groups:

(i) 63% were due to deceased's own causal actions, i.e. deliberate self-harm and substance misuse comprising of 17 deliberate overdoses before arrest and 73 self-hangings;
(ii) 29% due to medical conditions (the most common five causes of death were—heart problems, head injury, lung problems, epilepsy and liver problems); and
(iii) 8% deaths were such in which another person's actions might have been associated (including police restraint).

Authors concluded that detention perhaps had little bearing on the death, and drew attention to the need for careful consideration of the term 'custody death'.

DEATHS RESULTING DURING CONFRONTATION WITH POLICE

According to Bittner, the central thread that runs through the police work is that it frequently consists of coping with problems in which force may have to be used. However, police need to be aware of the difference between reasonable force and lethal force. Situations like deaths in police confrontation may be considered pari passu with custody deaths, though technically such deaths may not have occurred in police custody. The use of 'batons' is frequent and has occasionally been implicated in causing fatal head injury as occurred in case of death of a school teacher in London in 1979 (R vs. HM Coroner at Hammersmith, ex parte Peach [1980] QB 211). Suicides have also been reported to occur in association with confrontations with police. Haruff et al. (1994) found 14 such cases from 1984 to 1992 in Marion County, Indiana. All were male, with a peak age between 30 and 34 years.

DEATHS FROM PHYSICAL RESTRAINT

Deaths occurring from physical restraints constitute an important controversial sphere in the investigation of custodial deaths. Restraining someone may become necessary in certain circumstances, especially when dealing with a noncompliant or violent person. Methods of restraint may be varied and at one occasion, more than one method may be employed. Methods may include hand cuffing, arm restraint, neck holds, carotid sleeper, etc. [**Neck hold** is designed to occlude the airways by forearm compression of the front of the neck. Resistance by the victim may worsen the situation in the sense that he/she may contribute to increasing the force on the neck. **Carotid sleeper** is designed to occlude common carotid arteries by compression and producing transient cerebral ischaemia leading to loss of consciousness. The hold is released when the person has been incapacitated and usually complete recovery ensues. Reay and Holloway (1982) assessed the carotid blood flow during compression of neck using ultrasonic and laser Doppler blood flow monitoring devices. They observed that blood flow to head decreased by 85% under the neck hold and reached its lowest point in 6 seconds. Because of their relatively protected positions, vertebral arteries continue supplying blood to the brain. However, the vertebral arterial supply cannot completely compensate for the occluded carotid blood flow and therefore, cerebral ischaemia is the usual result. Another pathway for the ill effects may be through the bradycardia produced due to stimulation of carotid sinus. Such holds, now-a-days, are severely restricted rather banned.]

DEATHS FROM CHEMICAL RESTRAINT

This may include substances used for immobilisation or drug administration. **Irritant spray devices** like chloroacetophenone (CN), orthochlorobenzalmalononitrile (CS), oleoresin capsicum (OC), etc. have been used for such purposes. The increasing use of OC in the United States has been associated with a rise in the number of deaths in custody—from 1 death in 1990 to 26 in 1993—following its use (Granfield et al., 1994). OC is a crude extract of hot peppers. On application to ocular membranes, it causes stinging, lacrimation and blepharospasm, which may vary from involuntary blinking to sustained eye closure. Capsaicin in aerosol causes tingling, coughing and shortness of breath to a varying degree. Immediate decontamination with soap and water (intact skin) or saline (vesiculated skin and eyes) followed by symptomatic measures are usually successful in bringing back visual acuity and nasal breathing in a few minutes.

'Chemical mace agents' like CN and CS have also been in use. [These chemicals, the so-called 'lacrimators', used to be employed for temporarily incapacitating/immobilising the enemy or the public. They were popularly known as 'riot control agents'. The eye damage is complicated by the method of delivery of these agents. The two most common modes/forms of delivery were the pencil-like tear gas gun and the aerosol can, used by law enforcement agencies in the United States under the trade name Mace.]

A couple of cases are being cited below to make the readers apprise of intricacies of the issues related to such deaths.

CASE: DEATH OF A SUSPECT RESULTING FROM INJURIES RECEIVED WHILE BEING CHASED BY THE POLICE

On receiving a tip, police party assumed postures nearby a place where some hardcore miscreants were to visit. Seeing the suspects coming on the motorcycle, police tried to chase them and in the process had to open fire. However, it missed the target and the suspects managed to make their way through narrow lanes. Instinctively, the suspect driving the motorcycle

momentarily turned his head back to gauze distance of the police and in the process, motorcycle struck against an electric pole. The suspect received head injury and the handle of the motorcycle significant dents. Both suspects were caught by the police and put under custody. During night, the one who had received head injury complained of severe headache and requested the police to take him to the hospital. However, the police ignored his request thinking it to be malingering as there was minimal external evidence of injury. Consequently, the suspect died in the custody and the happiness of being successful for an 'important catch' turned into feelings of apprehension. Postmortem examination showed a large longitudinally running basal fracture with accumulation of blood. Insignificant injuries were present on the external surface. This coupled with circumstantial evidence and the account offered by the accompanying suspect led to resolution of various controversies.

Though the mode of injury and its effects may be obvious, various scenarios/situations may be put before the doctor for consideration at a later inquiry and the queries as to the following must cross the mind of the doctor in order to have a comprehensive approach to the problem:

- How the fracture of skull occurred? Is the version compatible with the nature and extent of injury or the injuries?
- Was it prior to detention, which was ignored and no measures taken to provide treatment?
- Any consideration of accidental head injury within the cell itself?
- Is it possible that the detainee has been injured by the police, prison wardens, etc.?
- Any consideration that the detainee has been injured by another prisoner/inmate?
- Queries as to the adequacy of care extended to the detainee who had been found dead in the cell?

(Vide communication from Dr. TD Dogra, Head of Forensic Medicine, AIIMS, New Delhi.)

CASE: DEATH OF A SUSPECT WITHIN A COUPLE OF DAYS AFTER HIS RELEASE FROM THE CUSTODY

A suspect was allegedly beaten by the police on 02.03.2005 at about 2.30 p.m. when police tried to catch him. He was taken to the police station and released in the evening. Since then the suspect was not feeling well and his condition deteriorated on 04.03.2005. He was taken to the hospital where he was declared 'brought dead' at 4.15 a.m. Initially, the police presented the 'police papers' under 174 CrPC. However, the wife of the deceased procured orders from the magistrate for the case to be treated as a case of 'custodial torture'. Accordingly, a postmortem was conducted after observing due requisites. The autopsy did not reveal any external physical injury. Internally, peritoneal cavity showed about 200 ml of greenish fluid. Toxicological analysis revealed presence of ethyl alcohol in the contents of stomach, intestines, liver, spleen, and kidney. Histologically, lungs showed acute pulmonary oedema and pulmonary haemorrhages with emphysema. Kidneys showed ischaemic acute tubular necrosis. Mesentery showed haemorrhagic enteropathy with changes of peritonitis and fat necrosis (injury to the mesentery may damage local arteries without causing severe bleeding, but may occlude or thrombose them; with infarction of the bowel as a consequence).

It is understandable that kicking, stamping, and punching, etc. in the abdomen can lead to serious and intractable bleeding. The victim may not seek immediate help/surgical intervention as the condition may remain unrecognised, especially when the victim is intoxicated. Further, trauma to the upper abdomen can precipitate 'acute pancreatitis' (probably through contusion of the acinar tissue and disruption of the duct system with the subsequent interstitial leakage of enzymes).

Bernard Knight reported a case of a man, who was forcibly restrained by the police (allegedly after an assault during which he, probably, had been struck in the abdomen). Owing to drunkenness, little did he realise his condition and remained for a few hours in the police cell. He collapsed while going to the lavatory, never having complained of any abdominal pain. Autopsy revealed several litres of blood in the peritoneal cavity occasioned through the several tears in the mesentery.

Role of Autopsy Surgeon

Persons held in custody by police or by prison authorities retain their basic constitutional rights, except for their right to liberty and a qualified right to privacy. The occurrence of such deaths arouses public interest and raises volatile emotions amongst family and friends of the deceased, media and politicians; therefore, such deaths require effective handling and investigation. There is often an immediate complaint or rumour by the relatives or media about the ill-treatment shown by law enforcing agencies. The assessment towards accidental, suicidal, homicidal or 'purely natural' nature of death is based on meticulous autopsy coupled with thorough investigation of surrounding circumstances leading to death. This may invite the experience and skill of various investigators whose efforts become complementary in providing some satisfactory conclusion needed to dispel or sometimes confirm allegations that an act of commission or omission on the part of custodians has led to or contributed to the death. In such cases, it is desirable that at the time of autopsy, interested parties such as relatives or their representative and the police/prison authorities be properly heard. To carry out another autopsy at a later date may not be free from hazards because the organs would have already been dissected and samples obtained, wherever necessary. Further, with the passage of time elapsing death, bruises undergo changes including varied degree of spreading and coalescing with surrounding injuries. There may, however, be occasion(s)

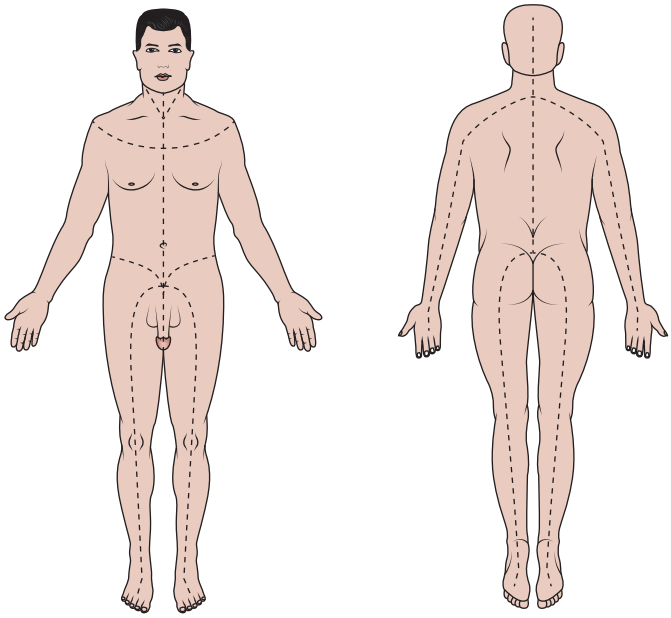


Fig. 12.1 Necessity of incisions on the front as well as back of the body during autopsy and examination/evaluation of relevant specimens/material.

when no clear answer can be approached despite exhaustive exercises.

Incisions at autopsy deserve special mention. Physical methods of torture challenge any possible classification and most of the time, torture is selectively tailored to the characteristics of the victims. This calls for meticulous scrutiny of front as well as back of the body. In addition to the usual Y-shaped front incision, backside of the body also needs be scrutinised especially against regions of shoulders, buttocks, back of thighs and calf muscles, etc. (Fig. 12.1 shows the necessity for incisions on the front as well as back of the body).

Critically examining the body from front as well as back pays dividends, especially when there is a suspicion of crush injuries (probably produced through moving a roller upon selected areas or through other means). In such a case, there may be considerable bruising and crushing of the muscles including soft parts with minimal evidence of surface findings (CR vs. Hopeley, under the Chapter "Complications of Trauma"). **This was typically observed in a case** wherein the victim (about 38-years male) had been apprehended under the Arms Act and died while under custody. At autopsy (conducted by a board of doctors led by Dr. Dalbir Singh, Head of Forensic Medicine at PGIMER, Chandigarh), apart from gross discoloration of skin surface (bluish-black, reddish at places) no trauma was appreciable. However, on exploration, reddish black blood was present in the soft tissues and muscles of the whole of front and inner back of thighs and back of right leg. Chemical examination revealed negative results. Serum myoglobin was estimated to be 108 μg/L (normal <85 μg/L). Histopathology revealed massive necrosis of skeletal muscles (from different sites). Skin tissue (from different sites) showed necrosis and inflammation. Lung showed acute pulmonary edema, and kidney showed acute tubular necrosis. Death was attributed to "acute renal failure due to myoglobinuria caused by blunt force trauma to the lower limb muscles".

13
CHAPTER

Injuries: Medicolegal Considerations and Types

After going through this chapter, the reader will be able to describe: Wound, trauma, injury, etc. | Mechanism of production of mechanical injuries | Differentiation between antemortem and postmortem wounds | Wound healing | Important Sections of IPC relating to offences against the human body | Simple, grievous and dangerous injuries | Classification of injuries

A **wound** may be defined as 'Solution of the natural continuity of any of the tissues of the living body'. The injury may be visible externally but not essential, as fatal internal injuries may be inflicted in the absence of any external mark of violence. The definition includes burns from fire/heat, electricity, all lacerations and bruises of internal organs/tissues and the effects of any corrosive or solid upon the body. It also prevents any possible criticism about the skin being or not being severed. The definition also does not make any reference as to how or by what means it is produced and therefore is simple and generalised one. The word '**trauma**', as generally understood, means an insult to the living tissue. It applies as well to emotional or mental stress. The term '**injury**', under Section 44 of IPC, denotes any harm whatever illegally caused to any person, on body, mind, reputation or property. It will include any tortuous act also.

The characters of an injury caused by some mechanical force are dependent upon:

- The nature and shape of the weapon.
- The amount of energy in the weapon or instrument when it strikes the body.
- Whether inflicted on a moving or a fixed body.
- The nature of the tissues involved.
- The area over which the force acts.

The well-known formula—Force = $\frac{1}{2}MV^2$—is applicable in producing the wounds, where M is the mass and V is the velocity of the object/weapon. It is clear from the formula that **velocity is much more important** than the weight of the weapon used. For example, a brick pressed against the skull may cause minor abrasions or contusions but the same brick when thrown against the head at some velocity may smash the skull. This principle holds good not only in relation to blunt injury but also in missile injuries and stab wounds, etc. Furthermore, the area over which the force acts, forms another important factor, i.e. the damage to the tissues will be far greater if the narrow edge of the brick is applied than if the impact is from the flat surface. (Weapon: The variety of articles that may be put to use as weapons is, of course, without limit. It may be described as any article made or adapted for use for causing injury to the person or intended by the person having it with him, for such use by him. Sections 324 and 326 of IPC describe a **dangerous weapon** as any instrument used for shooting, stabbing or cutting or for offence and likely to cause death.)

Was the Wound Inflicted During Life or After Death?

In cases of death by physical violence, there is always one major point to be determined—whether the injuries were inflicted before death (intra vitam) or after death (postmortem). **Main points to determine antemortem/postmortem nature of the wound are (Table 13.1):**

- Haemorrhage (externally and into tissues).
- Retraction of the edges of the wound.
- Signs of inflammation or of repair.

HAEMORRHAGE

There is more or less copious haemorrhage in all the wounds inflicted during life except occasionally when the victim dies immediately from a fatal injury and shock. The effused blood is forced into the tissues in the vicinity of the wound and is found

Table 13.1 Distinguishing Features of Antemortem and Postmortem Wounds

Features	Antemortem wounds	Postmortem wounds
Haemorrhage	Usually copious, showing signs of arterial spurting	Comparatively very small, may even be absent
Wound edges	Swollen, everted and retracted except those on the neck and scrotum	Not swollen or retracted but are apposed to each other unless inflicted within a couple of hours of death (when muscles remain still contractile)
Extravasated blood	Extravasated blood will extensively infiltrate in and around injured areas with staining of tissues, which will resist washing	No extravasation and infiltration of tissues, no staining of injured tissue (if at all present, will be easily washable)
Blood clot	Coagulated blood is noticed in and around injured tissues. The clot is laminated and firmly adherent to the lining endothelium	Blood is usually not clotted. The clot, if found, is nonlaminated and weakly adherent to the lining endothelium
	Clot is rubbery and firm. On being pulled out from the vessel, it will come out like a horse tail because of elasticity	Clot is soft and friable. On being pulled, will invariably break due to absence of elasticity
	The surface shows apparent lines of Zahn (these lines are formed by alternate layers of light staining aggregated platelets admixed with fibrin meshwork and dark staining layer of red cells)	The surface shows yellow (chicken fat) appearance occasioned through separation of plasma and leucocytes covering the underlying dark red cell constituents (currant jelly) because of sedimentation after death
	Microscopically: composed of fibrin, platelets and RBCs	Mainly composed of fibrin and RBCs
Vital reaction	Sings of inflammation and repair are demonstrable depending upon the age of the wound/injury (antemortem bruises show colour changes)	No signs of any form of inflammation or repair (postmortem bruises do not show colour changes)
Microscopy	Leucocytic emigration appreciable in the surrounding tissue as to the age (neutrophils dominating for the first 6–24 hours)	Vessels distended with postmortem clot without showing any cells outside the vessel wall
Enzyme histochemistry	Adenosine triphosphatase +ve (as early as 1 hour) Aminopeptidase +ve (at about 2 hours) Acid phosphatase +ve (at about 4 hours) Alkaline phosphatase +ve (at about 8 hours)	No enzyme activity
Wound biochemistry	Serotonin peak (within about 10 minutes) Free histamine peak (within about 20–30 minutes)	Nil Nil

Vitality of wounds in decomposing bodies and bodies recovered from water needs careful evaluation. **In decomposing bodies**, there will be associated colour and other changes in the tissues. Wounds need be explored to disclose evidence of infiltrated blood into the tissues that tends to persist. **In the bodies recovered from water**, the wounds (though inflicted during life) become pale and lose the expected red vital reaction because blood is gradually leached out of the wounds by the water over time, and the area will appear bloodless, making the differentiation difficult on the basis of surface appearance. Factors that could help differentiation of antemortem from postmortem wounds under such circumstances may include:

(i) haemorrhage around the wound track;
(ii) location, nature and pattern of wounds/injuries and
(iii) congruency/incongruency in light of other autopsy findings, scene findings, and terminal events.

infiltrated into the cellular and muscular tissues. Consequently, staining of the edges of the wound and the neighbouring tissues occurs. In a wound inflicted after death, even when the body is still warm, these features are far less pronounced. It would be fair to say that the **quantity of blood lost** may assist in determining the antemortem or postmortem origin of wound. When the body has been moved and all marks of blood effaced by some means or the other, rules of this kind may invite further scrutiny.

Another marker helping to assess antemortem/postmortem nature of blood may be the **examination of the blood clot(s)** at the scene. Antemortem bleeding causes coagulation,

when the blood partly solidifies after separation of serum. The clot can be taken out en masse from the spot and the area usually retains the impression of fibrinous network due to process of clot formation. Postmortem solidification occurs without proper coagulative change; on removal from the spot, it does not leave the impression of fibrinous network. Furthermore, the blood that has effused during life can be separated into scales on drying, whereas the blood that has flowed after death tends to break into powder on drying.

Clotting of blood occurs normally in about 5–10 minutes; therefore, clots of blood will be found in the wounds and in the tissues and in the area adjacent to the body. However, bleeding in the pleural cavity because of rapid defibrination due to movement of the lungs does not usually show clotting. There has been some controversy about the conditions that lead to the fluidity of blood after death in certain cases. The observations obtained from in vitro and in mortuo investigations (Mant, AK, 1953. In: Simpson, K, Ed. *Modern Trends in Forensic Medicine,* London, Butterworths) provide a summary in this context [see Livor Mortis (Postmortem Hypostasis) in Chapter 4 "Death and its Medicolegal Aspects (Forensic Thanatology)"].

Sign of spouting of blood is another important factor in favour of antemortem process. The spouting may be present on the body, clothing or in the vicinity. But the spraying of blood by swinging a blood-covered weapon or splashing out of a wound by repeated blows, etc. should also be kept in mind. Christison's experiments lead to the conclusion that severe blows inflicted on a body recently dead produce no greater degree of ecchymosis or cutaneous discolouration than slight blows inflicted on the living.

RETRACTION OF WOUND EDGES

During life, healthy skin is slightly on the 'stretch' and so are the muscles in a condition of 'tone'. This causes the wounds to gape. But this elasticity does not cease at the moment of death and muscles also retain tone for sometime after death; therefore, the skin wounds inflicted before or shortly after death will retract, though the degree of gaping may vary and be appreciated by an experienced doctor.

SIGNS OF INFLAMMATION OR OF REPAIR

There are signs of vital reaction and will depend upon the period of survivability of an individual after infliction of wound. If the vicinity of the wound shows swelling, extravasation of lymph, blood or adhesion of edges, etc., it not only indicates that the wound was inflicted during life but also may give some indication as to the time of its infliction. If wound has become infected, pus may be seen after a period of about 36 hours. Once infection has supervened, healing may be delayed and it is often impossible to determine the age of the wound with any degree of accuracy.

Wound Healing

Questions regarding the age of the wound(s) may be raised under various circumstances so as to match the consistency or inconsistency in their sustaining/infliction as per account given by the victim/assailant. This can be assessed from the process of repair of the wound. In this context, it may be kept in mind that healing is not a distinct series of events but a concert of simultaneously occurring processes, some of which may continue for weeks or months after the physical integrity of the wounded tissue has been re-established. Some idea about the various changes and their timings can be obtained from the following description (here, I would like to describe briefly the healing of skin wounds, a process involving both epithelial regeneration and the formation of connective tissue scar).

HEALING BY FIRST INTENTION (PRIMARY UNION)

One of the simplest examples of wound repair is the healing of a clean surgical incision approximated by surgical sutures with a minimal loss of tissue and the repair occurring without significant bacterial contamination. As mentioned earlier, healing starts very early when the process of inflammation sets in. This process has three major components: (*i*) alternations in the vascular calibre that leads to a focal increase in blood flow (vasodilatation), (*ii*) structural changes in the microvasculature that permit plasma proteins to leave the circulation and (*iii*) emigration of the leucocytes from the microcirculation and their accumulation in the focus of injury. These components account for the earliest 'Triple Response' following the injury, i.e. heat (calor), redness (rubor) and swelling (tumour). Various events may be summarised as under:

Fresh	Bleeding may still be present or there may be fresh soft clot present at the site. Margins are swollen, red and tender.
By 12–24 hours	Margins appear swollen and red. Blood clot and lymph dry up. Histologically, leucocytic infiltration is appreciable. The identity of the emigrating leucocytes varies depending on the nature of the inciting stimulus and also changes as the inflammatory site ages. In most forms of acute inflammation, neutrophils predominate for the first 6–24 hours and are followed by monocytes in the subsequent 24–48 hours. In addition, neutrophils are rather short-lived, undergoing apoptosis within 24–48 hours after exiting the blood stream, while monocytes

	survive substantially longer and may persist for long periods as tissue macrophages. (Fateh A, 1966, noted polymorphonuclear infiltration after only 8 hours of injury in his studies on the human skin wounds.)
By 24–48 hours	Epithelial cells from both edges migrate and proliferate along the dermis, meeting in the midline beneath the surface scab, yielding a continuous but thin epithelial layer. Dominance of monocytes during this period is also exhibited as mentioned above.
By 2–3 days	Neutrophils have largely been replaced by macrophages and granulation tissue invades the incisional space. (The granulation tissue is an immature highly vascular connective tissue that on gross examination appears granular, hence the name.) Collagen fibres are now evident in the margins of the incision but at first, these are vertically oriented and do not bridge the incision. Epithelial cell proliferation continues, yielding a thickened epidermal covering layer.
By 4–5 days	Neovascularisation reaches its peak as the granulation tissue fills the incisional space. Collagen fibres become more abundant and begin to bridge the incisional gap.
By about a week	Epidermis recovers its normal thickness and differentiation of surface cells yields a mature epidermal architecture. A soft, reddish scar is left.

HEALING BY SECOND INTENTION (SECONDARY UNION)

When cell or tissue loss is more extensive, as in inflammatory ulceration, abscess formation, or even large wounds, the reparative process is more complex. In these situations, regeneration of parenchymal cells alone cannot restore the original architecture. As a result, there is extensive ingrowth of granulation tissue from the wound margin, followed in time by accumulation of extra collagen material (ECM) and scarring. This form of healing is referred to as secondary union or healing by second intention.

Secondary healing differs from primary healing in several respects:

- Large tissue defects intrinsically have a greater volume of necrotic debris, exudate, and fibrin that must be removed. Consequently, the inflammatory reaction is more intense with greater potential for secondary inflammation-mediated injury.
- Much larger amounts of granulation tissue are formed. Larger defects accrue a greater volume of granulation tissue to fill in the gaps in the stromal architecture and provide the underlying framework for regrowth of tissue epithelium. A greater volume of granulation tissue generally results in a greater mass of scar tissue.

HEALING OF A FRACTURE

Fracture is a complete or incomplete break in the continuity of a bone. Although there is a distinct history of trauma in most of the fractures, cases of fatigue fracture and of pathological fracture may not show such history. In case of malicious injuries, an accurate account of any incident may be deliberately withheld. A fracture is no more than a wound of bone and as such is subject to the same principles of healing that are applicable to all tissues. It is, therefore, evident that an orthopaedician dealing with fractures needs first be a physiologist and a clinician, and then only carpenter and an engineer.

Diagnosis of Fracture

The presence of fracture can nearly always be inferred from the history and clinical examination. However, clinical evidence must always be confirmed or refuted by radiological examination. Some fairly constant signs of fracture that should arouse suspicion include the following:

- Local swelling
- Local tenderness
- Visible or palpable deformity
- Impairment of function

A couple of more cardinal signs are following:

- Abnormal mobility between the fragments
- Crepitus or grating when the injured part is moved. Depending upon the communication of a fracture with the environment, it may be a **closed fracture** wherein the skin remains intact, protecting the fracture from the external environment or an **open fracture** wherein the skin over the fracture site is disrupted, and the fracture fragments are open to the external environment with every chance for contamination.

Stages of Healing

Healing of a fracture proceeds through a number of overlapping stages until the bone is consolidated. It may be kept in mind that the pattern of healing is not constant for all bones and in all circumstances. The repair of a tubular bone shows striking differences from the repair of a cancellous bone. For the purpose of simplicity, process of healing in a tubular

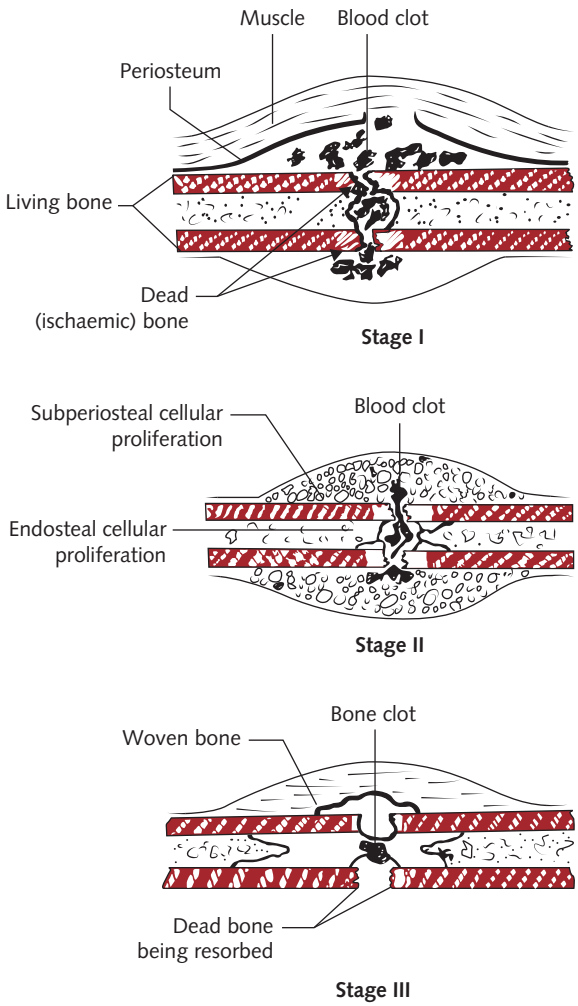


Fig. 13.1 Diagrammatic presentation of stages of healing.

bone fracture may be considered occurring in the following stages (Fig. 13.1):

Stage of Haematoma Formation

Haematoma gets formed between and around the fracture surfaces, and a ring of bone immediately adjacent to the fractured ends becomes ischaemic over a variable length.

Stage of Subperiosteal and Endosteal Proliferation

Proliferation of cells occurs from deep surface of periosteum. These cells are the precursors of osteoblasts and surround each fragment of the fracture. Similarly, cells from endosteum and the bone marrow also proliferate and try to bridge the gap.

Stage of Callus (Woven Bone) Formation

Osteoblasts lay down intercellular matrix of collagen and polysaccharide, which becomes impregnated with calcium salts to form immature bone. This is termed as 'woven bone'. This mass of callus or woven bone is visible in radiography and imparts the earliest radiological indication of a uniting fracture.

Stage of Consolidation

The woven bone that forms the primary callus (soft callus) is gradually transformed by the activity of osteoblasts into more mature bone (hard callus).

Stage of Remodelling

Newly formed bone often forms a bulbous collar surrounding the bone and obliterates the medullary canal. The size of this bulbous mass varies depending upon factors like extent of haematoma and displacement of fragments. The bone is gradually strengthened along the lines of stress at the expense of surplus bone outside the lines of stress. This is called 'remodelling'. In children, remodelling after fracture is usually so perfect that the site of fracture may become indistinguishable in the radiographs. However, in adults, remodelling usually falls short of this perfection, and the site of fracture is usually permanently marked by an area of thickness.

Has a Bone ever been Fractured?

Fractures have many important bearings in relation to medical jurisprudence. They may result from falls, blunt or sharp impact, or at times, a weakened bone may disintegrate or fracture spontaneously (as in old age when the bones are more porous, fragile and brittle). In criminal cases, reports have been available wherein the defence raised the contention of abnormal condition of the bones but the courts have been of view that an assailant, intending hurt, has to take his victim as he finds him.

The question, 'has a bone ever been fractured?' may sometimes be put to the expert in reference to the living body. It is well-known that a bone seldom unites so evenly that the point of the bony union is not indicated by a node or some irregularity/thickening, etc. Some bones like radius, collar bone, tibia, etc. can well be palpated for such an examination. In others, detection is difficult by palpation. X-ray examination is always warranted to localise an old lesion. However, in case of skull, things may not be as straightforward as are usually expected. **This is well-substantiated by an instant case:** The victim, about 45-year-old male, reported to the emergency with history of assault on the head with sharp edged weapon. On examination, an incised wound measuring 3.5 cm × 0.75 cm was found on the right parietal region. On X-ray, underneath fracture was present. Therefore, injury was declared as 'grievous' by the examining doctor. However after about 3 months, the opposite party approached the court asserting that they had been falsely implicated in the case and requested for re-examination of the victim by a board of doctors. The board of doctors got conducted another X-ray wherein no fracture was demonstrable. Though surprising, yet one should not get

alarmed at such finding of nondemonstration of fracture in the X-ray after about 3 months since repair of fractures of the skull is usually attended with a very slight amount of callus, probably owing to the absolute rest of the fragments. The edges of fissured fracture are usually glued together within a week or are gradually smoothened within 3–4 weeks and are united by the formation of bone within 2–3 months or more.

Raekallio in his studies of injured human skin noted the **differences in the activity of various enzymes** at varying intervals. Diminished activity may be demonstrable at the central zone (injured area) and increased activity at the peripheral zone. It was noted that the activity of esterases and adenosine triphosphatase increased as early as 1 hour after the injury, aminopeptidase in about 2 hours, acid phosphatase in about 4 hours and alkaline phosphatase in about 8 hours. Such determination of enzyme activity by means of histochemistry helps in determining the age of the wound in the early phase of healing (before cellular infiltration has taken place) and in differentiating between postmortem and antemortem wounds. Postmortem wounds do not show a central zone of diminished enzyme activity and a peripheral zone of increased enzyme activity. These changes can be demonstrated for a few days after death, if autolysis is prevented by refrigeration (Fig. 13.2).

Biochemical methods have also been employed to ascertain antemortem or postmortem status of the wounds. Histamine and serotonin are vasoactive amines known to participate in the acute inflammatory process, especially in the earliest phase after the injury. The maximum increase in the free histamine content occurs within 20–30 minutes after wounding, and the increase in serotonin content is demonstrable still earlier, the maximum increase occurring within 10 minutes after the injury, as reported by Raekallio J and Makinen PL.

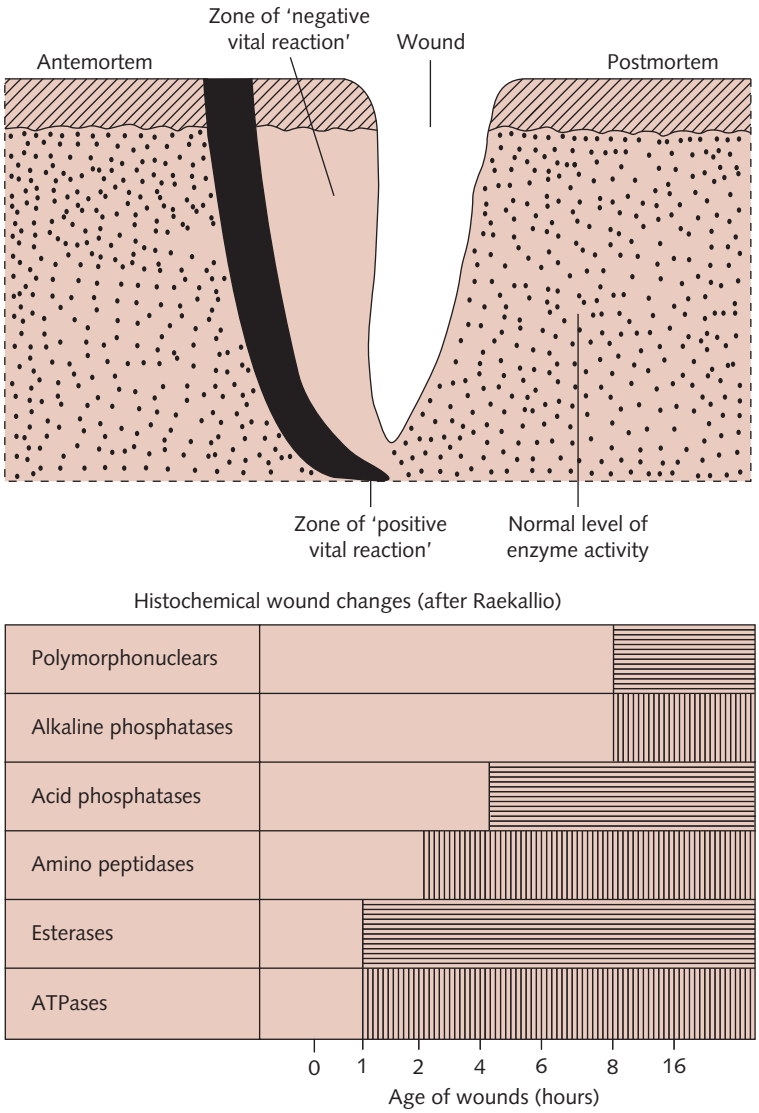


Fig. 13.2 Schematic diagram showing the histochemical estimation of the age of antemortem skin wounds (after Raekallio).

Fabricated Wounds

Also known as 'fictitious/forged' wounds; there are usually superficial injuries mostly produced by a person on his own body (*self-inflicted*) or occasionally caused by another person acting in agreement with him (*self-suffered*). The fabricator usually produces or causes to be produced only that much injury as he thinks necessary to confirm his story. The injuries are, therefore, usually multiple, superficial and not situated over vital parts of the body. Such wounds are commonly on the front of the body, but may be on those parts of the back that can easily be approached by hands or on the top of the head. The direction varies according to the site; for example, from above downwards and inwards on upper arm or multiple oblique or vertical interlacing superficial incisions on the abdomen. Though incisions are the usual wounds, yet punctures or other wounds can be there. Contused or lacerated wounds are rarely fabricated on account of the pain occasioned by them and the force required to produce them. Still rare are the firearm injuries and burns.

The object may be to support a false charge of assault or attempted murder against an opponent, to augment the seriousness of the injuries that one has already received during a quarrel/scuffle, to prove self-defence in an accusation of assault or murder, or to substantiate a charge of violence and robbery in a case where one had appropriated money or valuables placed under one's charge. Sometimes the injuries are inflicted to obtain release from army service. Soldiers and policemen may inflict such injuries to bring a false charge of beating against officers. Watchmen, servants or policemen may pose for sustenance of such injuries to avoid charge of collusion in theft or robbery. In all cases of suspected fabrication of wounds, examination of clothing is valuable. The individual will almost always bare the part before wounding it, in order to see what he is about. He may then forget to make cuts/defects in the clothing or even when the clothing are damaged, they may show cuts defects incompatible with the site, number, direction and nature of the wounds upon the body. (A study conducted by Dr. J Gargi et al. furnishes rich information about intriguing aspects of such injuries.)



Fig. 13.3 Photograph showing puncture wounds over upper chest and adjoining upper limb on the right side. The victim, a middle-aged man, posed himself to have suffered pellet injuries at the hands of some miscreants. However, radiological examination did not reveal any pellet neither in the chest wall nor in the cavity. Investigating agency too revealed that the case was concocted one, and the puncture wounds had been managed by using some heated *"sooa"*.

Not to talk of sharp or blunt force injuries, fake cases of firearm injuries may also be seen. In a case reported in *The Times of India* dated 5th October, 2002, two persons were admitted to the hospital claiming that they had received pellet injuries through the attack of their opponents. However, on investigations, the police had to cancel FIR when the case was found to be spurious. Doctors could not retrieve any pellet from the wounds present in the legs of the victims. The police asserted that such cases were mostly linked up with the land holding disputes. Interestingly, as reported, the four holes on the leg of one victim appearing to be of pellets covered a bigger circumference than ones etched out in the cloth over it. Supposing the cloth could have been in a folded position at the time when the shots were fired, the cloth should have unfolded to cover bigger area (circumference) than one on the leg. At occasions, victims can even collude with doctors who can etch out a small hole and implant a pellet inside the leg or arm of the victim. Figure 13.3 shows photograph of another case of false pellet injury.

Important Sections Related to Offences Against Human Body

HOMICIDE

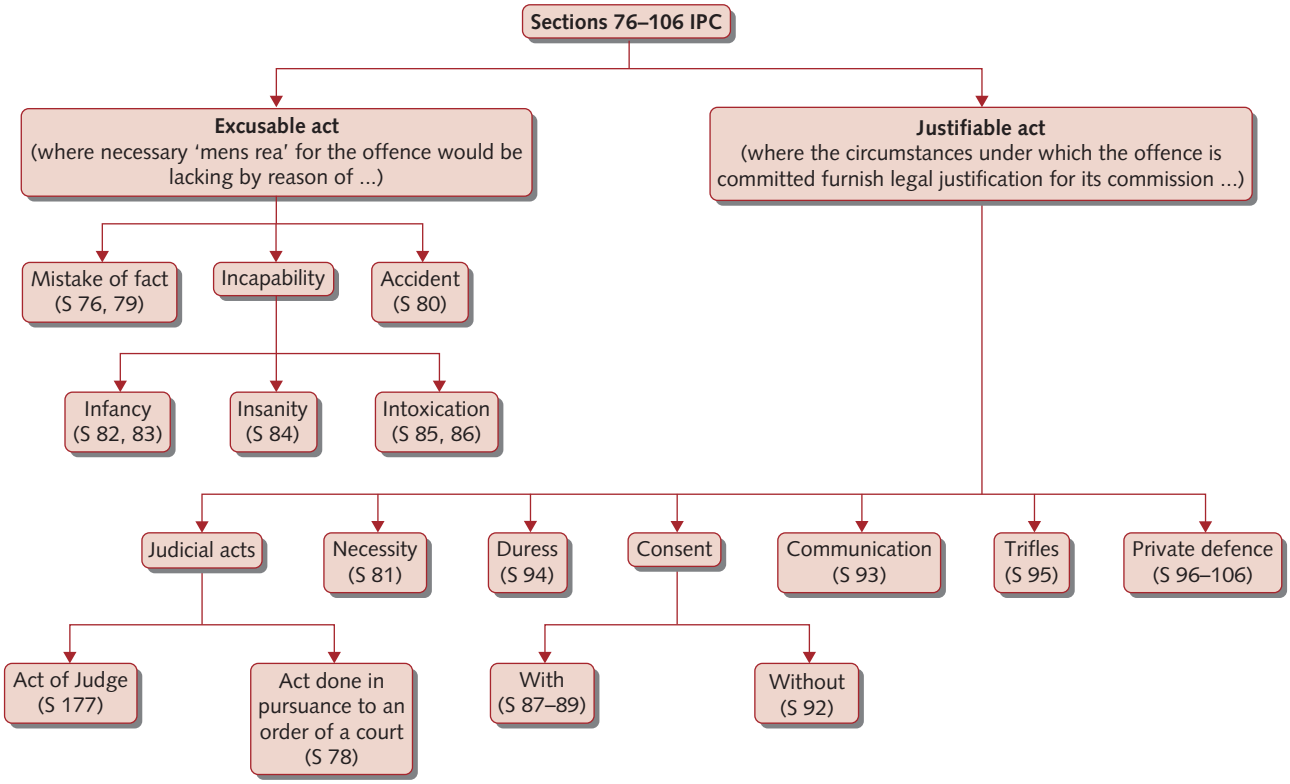
It means killing of a human being by a human being. Broadly speaking, it may be considered as destruction of human life by the act, agency, procurement or culpable omission of some other person(s). Homicide by starvation and medically curable disease whose obvious presence is purposefully or willfully ignored by the person shouldering the responsibility of the child may also be included under this.

Lawful Homicide (Flowchart 13.1)

Excusable Homicide

This includes homicides that are committed with no criminal intention and knowledge. For example:

- Where death is caused by an accident or misfortune, and with no criminal intention and knowledge in the doing of a lawful act in a lawful manner, and with proper care and caution (IPC S 80); or
- Where death is caused by a child or a person of unsound mind or an intoxicated person (IPC S 82, 83, 84, 85); or



Flowchart 13.1 Scheme of general exceptions as per Indian Penal Code.

- Where death is caused unintentionally by an act done in good faith, for the benefit of the person killed, when
  - The person killed is minor or lunatic, his guardian has expressly or impliedly consented to such an act (IPC S 87, 88, 89) or
  - It is impossible for the person killed or his guardian to signify consent in time for the thing to be done for the benefit of the person concerned (IPC S 92).

Justifiable Homicide

A homicide is considered in law to be justified if death is caused:

- By a person who is bound or by a mistake of fact, in good faith believes himself to be bound, by law (IPC S 76) or
- By a person who acts pursuant to lawful authority or by reason of a mistake of fact, in good faith believes himself so authorised (IPC S 79) or
- By judge when acting judicially in the exercise of any power that he possesses or in good faith believes that he possesses under the law (IPC S 77) or
- By a person acting in pursuance of the judgement or order of a court of justice (IPC S 78) or
- By a person acting with no criminal intention to harm and in good faith to avert other harm to person or property (IPC S 81) or
- By a person exercising his right of private defence (IPC S 96–106).

Unlawful Homicide

Culpable Homicide—Section 299

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.

Explanation 1: A person who causes bodily injury to another who is labouring under a disorder, disease or bodily infirmity and thereby accelerates the death of that other shall be deemed to have caused his death.

Explanation 2: Where death is caused by bodily injury, the person who causes such bodily injury shall be deemed to have caused the death, although by resorting to proper remedies and skilful treatment, the death might have been prevented.

Explanation 3: The causing of the death of a child in the mother's womb is not homicide. But it may amount to culpable homicide, if any part of that child has been brought forth, though the child may not have breathed or been completely born.

Murder—Section 300

Except in the cases hereinafter excepted, culpable homicide is murder:

- **Firstly,** if the act by which the death is caused is done with the intention of causing death.

- **Secondly,** if it is done with the intention of causing such bodily injury as the offender knows to be likely to cause the death of the person to whom the harm is caused.
- **Thirdly,** if it is done with the intention of causing bodily injury to any person and the bodily injury intended to be inflicted is sufficient in the ordinary course of nature to cause death.
- **Fourthly,** if the person committing the act knows that it is so imminently dangerous that it must in all probability cause death; or such bodily injury as is likely to cause death, and commits such act without any excuse for incurring the risk of causing death or such injury as aforesaid.

Exception 1: Culpable homicide is not murder if the offender, while deprived of the power of self-control by grave and sudden provocation, causes the death of the person who gave the provocation or causes the death of any other person by mistake or accident.

The above exception is subject to the following provisos:

- That the provocation is not sought or voluntarily provoked by the offender as an excuse for killing or doing harm to any person.
- That the provocation is not given by anything done in obedience to the law, or by a public servant in the lawful exercise of the powers of such public servant.
- That the provocation is not given by anything done in the lawful exercise of the right of private defence.

Exception 2: Culpable homicide is not murder if the offender, in the exercise, in good faith of the right of private defence of person or property, exceeds the power given to him by law and causes the death of the person against whom he is exercising such right of defence without premeditation, and without any intention of doing more harm than is necessary for the purpose of such defence.

Exception 3: Culpable homicide is not murder if the offender, being a public servant or aiding a public servant, acting for the advancement of public justice, exceeds the powers given to him by law, and causes death by doing an act that he, in good faith, believes to be lawful and necessary for the due discharge of his duty as such public servant and without ill-will towards the person whose death is caused.

Exception 4: Culpable homicide is not murder if it is committed without premeditation in a sudden fight, in the heat of passion, upon a sudden quarrel and without the offender's having taken undue advantage or acted in a cruel or unusual manner.

Exception 5: Culpable homicide is not murder when the person whose death is caused, being above the age of eighteen years, suffers death or takes the risk of death with his own consent.

Culpable Homicide and Murder Distinguished

The distinction between these two offences is very ably set forth by Melvill J in Govinda's case. For convenience of comparison, the provisions of Sections 299 and 300 may be stated thus in Table 13.2.

As per Table 13.2, (a) and (1) show that where there is an intention to kill, the offence is always murder.

Under (c) and (4) there is no intention to cause death or bodily injury. Furious driving, firing near a public road would be cases of this description. Whether the offence is culpable homicide or murder depends upon the degree of risk to the human life. If death is the likely result, it is culpable homicide; if it is the most probable result, it is murder.

Under (2), the offence is murder, if the offender knows that the particular person injured is likely, either from peculiarity of constitution, or immature age, or other special circumstances, to be killed by an injury that would not ordinarily cause death.

There remain to be considered (b) and (3) and it is on the comparison of these two clauses that the decision of doubtful cases must generally depend. The offence is culpable homicide if the body injury intended to be inflicted is likely to cause death; it is murder, if the injury is sufficient to cause death in the ordinary course of nature. The distinction is fine but appreciable. It is the question of degree of probability, for example, a blow with a fist or a stick on the vital part may be likely to cause death, whereas a wound from the sword on

Table 13.2 Distinguishing Features of Culpable Homicide and Murder

A person commits culpable homicide if the act by which the death is caused is done:	Subject to certain exceptions culpable homicide is murder, if the act by which the death is caused is done:
(a) with the intention of causing death	(1) with the intention of causing death
(b) with the intention of causing such bodily injury as is likely to cause death	(2) with the intention of causing such bodily injury as the offender knows to be likely to cause death of the person to whom the harm is caused
(c) with the knowledge that the act is likely to cause death	(3) with the intention of causing bodily injury to any person, and the bodily injury intended to be inflicted is sufficient in the ordinary course of nature to cause death (4) with the knowledge that the act is so imminently dangerous that it must in all probability cause death, or such bodily injury as is likely to cause death

a vital part is sufficient in the ordinary course of nature to cause death. The difference between culpable homicide and murder is only a question of different degrees of probability that death would ensue. It is culpable homicide where death must have been known to be a probable result. It is murder where it must have been known to be the most probable result.

It cannot be said that an injury sufficient in the ordinary course of nature to cause death is an injury that inevitably and under all circumstances must cause death. Even if none of the injuries by itself is sufficient to cause death in the ordinary course of nature, cumulatively such injuries may be sufficient in the ordinary course of nature to cause death.

Culpable Homicide by Causing Death of a Person Other than the Person Whose Death was Intended—Section 301

This Section lays down that culpable homicide may be committed by causing death of a person whom the offender neither intended nor knew himself to be likely to kill. This Section embodies what the English authors describe as the doctrine of transfer of malice or the transmigration of motive. If A intends to kill B but kills C, whose death he neither intends nor knows himself to be likely to cause, the intention to kill C is by law attributed to him.

Punishment for Murder—Section 302

Whoever commits murder shall be punished with death or imprisonment for life and shall also be liable to fine. Death sentence is given only in 'rarest of rare cases'.

Punishment for Culpable Homicide not Amounting to Murder—Section 304

Under this Section, there are two kinds of punishments applying to two different circumstances:

- If the act by which the death is caused is done with the intention of causing death or such bodily injury as is likely to cause death, the punishment is imprisonment for life or imprisonment of either description for a term that may extend to 10 years and fine.
- If the act is done with the knowledge that it is likely to cause death but without any intention to cause death or such bodily injury as is likely to cause death, the punishment is imprisonment of either description for a term that may extend to 10 years or with fine or with both.

Causing Death by Rash or Negligent Act—Section 304A

The provisions of this Section apply to cases where there is no intention to cause death and no knowledge that the act done in all probability would cause death. Criminal negligence is the gross and culpable neglect or failure to exercise the reasonable skill and proper care and caution to guard against injury either to the public generally or to the individual in particular, which the accused had the duty to adopt, under the circumstances of the case.

Culpable rashness is acting with the consciousness that the mischievous and illegal consequences may follow, but with the hope that they will not and often with the belief that the actor has taken sufficient precaution to prevent the happening. The imputability arises from acting despite the consciousness, whereas culpable negligence is acting without the effect that may follow. Of the two, rashness is a graver offence.

Dowry Death—Section 304B

(1) Where the death of a woman is caused by any burns or bodily injury or occurs otherwise than under normal circumstances within 7 years of 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.

(2) Whoever commits dowry death shall be punished with imprisonment for a term that shall not be less than 7 years but may extend to imprisonment for life.

According to a circular from the Home Ministry, a panel of two doctors is required to carry out the postmortem on the body of a married woman, dying of burns or other suspicious reasons within 7 years of her marriage or if her age was less than 30 years at the time of her death.

Abetment of Suicide—Section 306

Abetment of suicide is punishable under this Section and attempt to commit suicide under Section 309. The term 'abetment' has been defined under Section 107 IPC wherein it has been stressed that there must be instigation, cooperation or intentional assistance given to the would-be suicide. However, if he consents to be killed by another and in consequence is killed by that other, the offence is culpable homicide (homicide by consent). For example, supposing A and B conspire to produce B's miscarriage. For that purpose, A procures arsenic and gives it to B, which she takes and dies. Here, A is an abettor. However, if A had himself administered the poison to B and thus caused her death, he would have been guilty of culpable homicide (homicide by consent).

Two persons may agree to commit suicide (suicide pact/mutual suicide). If one dies, and the other survives by accident, the latter would be guilty of an abetment punishable under this Section as well as of an attempt under Section 309, though he could not be sentenced to cumulative sentences.

Attempt to Murder—Section 307

Whoever does any act with such intention or knowledge, and under such circumstances that if he by that act caused death, he

would be guilty of murder, shall be punished with imprisonment of either description for a term that may extend to 10 years, and shall also be liable to fine; and if hurt is caused to any person by such act, the offender shall be liable either to imprisonment for life, or to such punishment as is herein before mentioned.

Attempt to Commit Culpable Homicide—Section 308

Whoever does any act with such intention or knowledge and under such circumstances that if he by that act caused death, he would be guilty of culpable homicide not amounting to murder, shall be punished with imprisonment of either description for a term that may extend to 3 years, or with fine, or with both; if hurt is caused to any person by such act, he/she shall be punished with imprisonment of either description for a term that may extend to 7 years, or with fine, or with both.

Attempt to Commit Suicide—Section 309

Whoever attempts to commit suicide and does any act towards the commission of such offence shall be punished with simple imprisonment for a term that may extend to 1 year or with fine or with both. On 21st March, 1996, a five judge bench of the Supreme Court reversed its April 1994 verdict and held that the right to life as guaranteed under Article 21 of the Constitution did not include the right to die and therefore attempt to commit suicide and its abetment would continue to be an offence under Sections 306 and 309 of the IPC.

The threat of going on 'hunger strike' to achieve some justified/undesirable ends may well fall within the ambit of attempt to commit suicide. In March, 1999, police registered a case of 'an attempt to commit suicide by hunger strike' against two teachers who had been on fast unto death to protest against the dismissal of the teachers by the Government. One of the teachers was removed by the police and admitted to the hospital on the 9th day of her hunger strike. In the same light, it may be remembered that forcible feeding of prisoners on their refusal to take food on account of passive resistance is also lawful.

Hurt—Section 319

Whoever causes bodily pain, disease or infirmity to any other person is said to cause hurt. The Section comprises three elements:

- **Bodily pain:** To cause hurt, there need not be any direct physical contact. Where the direct result of an act is the causing of bodily pain it is hurt, whatever may be the means employed. Hurt is constituted by causing bodily pain and not mental pain. Giving alarming news may cause pain but not hurt. Dragging a person by the hair or fisting him, falls under this Section.
- **Disease:** A person communicating a particular disease to another would be guilty of causing hurt to another. However, there appear to be conflicting judicial decisions with respect to cases of communicating sexual diseases by one to another. In Roka vs. Emperor, the Bombay High Court held that a prostitute who had sexual connections with the complainant and thereby communicated syphilis is liable under Section 269 IPC for spreading of infection and not of causing hurt because the interval between the act and the disease was too remote to attract Sections 319 and 321 of IPC.
- **Infirmity:** Infirmity denotes an unsound or unhealthy state of body. The same remarks apply to the causing of an infirmity as to that of disease because it is something akin to, but not identical with disease. The term 'infirmity' is used to convey any inability of an organ to perform usual function. It may be temporary or permanent. A state of temporary mental impairment or hysteria or terror would constitute infirmity. For example, a boy of 16 years of age, being in love with a girl, gave her some sweetmeats. The girl and some of her family members ate them and all of them were seized with violent symptoms of poisoning, though none of them died. It was held that the boy was guilty of causing hurt.

Grievous Hurt—Section 320

The code on the basis of gravity of physical assault has classified hurt as 'simple' and 'grievous', so that the accused might be awarded punishment commensurate to his guilt. This Section designates eight kinds of hurts as grievous and provides enhanced punishment in such cases; these are following:

Emasculation This clause is confined to males only. It means unsexing of a man or depriving him of his virility. The clause was inserted to counteract the practice prevailing in women to squeeze men's testicles on the slightest provocation. Emasculation may be caused in a variety of ways. It may be caused by inflicting an injury to the scrotum as has the effect of rendering the person impotent or to the vertebral column leading to failure of erection of penis. The impotency caused must be permanent. An injury to the scrotum may lead not only to emasculation but to even death. Indeed, as remarked by Dr. Chevers, 'it is a form of assault, which is extremely liable to prove fatal'. In that case, the accused will be guilty of not only grievous hurt, but of culpable homicide.

Injuring Eyesight Such injury must have the effect of permanently depriving the injured of the use of one or both the eyes. The test of gravity is the permanency of the injury, which may be caused by hand as by gouging out one's eye with thumbnail or by poking it with a stick or the like. The injury is grievous, both because it deprives a man of an organ of sight, and also because it disfigures him for life.

Causing Deafness In this respect, the preceding injury is more serious than the 'permanent privation of hearing of either ear', which deprives a man of the use of his auricular organ but does not disfigure him. Such injury may be caused by a stunning blow on the head or the ear, injuring tympanum or other auditory nerves, thrusting a stick into the ear or pouring some substance into the ear leading to deafness.

In both these clauses, the loss or privation of sight or hearing may be partial but has to be permanent. However, 'permanent' does not mean that it should be incurable. For instance, loss of sight occurring due to corneal opacity resulting from injury to the cornea may be curable by corneoplasty but being permanent by itself constitutes a grievous hurt and chances of its treating by corneoplasty do not lower its gravity for this purpose.

Privation of Any Member or Joint This clause and the next refer to the old offence of 'mayhem', which Hawkins defined to be violently depriving a man of the use of such of his member as may render him less able in fighting. This offence was, in England at one time, visited with the penalty of death and by the ancient law, the penalty sanctioned for the same crime was, 'membrum pro membro', i.e. an eye for an eye and a tooth for a tooth. The punishment provided by the code depends upon the nature of the member or joint lost because the same penalty cannot be attached to the mutilation of an arm as to the loss of one's little finger. The term 'member' means nothing more than an organ or a limb. Therefore, it includes both the eyes, ears, nostrils, mouth, hands, feet, etc. A 'joint' means a place where two or more bones and muscles join. Their privation must involve such injury as makes them permanently stiff, so that they are unable to perform the normal function assigned to them in human physiology.

Permanent Impairing Powers of Any Member or Joint Deprivation of a limb or joint involves life-long crippling with its attendant defencelessness and misery. This clause sanctions the same policy of law in making it a grievous hurt to permanently impair the use of any limb or joint without causing its total destruction. Indeed, the mere retention of a limb, when it cannot be put to use for which it was created, is as great a hardship as if it had been lost by amputation or otherwise. Any permanent decrease in utility would constitute a grievous hurt. For example, formation of strictures due to burns, corrosives or any other injury resulting in permanent impairment of power/function of the concerned organ/tissue; damaging of some tendon(s) due to blunt or sharp force injury leading to permanent impairment of power/function of the concerned muscle/joint, etc. **A communication** (From Dr. Ashok Chanana, Associate Professor of Forensic Medicine at GMC, Amritsar) is quite illuminating: The victim, a male of about 25 years, allegedly received sharp force injuries during an assault. The injuries were (*i*) an incised wound, 5.0 cm × 1.5 cm, on the left parieto-occipital region of the head; (*ii*) an incised wound 6 cm × 1 cm, on the back of right forearm and (*iii*) an incised wound, 3.5 cm × 0.5 cm, on the front of right thumb, bone deep. The doctor on duty who had conducted the medicolegal examination, after going through X-ray reports, declared the injuries as 'simple' (casually going by the notion that if there was no bone injury, 'it is not grievous'. And, not contemplating that other relevant Clause(s) of 'grievous hurt' too need exclusion in a given scenario). However, the victim constantly complained of pain and restricted movements of the right thumb. He, consequently, requested the head of the institution to look into the matter. **An inquiry was ordered** and the injury number (*iii*) was declared as 'grievous' as it had resulted in cutting of the tendon of flexor pollicis longus. And, in the opinion of the orthopaedic surgeon (who had repaired the tendon), "it amounted to permanent impairment of the power/function of the thumb, and the victim would be unable to hold things in the thumb and fingers, and to write".

Permanent Disfiguration of the Head or Face The word 'disfigure' must be distinguished from the word 'disable'. 'Disfigure' means to inflict on a man some external injury that does not weaken him, but to 'disable' means to incapacitate him permanently. Such disfigurement may be caused by lopping off a man's ear or nose in which case there would be sufficient disfigurement without consequential disability, so as to constitute a grievous hurt under this Clause (Fig. 13.4). A nasty gash on the face leaving a permanent scar would be another instance of this type of injury. An injury may both disfigure and disable a person, e.g. gouging out an eye. In that case, the offence will be the same, namely, a grievous hurt, though the injury would then fall both under this Clause and Clause (2).

Fracture or Dislocation of a Bone or Tooth The fracture or dislocation of a bone or tooth is another type of grievous hurt that may or may not be attended with permanent disability, e.g., a bone though fractured or dislocated may rejoin or be set and leave little or no trace behind of its fracture or dislocation. But the injury is esteemed grievous on account of the intense pain and disability it causes to the sufferer. The same disability may be attached to the dislocation or fracture of a tooth.

A fracture is not defined in the Indian Penal Code, but it is beyond the plea of controversy. If there is a break by cutting or splintering of the bone or there is a rupture or fissure in it, it would amount to a fracture within the meaning of Cl (7) of Section 320 of the IPC. Partial cut of the skull vault would amount to a fracture within the meaning of this Clause.

Usual X-raying of the part not revealing fracture—need for repeat X-ray in a given scenario: A middle aged man was allegedly assaulted on 02.03.07 at about 7.00 p.m. He showed graze measuring 12 cm × 2 cm on the outer aspect of right scapular region with surrounded reddish bruise. On clinical examination, no body deformity, no abnormal mobility, etc. were noted. Usual X-ray revealed absence fracture. All injuries, including this, were declared as 'simple'. However, the victim continued complaining of pain and restricted movements

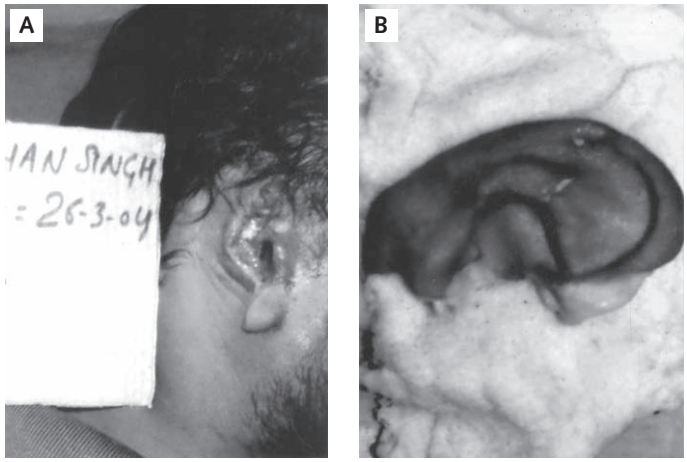


Fig. 13.4 Photographs **(A)** and **(B)** showing chopped off left pinna and the separately lying severed part, respectively. The victim, aged about 30 years, landed into scuffle with his friend while both were intoxicated and suffered this injury. (Contributed by Dr. Parmod Goyal, Associate Professor of Forensic Medicine, AIMSR, Bathinda)



Fig. 13.5 X-ray of right shoulder showing incomplete fracture of acromian process of scapula.

of the right shoulder; therefore, the victim was subjected to digital X-ray, which showed fracture of acromian process of right scapula (Fig. 13.5). And then, a supplementary report/opinion was extended, declaring the injury as 'grievous'. (Contributed by Dr. Parmod Goyal, Associate Professor of Forensic Medicine, AIMSR, Bathinda).

Hurt Endangering Life or Causing Severe Pain or Refraining from Ordinary Pursuits There is nothing corresponding to this Clause in the English law, and it is admitted by the framers that they borrowed this from the French Penal Code. The period of '20 days' fixed by them for making the injury 'grievous' was, of course, arbitrary, but any period fixed would have been the same. The Clause encompasses provisions for cases not only where violence has been used but also for cases where hurt has been caused without any assault as by administration of drugs, setting of traps, digging of pitfalls, etc.

The Clause refers to three classes of injuries, which it designates grievous, namely:

- Those that endanger life
- Those that cause severe bodily pain for 20 days
- Those that disable the sufferer from following his ordinary pursuits for 20 days.

Endangering life: It refers to injuries that endanger life. The question is one of degree, and it must be ascertained in each case to what extent the hurt bears proximate relation to the risk of life. Ordinarily, it is true that injuries inflicted on a vital part of the body, such as head, chest, etc. tend to endanger life. The line between culpable homicide not amounting to murder and grievous hurt is a very thin and subtle one. In one case, the injuries must be such as are likely to cause death and in the other, the injuries must be such as endangering life.

A dangerous injury is one that poses imminent danger to life, either by involvement of important organ(s)/structure(s) or extensive area of the body. The word 'imminent' implies a danger that is impending. That 'dangerous injury' is one which may prove fatal in the absence of surgical aid, may not be adhered to literally. Contusion/laceration of brain stem may prove fatal, notwithstanding surgical aid. For declaring the injury 'dangerous to life', the doctor may take into consideration the nature and extent of injury, the kind of weapon used, the part of the body struck and the condition of the patient/victim including nature and extent of medication during the hospital stay (if any).

Causing severe bodily pain: An injury that is dangerous to life would necessarily cause severe bodily pain, unless death supervenes instantaneously. But an injury may cause such pain, and yet be not dangerous to life.

Disabling the sufferer from following ordinary pursuits: The test for grievousness is the sufferer's inability to attend to his ordinary duties for a period of 20 days. This clause necessarily involves many elements of uncertainty. But here again, to prevent its misapplication in cases of feigned

inability, regard must be paid to the nature and severity of the injury, as well as the probability of the disability it was likely to cause. The mere fact that the sufferer did not attend to his duty for the statutory period or that he remained in a hospital for that period is no indication of his inability. As was observed in a Mumbai case: 'An injured man may be quite capable of following his ordinary pursuits long before 20 days are over and yet for the sake of permanent recovery or greater ease or comfort be willing to remain as a convalescent in a hospital, especially if he is fed at the public expense'.

The law requires that the injured person should during the space of 20 days be in severe bodily pain or unable to follow his ordinary pursuits. It is not correct to say that these things can be established only on medical evidence and not by other evidence. The medical evidence may be more reliable but is not legally necessary.

Assault—Section 351

Whoever makes any gesture, or preparation intending or knowing it to be likely that such gesture or preparation will cause any person present to apprehend that he who makes that gesture or preparation is about to use criminal force to that person is said to commit an assault.

Explanation: Mere words do not amount to an assault. But the words, which a person uses, may give to his gestures or preparations such a meaning as may make those gestures and preparations amount to an assault.

Assault and Battery Distinguished An assault is a threat by one to the unlawful force against another. But a mere threat not in the slightest degree executed, or acts or gestures done under such circumstances or at such a distance that the threat cannot possibly be carried out, does not amount to an assault.

A battery includes touching a person and laying hold on his clothes in an angry, revengeful, rude, insolent, or hostile manner. Even an act such as striking a horse on which a man is riding so that he is thrown off will amount to battery. A threat to throw boiling water on a man amounts to an assault, and when it touches his body, it amounts to battery.

Assault or Criminal Force to a Woman with Intent to Outrage Her Modesty—Section 354

Whoever assaults or uses criminal force against any woman intending to outrage or knowing it to be likely that he will thereby outrage her modesty shall be punished with imprisonment of either description for a term that may extend to 2 years or with fine or with both.

Woman's Modesty What constitutes an outrage to a female's modesty is nowhere defined. This will depend upon the facts and circumstances of each case and according to the moral, social and legal ethos and traditions of the country and the race to which the woman belongs. Thus, assault can be committed on any woman irrespective of her age. Decency means propriety of behaviour, avoidance of obscene language and gestures and of undue exposure.

The word 'outrage' means 'gross infringement of decency/morality, something that violates the feelings or the proprieties, dangerous display of passion, etc. In State of Punjab vs. Major Singh, the question arose whether a female child of seven and a half months could be said to be possessed of 'modesty' that could be outraged. In answering this question, the Supreme Court observed: 'When any act done to or in the presence of a woman is clearly suggestive of sex according to the "common notions of mankind", that must fall within the mischief of Section 354 IPC'. Needless to say, the 'common notions of mankind' referred to by the learned judge have to be gauged by contemporary societal standards.

Adultery—Section 497

Whoever has sexual intercourse with a person who is and whom he knows or has reason to believe to be the wife of another man, without the consent or connivance of that man, such sexual intercourse, not amounting to the offence of rape, is the offence of adultery, and the guilty shall be punished with imprisonment of either description for a term that may extend to 5 years, or with fine, or with both. In such a case, the wife shall not be punishable as an abettor.

Enticing or Taking Away or Detaining with Criminal Intent a Married Woman—Section 498

Whoever takes or entices away any woman who is and whom he knows or has reason to believe to be the wife of any other man, from that man, or from any person having the care of her on behalf of that man, with intent that she may have illicit intercourse with any person, or conceals or detains with that intent any such woman, shall be punished with imprisonment of either description for a term that may extend to 2 years, or with fine, or with both.

This Section deals with the offence of criminal elopement. The provisions of this Section like those of Section 497 are intended to protect the rights of a husband over his wife. The Section requires following ingredients:

- Taking or enticing away or concealing or detaining the wife of another man from that man or from any other person having care of her on behalf of that man.
- Knowledge or reason to believe that she is the wife of another man.
- Such taking, concealing or detaining must be with the intent that she may have illicit intercourse with any person. Use of physical force is not an ingredient of the offence.

See Chapter "Thermal Deaths" for Section 498A.

Criminal Intimidation—Section 503

Whoever threatens another with any injury to his person, reputation or property, or to the person or reputation of anyone in

whom that person is interested, with intent to cause alarm to that person, or to cause that person to do any act that he is not legally bound to do, or to omit to do any act that that person is legally entitled to do, as the means of avoiding the execution of such threat, commits criminal intimidation.

Explanation: A threat to injure the reputation of any deceased person in whom the person threatened is interested is within this Section.

Sections 503–510 of the code deal with criminal intimidation, insult and annoyance. This Section has the following essentials:

- Threatening a person with any injury to his person, reputation or property or to the person or reputation of anyone in whom the person is interested.
- Threat must be with intent to cause alarm to that person or to cause that person to do any act that he is not legally bound to do as the means of avoiding the execution of threat or to cause that person to omit to do any act that that person is legally entitled to do as the means of avoiding the execution of such threat.

Word, Gesture or Act Intended to Insult the Modesty of a Woman—Section 509

Whoever intending to insult the modesty of a woman utters any word, makes any sound or gesture, or exhibits any object, intending that such word or sound shall be heard, or that such gesture or object shall be seen by such woman, or intrudes upon the privacy of such woman, shall be punished with simple imprisonment for a term that may extend to 1 year, or with fine or with both.

While Section 375 IPC deals with the forcible ravishment of woman (rape), Section 354 and 509 IPC deal with the lesser acts of indecency such as solicitation for sexual connection or to enjoy pleasurable feelings, etc. Essential component of the offence under Section 354 IPC is the 'intention to outrage modesty of a woman', whereas under Section 509 IPC, it is the 'intention to insult the modesty of woman'. The term 'outrage' is much stronger than the term 'insult', and that is why the resulting punishment in the former case is extendable up to 2 years' imprisonment, whereas in the latter case, up to 1 year only. (The term **outrage** implies gross infringement of morality or decency, gross violation of other's rights, sentiments, emotions, etc. The term **insult** implies any act or speech meant to hurt the feelings or self-respect of another or treat a person with offensive disrespect/insolence/contempt, etc.). However, both these Sections have a common feature, i.e. bailable nature of the offence. Reports suggest that such weakness in the law has been exploited frequently. This came to be seen when a few miscreants molested two NRI women near a Mumbai Hotel on the eve of the New Year 2008, but were granted bail soon after their arrest (the term **modest** implies: to trouble, annoy, disturb or vex, etc. In its ordinary connotation, it applies to any conduct that can be regarded as such a degree of harassment as constituting a criminal offence).

Misconduct in Public by a Drunken Person—Section 510

Whoever in a state of intoxication appears in any public place or in any other place where it amounts to a trespass and there conducts himself in such a manner as to cause annoyance to any person shall be punished with simple imprisonment for a term that may extend to 24 hours or with fine or with both.

Mere intoxication is not an offence. It is only when the person appears in a state of intoxication in a public place or goes to a place where he has no right to go and causes annoyance to the people then he becomes liable under this Section.

Attempts to Commit Offences—Section 511

Whoever attempts to commit an offence punishable by this code with imprisonment or to cause such an offence to be committed and in such attempt does any act towards the commission of the offence, shall, where no express provision is made by this code for the punishment of such attempt, be punished with imprisonment of any description provided for the offence for a term that may extend to one-half of the imprisonment for life or, as the case may be, one-half of the longest term of imprisonment provided for that offence, or with such fine as is provided for the offence, or with both.

In every crime, there are four successive stages in its commission, viz.,

- Intention to commit it
- Preparation
- Attempt to commit it
- The actual commission of the offence

Intention is the direction/design of conduct towards an object or aim, based upon some motive. The law does not take notice of intention without an external act showing some progress towards maturing and affecting it.

Preparation consists in devising or arranging means or measures necessary for the commission of an offence.

Attempt is made punishable because though it fails, it creates alarm/shock, which itself is an injury and the moral guilt of the offender is the same as if he had succeeded. An attempt is direct movement or action towards the commission of an offence after the preparations are made. The action fails in its object due to the circumstances beyond the control of the offender or independent of his volition.

Differentiating Kidnapping and Abduction

Table 13.3 describes distinctions between kidnapping and abduction.

Table 13.3 Kidnapping and Abduction Distinguished

Kidnapping	Abduction
Kidnapping from lawful guardianship defined in Section 361 is committed (*i*) in respect of minors, if male under 16 and if female under 18 years of age and (*ii*) in respect of a person of unsound mind (of any age).	Abduction may take place against any person of any age. Likewise, kidnapping from India as described in Section 360 can take place in respect of any person irrespective of his or her age.
Kidnapping is the removal of a person from lawful guardianship.	Abduction has reference only to the person abducted.
Kidnapping is simply taking away of a minor or a person of unsound mind. The means used are not relevant.	In abduction, force, compulsion, or deceitful means are used.
Consent of the person kidnapped is immaterial.	Free and voluntary consent of the person abducted condones abduction.
Intention of the kidnapper is immaterial for the offence.	Intention of the abductor is an important factor in determining guilt of the accused.
Kidnapping is a substantive offence.	Abduction is not a substantive offence and is not punishable in itself. It is an offence only when done with some other intent as given in Sections 364–369.
Kidnapping is not a continuing offence. It is complete as soon as a minor or a person of unsound mind is removed from lawful guardianship.	Abduction is a continuing offence and continues so long as the abducted person is removed from one place to another.
In kidnapping, there must be taking or enticing from a lawful guardian.	In abduction, the question of taking or enticing does not arise.

Classification of Injuries

Wounds may be classified in a variety of ways, but it should not be forgotten that the injuries are often mixed. Thus, an abrasion may be found in association with contusions and lacerations and any combination of these three is common.

- **Depending upon Causative Factor**
  - ***Mechanical or Physical Injuries***
    - — Those caused by *blunt force*
      - – Abrasions
      - – Contusions (bruise)
      - – Lacerations
    - — Those caused by *sharp force*
      - – Incisions
      - – Punctures (punctures may be incised puncture or lacerated puncture)
    - — Caused by *firearms*
      - – By rifled firearms
      - – By smooth bored firearms
      - – By country made weapons
  - ***Thermal Injuries***
    - — Due to *heat*
      - – Generalised effects of heat, i.e. heat hyperpyrexia (heat stroke), heat exhaustion (heat collapse) and heat cramps (miner's cramps)
      - – Localised effects of heat, i.e. burns (due to application of dry heat) and scalds (due to application of moist heat)
    - — Due to *cold*
      - – Generalised effects of cold, i.e. hypothermia
      - – Localised effects of cold, i.e. frostbite (due to dry cold) and trench foot (due to wet cold)
  - ***Caused by Chemical Agents***
    - — Corrosions (due to strong acids or alkalies)
    - — Irritation (due to weak acids, alkalies, vegetables or animal extracts, etc.)
  - ***Miscellaneous***
    - — Lightning
    - — Electricity
    - — Radiation (X-rays, ultraviolet rays, radioactive substances, etc.)
    - — Blast injuries
- **Depending upon Gravity**
  - Simple
  - Grievous
  - Dangerous
- **Depending upon Time of Infliction**
  - Antemortem
  - Postmortem
  - Perimortem
- **Depending upon the Manner of Infliction/Sustaining**
  - Suicidal
  - Accidental
  - Homicidal
  - Defence wounds
  - Self-inflicted/self-suffered
  - Fabricated/fictitious injuries

14 CHAPTER

Injuries by Blunt Force

After going through this chapter, the reader will be able to describe: Forensic aspects of the anatomy of skin | Abrasion and its types | Patterned abrasions | Fate of an abrasion | Differentiation between antemortem and postmortem abrasions | Medicolegal aspects of abrasions | Bruise (contusion) and factors influencing its production | Migratory/ectopic bruising | Fate of a bruise | Patterned bruising | Differentiation between antemortem and postmortem bruising | Medicolegal aspects of bruising | Lacerations and its types | Incise-looking wounds | Features of lacerations | Differentiation between antemortem and postmortem lacerations | Medicolegal aspects of laceration

Skin Anatomy and Its Forensic Aspects

As most of the wounds involve the body surface, a peep into the structure of the skin and subcutaneous tissue will be highly appropriate at the very onset. **Forensic aspects of the anatomy of skin are described as under:**

EPIDERMIS

It is the superficial protective layer of the skin and is composed of stratified squamous epithelium that varies in thickness from 0.007 to 0.12 mm. It is the thickest over the soles and palms, while it is much thinner over the protected areas like scrotum, eyelids, etc. This bears a forensic relevance showing the varying amount of force needed to penetrate the skin at different parts of the body.

The names and characteristics of epidermal layers are as under (Fig. 14.1):

- **Stratum corneum:** It consists of 25–30 layers of flattened scale-like cells, which are continuously shed as flake-like residues of cells. This surface layer is cornified and is the real protective layer of the skin. Cornification is brought about by keratinisation and the hardening flattening process that takes place as the cells die and are pushed to the surface.
- **Stratum lucidum:** It exists only in the lips and thickened layers of soles and palms.
- **Stratum granulosum:** It consists of only three or four layers of flattened cells. The cells within the layers appear granular due to the process of keratinisation.
- **Stratum spinosum:** The spiny appearance of this layer is due to changed shape of the keratinocytes.
- **Stratum basale:** It is composed of a single layer of cells in contact with the dermis. Four types of cells compose the stratum basale, i.e. keratinocytes, melanocytes, tactile cells and nonpigmented granular dendrocytes (Langerhans cells). With the exception of tactile cells, these cells are constantly dividing mitotically and moving outwards to renew the epidermis. It usually takes between 6 and 8 weeks for the cells to move from the stratum basale to the surface of the skin.

All except the stratum basale and the stratum spinosum are composed of dead cells. That is why these two layers are sometimes collectively called as stratum germinativum.

Dermis

It is deeper and thicker than epidermis. Blood vessels within the dermis nourish the living portion of the epidermis, and numerous collagenous, elastic and reticular fibres give support to the skin. The fibres within the dermis radiate in different definite directions producing lines of tension on the surface of the skin, called the **cleavage lines of Langer**. Gaping of the stab wounds or incisions will depend upon their location and orientation with respect to these cleavage lines.

The dermis is highly vascular and glandular and contains many nerve endings and hair follicles. **Dermis has two layers:** (*i*) Upper layer called the stratum papillarosum (papillary layer),

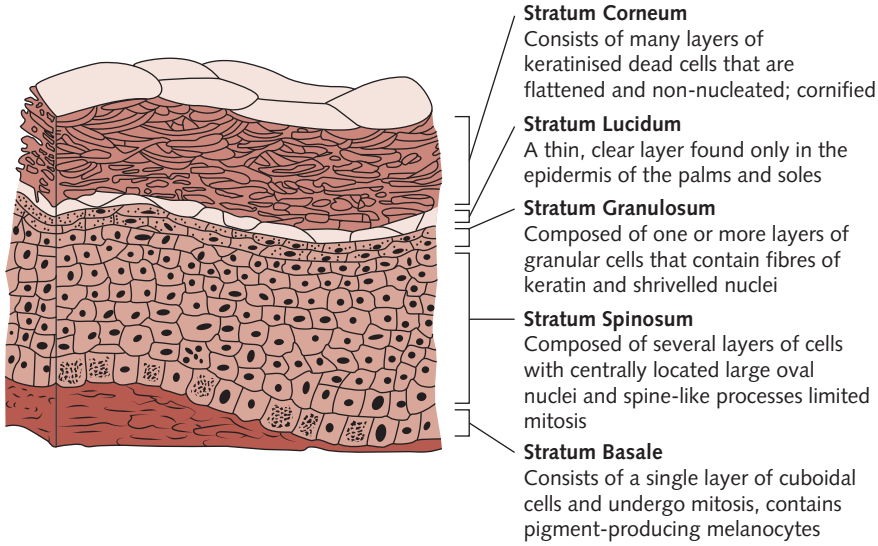


Fig. 14.1 Layers of the epidermis.

which is in contact with the epidermis. Numerous projections (papillae) extend from this layer into the epidermis. Papillae form the base for the friction ridges on the fingers and toes. (*ii*) The lower layer is called stratum reticularosum. (Tattooing colours the skin permanently, because the pigmented dyes are injected below the mitotic basale layer into the dermis.)

The skin manifestations of blunt trauma differ depending upon the force and nature of the impact. Three basic lesions are recognised:

- Abrasions
- Contusions
- Lacerations

Abrasions

Abrasions are the **injuries involving superficial layer of the skin (the epidermis or mucous membrane) due to impact against some hard, blunt and rough object/weapon**. Thus, the pure abrasions do not ordinarily bleed, because the vascular supply of the skin comes through the vascular network running in the dermis. Practically, because of the corrugated nature of the dermal papillae as detailed in the beginning, quite often dermis is also involved and consequently abrasions often exhibit bleeding. At places, abrasions may penetrate the full thickness of skin. Therefore, large areas of abrasions [such as 'brush abrasions' (vide infra)] may bleed, though the bleeding is rarely serious as only small blood vessels are involved. For an abrasion to appear, some movement along with pressure is essential between the object/instrument/weapon and the skin. This movement and pressure may be exerted either by the body itself or by the abrading instrument.

TYPES OF ABRASIONS

Depending upon the mechanism of the pressure and nature and movement of the weapon/agent involved against the skin surface, abrasions may be classified as follows.

Scratches or Linear Abrasions

These are **produced by horizontal or tangential friction by the pointed end of some object like thorn, nail, needle or tip of any weapon**. Therefore, the contention that abrasions cannot be produced by a sharp weapon does not stand on sound footings as the scratches or linear abrasions can conveniently be produced by pointed terminal portion of even a sharp weapon. All that matters is the manner in which the weapon is being used, the force applied, the area of the body involved and the circumstances governing the positions of the victim and the assailant (Fig. 14.2A).

Grazes (Sliding/Tangential/Brush Abrasions)

These are **caused due to horizontal or tangential friction between the broader area of the skin and the object/weapon or hard rough surface of the ground**. The epidermis will get heaped up at the opposite end and the pattern of heaping will indicate the direction of movement of the object/weapon against the skin (Fig. 14.2B).

These sliding abrasions are most commonly encountered in traffic accidents where a pedestrian has been knocked down and is dragged over the ground for a varying distance. They may frequently be associated with underlying bruising. These types of abrasions caused by violent lateral (tangential) rubbing against a rough surface are called **brush burns**. The sliding of the body against the rough surface scrapes linear furrows

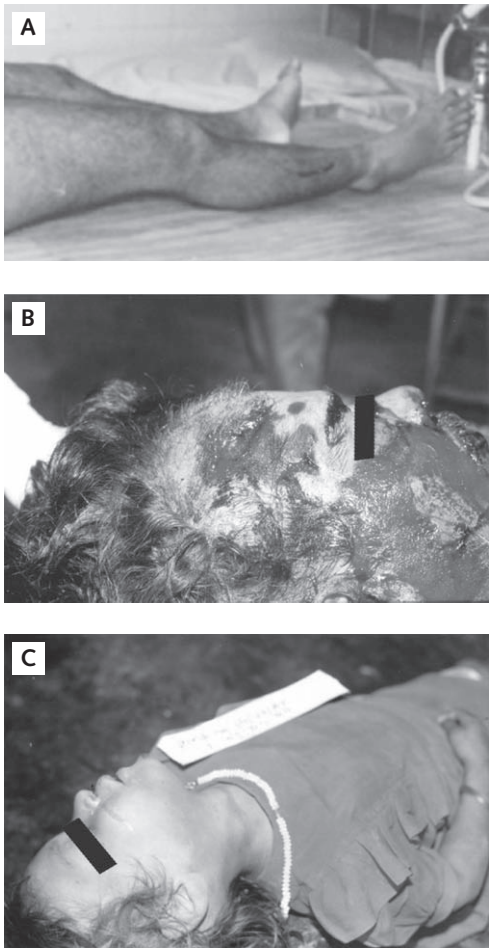


Fig. 14.2 Photograph showing various kinds of abrasion: **(A)** linear abrasion on the leg in an accident victim; **(B)** graze on the side of the face in an accident victim; **(C)** imprint abrasion produced by necklace upon the neck in a victim of strangulation.

across the skin. Strands and tags of epidermis may be peeled along these furrows and get heaped up at the other end where the contact ceased. Similarly, where a victim is imparted a glancing blow with a rough object, similar epidermal strands may indicate the direction of the blow.

Pressure Abrasions (Crushing Abrasions/ Imprint Abrasions)

When the **impact is vertical to the skin surface, the epidermis gets crushed and pressure type of abrasions result and the imprint of the impacting object may be produced**. These may be seen in manual strangulation (abrasions produced by fingernails) and in hanging, where the weave of the ligature material may be reproduced. If the impact is forcible then the dermis may also be injured and show underneath bruising.

Patterned Abrasions

Patterned abrasions occur **when the force is applied at or around right angle to the surface of the skin**, as already mentioned. If a weapon with patterned surface strikes the body or body falls upon a patterned rough hard surface, the abrasions will usually follow the pattern of the object. The classical example of this is seen in traffic accidents when a tyre of a motor car passes over the skin leaving the pattern where the skin has been squeezed into the grooves of the rubber tread. Abrasions from objects with a recurring pattern such as chain of a cycle or a serrated knife or a necklace, etc. may be the other examples (Fig. 14.2C). In such cases, skin may also get compressed in the depressions of the pattern, often leading to an associated intradermal bruising due to capillary damage. Patterned abrasions are sometimes produced by the recoil of a firearm when discharged at a contact range.

Atypical Abrasions

Nail marks and teeth bites may conveniently be included in this category of abrasions though they may produce lacerations too, depending upon the force applied. Nail marks are especially important in cases of child abuse, sexual offences and manual strangulation. They may appear as linear scratches or short straight or curved marks depending upon the circumstances. The pattern may often be fragmentary rather than typical crescentic. Shapiro, Gluckman and Gordon have shown that because the skin is put under tension when it is indented by the nails, it may get distorted so that on releasing tension the elasticity of the skin brings it to its original position, carrying the nail mark also. The curve, which is usually expected to be produced, may then get reversed to produce either a straight line or a convexity. The overall configuration of the nails and the circumstances under which the marks are produced materially influence their pattern; therefore, it is always advisable to use utmost caution in their interpretation.

AGE (FATE) OF AN ABRASION

Abrasions usually heal rapidly without any scar formation, unless they are deep enough to involve the dermis, which prevents regrowth of hair follicles and sebaceous glands. A rough idea about their age may be gathered from the following changes:

Fresh:	The area will appear reddish due to oozing out of serum and little blood. The dermis will be congested and painful.
12–24 hours:	The exudation dries up to form a reddish scab, comprising dried blood, lymph and injured epithelial cells.
2–3 days:	The scab is reddish-brown, less tender and adhering over the abraded area.
4–5 days:	The scab is dark brown in appearance.
5–7 days:	Scab is brownish black in appearance and starts falling off from the margins.
7–10 days:	The scab shrinks and falls off, leaving some depigmented area underneath. The depigmented area gets gradually pigmented in due course of time.

ANTEMORTEM/POSTMORTEM ABRASIONS

In the living, detection of abrasions does not pose much problem as the victim is aware of their situation since these are painful and moist. **In the dead,** as the circulation of blood has ceased, there is no exudation of serum and therefore, the surface gets dried up and becomes hard acquiring the consistency of parchment and also appears brownish. The dried abrasion often appears to be a much more extensive injury than it was at the time of death.

Postmortem abrasions may be caused during transportation of the dead body. These abrasions may also leave hard yellow areas that can be differentiated if examined with care. Abrasions sustained at or about the time of death cannot be distinguished with certainty. If, however, any associated bruising or vital reaction can be shown either by naked eye or by microscopy, then differentiation can be established. The presence of fair amount of bleeding, of course, favours antemortem production.

DIFFERENTIAL DIAGNOSIS

Sometimes, abrasions may have to be distinguished from the following:

Erosion of the Skin Produced by Insects, Ants, etc.

Ants produce erosions with irregular margins of the superficial layers of the skin and do not show any vital reaction. These are most commonly present at mucocutaneous junctions and at the moist folds of skin.

Excoriation of the Skin by Excreta

This is more likely to be seen in infants, and its distribution is self-explanatory. After death, the napkin area may become dry, depressed and parchment-like, especially when the plastic rompers or knickers are worn over a wet napkin.

MEDICOLEGAL CONSIDERATIONS

Abrasions, though trivial injuries involving only the superficial layers of the skin, should not be considered too insignificant to be ignored in the medicolegal investigations. They may be the only external visible signs of a severe or even fatal internal injury.

- Their importance lies in the fact that they are produced at the point of impact of the blunt force, e.g. abraded knuckles or knees may show the involvement of these areas.
- They may exhibit a pattern thereby providing information regarding the nature of force. There seems to be no need to list all the possible patterns that can be distinguished on the body and a few having a particular medicolegal significance have already been described under 'patterned abrasions'.
- Their sites and distributions over the body may yield some clue towards the nature of crime, for instance, presence of crescentic abrasions or scratches over the neck, face or inguinal regions of a female may well have been produced by the fingernails of assailant during sexual assault.
- Direction of application of force may be inferred from the collected epithelial tags at distal end of the abraded area. This is particularly so in a drag or brush marks ruffling of the skin, indicating the direction of the force applied to the skin.
- Presence of some material like mud, grit, coal dust, cement, sand, lime dust, pebbles or any vegetation, etc. in and around the abraded area will suggest the nature of the surface or agent responsible for its causation.
- Various stages of healing of an abrasion are helpful in determining the approximate period of infliction of the injury.
- It is a well-known postmortem phenomenon that abrasions and bruises become more prominent after sometime following death. The appearance of body after a lapse of 24 hours or so following death may be quite different from the appearance immediately succeeding death. In a body recovered from water, abrasions may be appreciated sometimes after its recovery when they manifest more prominently on drying.
- Lastly, fabricated abrasions may be produced over accessible parts of the body to bring a false charge of assault or for some other extraneous motive. The circumstances are self-explanatory in such cases, and careful examination will solve the problem.

Contusions (Bruises)

Contusions are **characterised by infiltration of extravasated blood into the subcutaneous and/or subepithelial tissues resulting from rupture of small blood vessels due to application of blunt force**. Therefore, not only the skin but the internal organs like heart, liver, spleen, kidney or muscle could be contused. In all such cases, the integrity of the skin or of the architecture of the organ is not disturbed, though under rare circumstances the overlying epidermis may be abraded, where they may be termed as **'abraded contusion'**.

The term **'ecchymosis'** too implies extravasation of blood into the cellular tissues but is no longer in use and is more often caused by factors other than direct mechanical trauma and usually involves the serous membranes, though can also be seen on the skin.

The lay term for superficial contusion is 'bruise', which is observed through the overlying intact skin as a bluish-purple discolouration and, in some instances, swelling of the involved area. The two words are often interchanged but the term **'bruise'** is more popular and may be preferred in the usual medicolegal

examinations. The black-eye following a fistfight, the scalp haemorrhage from a fall and the bluish-purple appearance after an upper arm is squeezed too tightly are a few examples.

FACTORS INFLUENCING THE PROMINENCE OF A BRUISE

Many factors influence the extent, size, time of appearance and the prominence of a bruise; therefore, opinions regarding their time of appearance and the amount of force required to produce them must be furnished in the generalised terms rather than in precise terms.

Amount of Force

As a general rule, the greater the force of violence, the more extensive will be the bruise. This generalisation is subject to some modifications, i.e.

- If the tissue involved is loose and lax such as face, scrotum, genitalia, eyelids, etc., a moderate blow may result in a relatively large bruise as there is sufficient space for the blood to accumulate.
- If the tissues are strongly supported containing firm fibrous tissues and covered by thick dermis such as back, scalp, palms and soles, etc., a blow of moderate violence may produce a comparatively small bruise where dense fibrous tissue and restrictive fascial planes prevent easy accumulation of blood.

Peculiarities of the Victim

- Children bruise more rapidly than adults because of softer tissues and delicate skin.
- Old persons too bruise easily owing to loss of flesh and accompanying cardiovascular changes.
- Chronic alcoholics bruise easily because of cutaneous vasodilatation.
- Boxers and athletes show comparatively less bruising due to good muscle tone, which may prevent easy rupture of blood vessels.
- Women bruise more easily than men because of delicacy of the skin and greater amount of subcutaneous fat.
- Strong healthy persons with active habits may stand considerable blow without appreciable bruising.
- Obese flabby persons bruise easily even with relatively lesser amount of violence.
- Naturally, bruises are more easily appreciated in fair skinned people than in heavily pigmented individuals.
- Presence of some disease like scurvy, vitamin K and prothrombin deficiency, haemophilia, leucaemia, atherosclerosis, etc. may produce exaggerated bruising. Same may be the case if the person is suffering from toxic manifestations of certain drugs.
- Diet of the individual may also have a bearing on the development of bruising on account of having its effect on bleeding and clotting time and its ability to affect the consistency of body fats in supporting connective tissues of the body.

Vascularity of the Area

The apparent prominence of a bruise beneath the skin obviously varies with the amount of blood in the extravasation. The size and density of the vascular network varies from area to area and that is why bruising over the areas like face, genitalia, scrotum, etc., having rich vascularity, will be more as compared to other areas.

Resiliency of the Area

Resilient areas such as abdominal wall, buttocks, etc. bruise less with a given impact than the region having a bone immediately underneath and with least amount of subcutaneous tissue, like head, shins and areas against iliac crests. Abdominal wall is notorious in this respect and seldom gets bruised even with good amount of violence. Resilient and yielding anterior abdominal wall, giving way to the force, may allow the full brunt of the force to be suffered by the more resistant internal organs, which may get ruptured, without showing any evidence of injury. This is especially seen in vehicular accidents, where grave internal injuries to the organs may be present without any external evidence, particularly when the victim is stuffed with multiple layers of clothing.

DEEP/DELAYED/MIGRATORY BRUISES

The depth at which the bruise is placed affects its appearance on the surface. Most bruises are in the subcutaneous tissues above the deep fascia and therefore quite easy to appreciate but others may lie deeper. These deep bruises may not only take a long time to become visible **(delayed bruising)** but may not appear against the actual point of impact. Blood escaping from the damaged vessels tracks along the fascial or muscular planes and following the path of least resistance may make its appearance hours or even days after the impact, at a place where the tissue layers become superficial. This may be termed as **migratory or ectopic bruising**. Another factor operating in this context may be haemolysis, when the freed haemoglobin is able to stain the tissues, which therefore become more noticeable. This latter factor may be responsible for the well-known postmortem phenomenon of bruises becoming more prominent after death and thereby creating differences in opinions, as referred under 'Abrasions' too. Examples of ectopic bruising may be **black eye (spectacle haematoma)** presenting as haemorrhages into the soft tissues of the eyes and in the eyelids following a blow on the forehead and blood gravitating downwards over the supraorbital bridge; in fracture of pelvis, bruise may appear in the thigh; a kick on the calf muscles of the leg may appear as a bruise around the ankle and so on.

PATTERNED BRUISING

Bruises do not display such a detailed pattern as encountered in abrasions, due to padding action of the skin that causes the force to be distributed diffusely and thus preventing the production of a distinct outline. However, there may still be circumstances where some recognisable patterns may be produced. Instances may be legion, but a few of common occurrence may be cited as under:

- One particular type of bruise often seen is the so-called **'tramline' or 'railway line' bruise**. Here, two parallel linear haemorrhages, frequently resulting from rod, stick or the like object, are produced with an intervening almost unbruised area. The mechanism of its production seems to be that when the rod or stick or the like object forcibly dents the skin and underneath tissues in the area where it makes its impact, the tissue on each side of this impact gets stretched. This stretching of the sides results in rupture of vessels leading to formation of line of bruising on either side. In addition to this, blood from the bed of the dent also gets displaced to the sides with the pressure exerted by the impact by such objects. Thus, as the impact is removed and the skin springs back to its usual position, the two sides of the compressed area manifest as bruised lines.
- In strangulation, the pattern of a necklace (Fig. 14.2C) or string of beads may be impressed upon the neck or if some coarse weave of fabric has been used, it may leave a patterned bruise.
- Whipping with cords, plated leather thongs, etc. may also leave peculiar traces, which may encircle a part of limb or the trunk of the victim.
- As detailed under 'Abrasions', a cycle chain, a strap or a tyre may produce a patterned bruise in association with the abrasions.
- **Intradermal bruises deserve special mention.** Ordinarily, the bruise is situated in the subcutaneous tissue, in the fatty layer. However, when it is situated in the immediate subepidermal layer, the pattern of the impacting object may be more distinct. This is obviously partly due to its superficial situation and partly due to translucency of the comparatively thin layer of skin overlying it. Typical examples may be production of tyre marks, impacts from whips or soft canes and rubber soles of shoes.

Case—'Tramline' Bruising by Beating with a Cane

The victim, a married woman of about 28 years, had been running fever for few days prior to her admission to hospital. Before admission, as usually prevalent amongst the village folk, she was taken to some *tantrik* to effect withdrawal of the 'evil spirit' responsible for causing fever as her husband had lost all his patience and hopes due to her chronic illness. He developed a belief that it was due to some 'evil spirit' hiding within his wife and thereby thought of treatment (withdrawal of spirit) from some tantrik. The tantrik, as we usually observe and see in the cinema and otherwise, commenced his operation by beating her with some specific cane and also slapping her on the face. This was based on the theory that he was beating the spirit within the lady rather than the lady herself. (See Figs. 14.3A and B, showing 'tramline/railway line' type of multiple contusions on the back, distributed in an irregular fashion, some superimposing others; typically consistent with production by some stick or a like instrument. Left side of the face exhibits characteristic linear intradermal haemorrhages with some haemorrhages lying scattered within these linear haemorrhages, as shown in Fig. 14.3C).

AGE (FATE) OF A BRUISE

The time taken for a bruise to disappear will depend upon its site, size and constitution of the victim including personal idiosyncrasies towards the process of healing. The more common

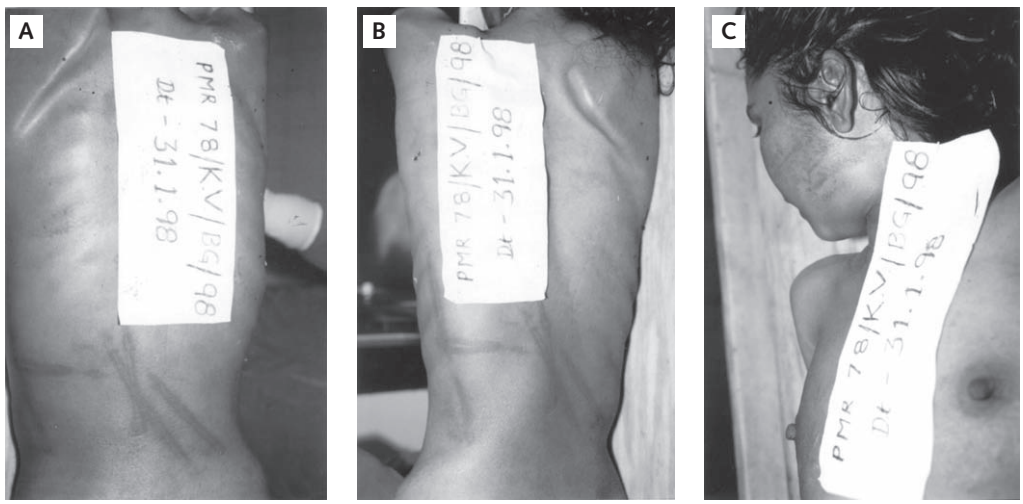


Fig. 14.3 Images **(A)** and **(B)** show tramline bruising with a cane; image **(C)** shows intradermal bruising upon the face.

superficial bruise, however, resolves and disappears after a succession of visible colour changes due to disintegration of red blood cells of the extravasated blood releasing the haemoglobin, which is then acted upon by tissue enzymes and broken down into haemosiderin, haematoidin and bilirubin, imparting respective colours to the tissues. These colour changes start at the periphery and proceed towards the centre of the contused area.

As in case of abrasions, only a general approximation of the interval between the infliction of a bruise and examination may be gathered. Indeed, the degree of reluctance of the doctor to label the precise timings for infliction of the injury may be a measure of his/her competence. The colour changes in a common superficial bruise of average size may occur more or less in the following order:

- When fresh, a bruise is reddish in appearance.
- Within a few hours, it becomes bluish and changes to bluish-purple by the second day, bluish-black by the third day and continues as such till the fourth day.
- By the fourth/fifth day, it appears brownish due to presence of haemosiderin, an iron-containing pigment.
- By the fifth/seventh day, it is greenish due to presence of haematoidin.
- By the seventh/tenth day, the bruise assumes yellow colour due to presence of bilirubin.
- The yellow colour slowly fades in tint, and the normal colour of the skin is restored by about 2 weeks.

In subconjunctival haemorrhages, all the colour changes may not be noticeable due to diffusion of atmospheric oxygen through the conjunctival tissue, and the pigments liberated after the breakdown of RBCs change to bilirubin without showing noticeable stages of haemosiderin and haematoidin. More or less similar observations are seen in meningeal haemorrhages, where some oxygen is supplied by the CSF.

CIRCUMSTANCES WHERE EXTERNAL EVIDENCE OF BRUISING MAY BE ABSENT

- Where the site of injury is on yielding part such as anterior abdominal wall, there may not be any external bruising, though internally rupture of viscera may be noticed.
- When the offending weapon is yielding in nature and of flat surface type such as sand bag.
- In case of blow over sole of foot and palm of hand, where subcutaneous tissues are thick with no pliable subcutaneous fat, bruising may be absent notwithstanding application of gross violence.
- Where the body surface to be hit is covered with thick rug, blanket, quilt, etc., there will not be any bruising externally, but deep seated bruising underneath may be evident.
- Bruising may also be absent, notwithstanding the application of great violence, if the pressure is maintained until death ensues. Spilsbury mentioned a case where continued pressure of a wheel of a vehicle on chest left no external bruising. Similarly, external bruising may be absent when a grip in throttling is maintained until death occurs, particularly when some soft material is interposing between the skin of the victim and grip of the assailant.

ANTEMORTEM/POSTMORTEM BRUISING

A certain amount of swelling and colour changes are usually found in a bruise caused during life. Coagulation of the effused blood into the subcutaneous tissues along with infiltration of blood in the tissues is no longer to be seen in postmortem bruises. However, a bruise is likely to be disfigured by decomposition and then it may pose a problem in its differentiation. Sir Robert Christison proved by experiments that it was possible to produce a bruise within about 2 to 3¼ hours after death which, would be difficult to distinguish from the one caused during life but he observed that great violence would have to be applied and even then the resulting bruise might be smaller than what would have been produced by similar means during life as there would be no internal pressure in the small vessels and bleeding would be a passive ooze rather than active extravasation.

MEDICOLEGAL CONSIDERATIONS

- A bruise is an evidence of application of blunt force and usually the circumstances are of accident or homicide. It is unusual for a suicide to bruise self by blows or some other object because they occasion pain. But **with a view to supporting a false charge**, bruises are sometimes simulated by application of some irritant substance such as the juice of *bhilawa* (marking nut) or the root of *chitra* (*Plumbago zeylanica*) or *lal chitra* (*Plumbago rosea*). The marks produced by these substances appear like bruises but are dark-brown in colour with the margins covered by vesicles and the adjoining skin is red and inflamed. Similar marks may also be seen on the tips of the fingers that might have been produced during the process of scratching [see *Plumbago rosea* (*Lal Chitra*) and *Plumbago zeylanica* (*Chitra*) in the Chapter "Irritants of Plant Origin"].
- The importance of bruising **lies in the site and the organ involved** and the contusions of various organs, therefore, may assume different degrees of importance. Contusions of vital organs like heart or brain (especially the brain stem) may cause marked derangement of functions, and even death may ensue. In case of heart, larger contusions by virtue of swelling and disturbance in muscle action may prevent adequate emptying of heart and lead to heart failure. Furthermore, as the contusion ages, the area of heart becomes weaker, which may ultimately lead to rupture of heart and loss of blood into the pericardium and death due to cardiac tamponade.
- Since the contusion results from tearing or rupture of blood vessels with extravasation of blood into the tissues, the volume of the remaining circulating blood gets diminished

by each contusion. It has been reported that even a trivial bruise may lock up 20–30 cc of blood.

- **Patterned bruising,** as stressed already, may be quite helpful in providing clue towards the nature of the agent used in the assault, though they are comparatively less informative than abrasions because they may show delayed appearance, ectopic appearance, and unlike abrasions, incapable of indicating direction of application of force.
- Healing of a bruise imparts some help in the broad determination of age.
- Bruising and abrasions of the shoulder blades indicate firm pressure on the body against the ground or other resisting surface.
- Bruising of the scalp may easily be overlooked. **It is better felt than seen.** Careful search is necessary, and there should be no hesitation to shave the suspected area, if required.
- Like abrasions, bruising of some particular parts of the body may be indicative of some peculiar offences and the description need not be repeated. To cite an example, small bruises associated with nail marks on the neck and inner sides of the thighs of a female may be indicative of sexual assault. Typical small bruises, **the so-called 'six-penny bruises'** (name derived from the size of the small coin formerly used in Britain) may be produced from forcible poking or pressure with the fingertips.
- Some deep seated contusions, especially in the highly pigmented skin of some individuals (swarthy skinned persons or those with deep tans), may escape observation: **Incision of the suspected area** is a well-recognised and time-honoured technique to locate and demonstrate such deep seated and otherwise poorly appreciable contusions.

Lacerations

A laceration is a **rupture or tear or a split in the skin, mucous membrane, muscle or any internal organ, involving depth more than the covering epithelium of the skin or that of an organ that is produced by application of blunt force**.

Lacerations may be produced by any one or more of the following means:

- Passive agents like ground (lacerations due to falls are most frequent), edge of a pavement or stairs, parts of furniture or of a building, etc.
- Vehicles—it may be of any kind, and it is difficult to name them all in the present scenario.
- Blunt weapons—it may be any mechanical object/instrument or a part of the body being used as a weapon, which may include fist, hands covered with boxing gloves, feet (usually the soles) or the booted foot, and other easily available materials like sticks, stones, rods, bottles, etc.

TYPES OF LACERATIONS

Many factors influence the formation and appearance of a laceration such as the configuration of the object delivering the force, the type of the tissue, the area of the body involved and the velocity of the offending weapon. If the impact produces bleeding into the surrounding tissues also, it may be termed as **'contused laceration' or a 'bruised tear'**. If the margins of laceration are denuded of epithelium by the same impact, it may be called as **'abraded laceration' or a 'scraped tear'**.

Split Laceration

Split lacerations occur **when the soft tissues are 'sandwiched' between a hard unyielding deeper structure and the agent applying the force**. Scalp lacerations are the typical examples that occur when the scalp tissue is crushed between the skull and some hard object such as the ground or some blunt instrument so that many blunt injuries of the scalp may present difficulty in their differentiation from the incised wounds.

When the skin is closely applied to the bone and the subcutaneous tissue is scanty, blunt force may produce a wound that by linear splitting of the tissues resembles an incised wound. Such wounds may, therefore, be termed as **'incise looking wounds'**. The sites notorious for production of such wounds are scalp, face, eyebrows, zygoma, iliac crests, the perineum, the shin, etc. (Fig. 14.4). In general, they should be taken as lacerations unless shown to be incisions by careful examination. The wound should preferably be examined under good light and using a magnifying glass at the earliest available opportunity so that the process of repair may not affect/modify its appearance. With a fall on the knee or elbow with limb in a flexed position, same thing can also occur.

Stretch Lacerations

These may **result due to heavy forceful frictional impact by a blunt force exercising localised 'pressure with pull'**. Overstretching of the skin and subcutaneous tissues may cause laceration with flapping of the skin. Direction of application of the force may be gathered from this 'flapping'.



Fig. 14.4 Lacerations around the ankle in an accident victim.

Grinding Compression or Avulsion Lacerations

The grinding compression by a weight such as a wheel of a heavy vehicle or some heavy part of machinery passing over the limb may produce avulsion of the skin and subcutaneous tissues from the underlying structures (shearing lacerations). Commonly seen in traffic accidents where the rotating force of a wheel of a vehicle tears off the skin over the relatively large area. This is sometimes termed as **'flaying'** and most frequently occurs in legs where a heavy vehicle passes over the body that has already been knocked down to the ground. The torn skin may show extensive abrading and bruising of the margins. Internally, the organs can be avulsed or torn off partly or completely from their attachments. Rarely, the skin may not show signs of injury but the subcutaneous soft tissue is avulsed from the underlying fascia or connective tissue, producing a pocket. This is seen usually over the back of thighs of pedestrians struck by motor vehicles.

Tears

Tearing of the skin and subcutaneous tissue can occur from localised impact by or against some hard, irregular object like motorcar door handle, radiator mascots or from blows by broken glass bottles, fall over rough projected objects, etc.

Many books have described another type, viz., 'Cut Lacerations'. The term, though sounds good theoretically, encompasses two words with contradictory implications. The author has never used this term as it is likely to invite unnecessary questioning in the court. The edges of wounds produced by not so sharp edges of a heavy cutting weapon like axe, chopper, hatchet, *gandasa*, spade, etc. may not be as sharp as those of a wound produced by a light cutting weapon and usually show evidence of bruising in and around. Such wounds are often associated with injuries to the deeper structures or organs. If the weapon has been struck obliquely, 'bevelling' of the margins will suggest the direction of the blow. (Also see Chopping Wounds in Chapter 15.)

FEATURES OF LACERATIONS

The characters of a laceration that may be helpful in distinguishing it from an incised wound include the following:

- The margins are ragged, irregular and uneven and may show tearing of the extremities at angles diverging from the main laceration, the so-called 'swallow tails'.
- As stated earlier, because of crushing and tearing nature of production of lacerations, there is usually associated abrasion or bruising, though the zone may be narrow, if the lacerating force acts at right angles to the surface.
- The edges of laceration may give an indication of direction, in which the blow or force was applied.
- Depth of the wound presents bridges of irregularly torn fibrous tissue, blood vessels and nerves, etc. across the interior of the wound.
- Soiling of the wound by mud, sand, glass, brick particles, vegetation, machine oils, hair, fibres, etc. may cause these materials to be found embedded in the wounds and is of great medicolegal value.
- Lacerated wounds do not bleed much as the vessels are crushed and torn but not cut evenly, thus facilitating haemostasis to some extent. However, as mentioned under 'Injuries to the Scalp', temporal arteries may bleed freely as they are firmly bound and unable to contract.
- Lacerations over the hairy areas will show hair bulbs crushed or torn and the crushed hair bulbs may get thrust into the depth of the wound.
- Wounds produced by the edge of a broken glass, earthenware, crockery or projecting flints of stones or similar objects are basically lacerated injuries showing jagged, irregular contused margins. Pieces of the material may be found embedded in the wound, suggesting nature of the wounding material.

PATTERNED LACERATIONS

Lacerations do not reproduce the shape of the agent as distinct as are produced in case of abrasions and intradermal bruises. However, rarely the shape may be recognisable wholly or partially and, probably, the typical example may be cited in case of a hammer blow upon the head, giving rise to a crescentic laceration or the laceration corresponding to varying portion of the circumference of the striking head of the hammer depending upon the manner in which the blow is imparted and the portion of the edge of the hammerhead in operation.

Without going into many other instances mentioned in various books, I would stress that **it is unwise to be specific in extending opinion in case of lacerations** as the same may be misleading because a metal rod of circular cross-section may cause an injury identical to one with a square cross-section. Further, a single heavy blow with a hard blunt weapon may result in more than one lacerated wounds.

ANTEMORTEM/POSTMORTEM LACERATIONS

The distinction between lacerations inflicted before and after death should depend upon the presence or absence of vital reaction and, of course, the extent of bleeding, coupled with bruising of the margins. However, it may be kept in mind that quite copious bleeding can occur in the scalp even after death, especially when the body is in dependent position.

Eversion and gaping of the margins is usually seen when it has been inflicted during life but here again caution must be exercised as the muscles have the capacity to contract for sometime after death, and further the posture of the body at and after the time of death may cause gaping simply due to passive gravitational effects.

Bodies recovered from water may pose even greater difficulties as any blood will have been washed away and the decomposition, which is usually pronounced in such cases,

further hampers differentiation. Such bodies may also bear gross lacerations produced after death by striking against some hard pavement or blunt object. The nature and extent of injuries and absence of vital reaction are helpful in these circumstances.

Occasionally, when the wound is inflicted in the perimortem period, i.e. immediately before or after death, the distinction may be impossible as no vital reaction is likely to be found. However, general features like extent of haemorrhage, gaping of the margins, bruising of the edges and the experience of the doctor may be called into play for such differentiation.

MEDICOLEGAL CONSIDERATIONS

Lacerations are usually accidental or homicidal and rarely suicidal as in the case of a suicidal fall from a height or suicidal jumping/lying in front of any running vehicle.

- Organs may suffer extensive parenchymal damage beneath intact surfaces. Examples may be subcapsular hepatic lacerations and subpleural pulmonary lacerations. Hence, the absence of external trauma does not preclude the presence of grave internal injuries. In fact, the most common form of 'concealed' fatal trauma, whether involving head, neck, thorax or abdomen, is seen in blunt force impacts. Converse can also be true in some cases, especially in case of chronic alcoholics dying of some other natural causes but may present incidental external injuries.
- Blunt trauma can be fatal with neither external nor any internal evidence of injury. For example, a sudden forceful impact to the chest against the area of the heart may be responsible for immediate death of the victim without any demonstrable damage to the chest wall or the heart. Sudden disturbance in the functions of the heart may be the cause.
- Scalp lacerations, which are often encountered in homicidal attacks, carry great medicolegal importance and may classically be confused with incised wounds. Much has already been talked about on these lacerations.
- Another unusual or unexpected result that may be seen in laceration causing trauma is the delayed tearing or rupture of the organ, occurring after considerable time of infliction of blow. Heart, liver and spleen are likely to behave in this manner.

Associations of Abrasions, Contusions and Lacerations

Lacerations may be found in association with abrasions and bruises and, in fact, double or triple lesions are extremely common. The same object/weapon may cause abrasions under one impact, bruises with the other blow and lacerations with the next. Alternatively, a single blow/impact may even result in production of all the three types of lesions.

CASE: BLUNT FORCE IMPACTS UPON THE CHEST

On the night of 31st July, 1998, some altercation took place between two tenants over sharing of the common roof for sleeping. One was at the ground floor and the other at the first floor. Due to hot-humid atmosphere, both the tenants desired to use the common roof for sleeping. Hot exchanges supervened but they were soon brought to rest by the intervention of the neighbours. On the next day, the tenant of the first floor along with his brother and others beat up the tenant of the ground floor. As alleged, one inflicted a *lathi* blow on the chest, another dashed his head against the chest and some conveyed blows. The police, on receiving information, reached the scene and transported the victim (about 20 years of age) to a hospital where he was declared 'brought dead'.

Postmortem examination showed presence of a $10 \times 3\,cm^2$ reddish contusion on the front of chest running from a point 3 cm below the angle of the sternum, going obliquely downwards and to the right, ending at a point 6 cm medial to the right nipple. Heart showed rupture ($1.0 \times 0.75\,cm^2$) on the anterior surface, 2 cm above and to the right of the apex, with consequent accumulation of blood in the pericardial and pleural cavity. On 28th October, 1998, a query from the police appeared—whether the injury to the heart as mentioned in the postmortem report was caused by hitting the head or striking a *lathi* blow upon the chest?

In this context, it has repeatedly been emphasised that organs may suffer extensive damage beneath intact surface. Because of their thinner walls, wounds of the auricles are more dangerous than those of the ventricles; due to the same reason, right ventricular injuries are more dangerous than those of the left ventricle. In fact, most common form of 'concealed' fatal trauma is usually encountered in blunt force impacts. Traumatic rupture of heart, though it usually involves the right ventricle as it exposes its widest area on the front of the chest, can cause injury to the left ventricle too. Death is usually immediate, but may be delayed for hours or even days in cases where original rupture being small and sealed by blood clot in the state of shock but rent getting increased with the return of blood pressure or with its rise or the other situation may be where the rupture incompletely involves the wall and the rent getting enhanced by increase of blood pressure on exertion. Heart can get ruptured even in the intact pericardium; if the pericardium is also involved, heart may herniate through the tear and get self-strangulated.

Falls

"The higher you climb, the harder you fall"—a Chinese proverb.

In the ordinary course of affairs, falls are of common occurrence; the severity not necessarily being directly related

to the distance from which the person falls. Instances are available in the literature wherein skull fractures and brain damage have been reported from trivial falls. A case has been reported in the literature wherein occipital fracture occurred in the drunken person who was being lifted by his drunken friends, his head and shoulders being allowed to fall back on the concrete surface from about half sitting position. Falls in old people can lead to fractures of post-cranial skeleton, especially the neck of femur (osteoporosis being the major reason for such fractures). In infants, skulls could be fractured against a variety of floor surfaces from passive falls of only 34 inches (Weber). Falls from a standing position can occur during a drunken state, fainting attack, and of course, from an assault.

In circumstances of assaults, the gravity of injuries produced by direct trauma may be considerably less than the injuries sustained from the indirect result. For example, a blow on the chin (direct impact injury) may cause the victim to fall and receive head injury by striking the head against the ground or some object. Here, it is worth adding that contrecoup cerebral damage can occur notwithstanding absence of injury to the scalp or skull. The direct impact under such circumstances may be biologically insignificant from the standpoint of causing death, although it is considered as a crucial factor in the legal outcome of the assault. **Falls from a height** (usually from a building) are common in suicide and in some accidents. Falls from a height resulting from homicide are uncommon and may be associated with defence type or offence type injuries while resisting being pushed from a balcony, roof or window. However, such injuries may be absent if the deceased was managed by surprise. Further, when present, they are usually very difficult to be distinguished from the fall injuries unless they are quite distinctive, for example, fingertip pressure marks and other bruises plus abrasions that are inconsistent with an uncomplicated fall. Further, presence of any finding relating to sexual assault should raise suspicion in this direction. It is prudent to investigate the possibility of substance abuse when young/young adults fall from a height (see Case ahead).

Goonetilleke (1980) published some research on the aspect of distance from the wall where the body was likely to land and also showed that the severity of brain damage was not directly proportional to the height of the fall. Much depends upon the fact that whether the victim fell passively from near the wall

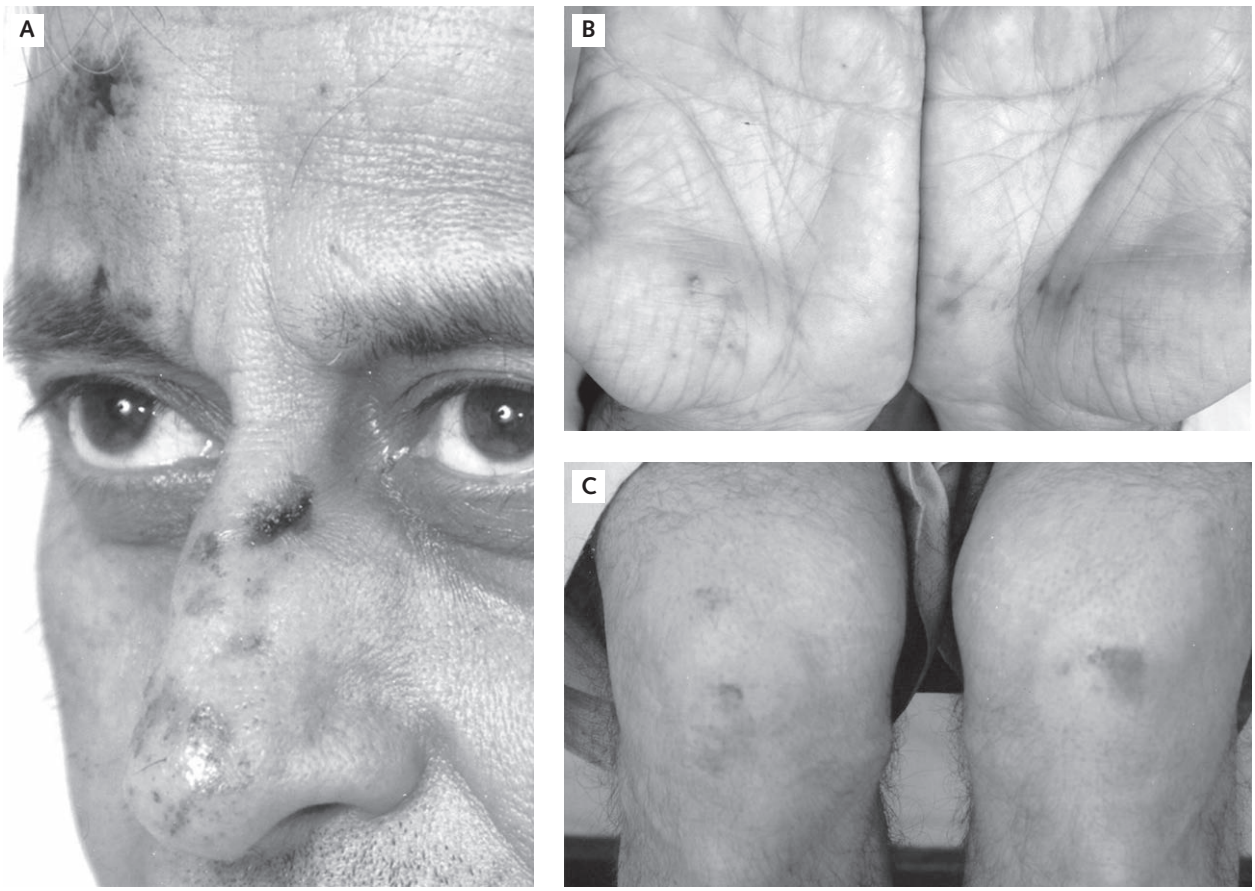


Fig. 14.5 Injuries in an ordinary fall: **(A)** Face showing (*i*) abraded contused right side of the forehead, (*ii*) involvement of front and left side of the nose near its root (this site, though an unanticipated one, received injury through dragging of antenna of the mobile phone being carried in the left pocket of the shirt). **(B)** Hands showing intradermal contusions against thenar areas. **(C)** Front of knees showing abraded contused areas.

or projected himself/herself outwards while commencing the fall. According to Isbister and Robberts, it is more common for a jumper from a lower floor to strike the ground feet first and from above the 13th floor to strike the ground head first. Further, the body may strike the ground in a number of different attitudes, the primary impact usually being exhibited through the area of the most severe injury. However, two areas of the body may strike almost simultaneously—such as head and shoulder.

It is obvious that factors like rate of change of direction and speed of movement are the major ones concerned with the outcome of the injuries to the tissues. Other factors like nature of the surface of impact (the more resistant the surface, the greater are the deceleration forces), the size of the impacted area, and the target factors play their own role. (Energy liberated by an impact may be transferred through tissues without producing significant local damage and yet, capable of producing serious injury at a site comparatively remote from the point of contact between the victim and the traumatising surface. Further, violent displacement of gas or fluid within the hollow viscera can result in injuries from explosive pneumatic or hydrostatic forces set into motion by the transmitted energy). According to Knight (1996), a deceleration of up to 30 G can be sustained without injury, while forces of up to 200 G may be survived for a short interval, provided they are applied to the long axis of the body. However, such theoretical considerations may be confounded by the following circumstances:

- Tumbling, rotation or spiraling of the body during the free fall resulting in some dissipation of the kinetic energy and therefore, the actual magnitude of primary impact.
- Secondary or even tertiary impact with the ground or other object in the vicinity due to bouncing of the body after the primary impact.
- Collision with intermediary objects/obstructions that is sufficiently sturdy to retard the fall and thereby reduce the final velocity at impact. Further, collision with such structures could also produce serious injuries unconnected with terminal impact. Bernard Knight reports a case wherein a suicide while falling from the twentieth floor, fell onto a fence and was completely transected at the waist level. However, at the other extreme, biological and circumstantial variability may allow for some remarkable escapes.

Figure 14.5 (A–C) shows areas likely to be involved in an ordinary fall. The victim received these injuries on getting stumbled and falling with outstretched hands (the usual reflex action to protect other important parts of the body) on a concrete floor while hurriedly boarding a bus.

15 CHAPTER

Injuries by Sharp Force

After going through this chapter, the reader will be able to describe: Incision/cut/slash, etc. | Features of incised wounds | Bevelled cuts | Hesitation cuts | Chopping wounds | Types and features of stab wounds | Factors influencing size, shape and configuration of stab wounds | Injuries by blunt penetrating/dull instruments | Wounds by glass | Pointers towards suicide/accident/homicide

Injuries by sharp-edged instruments/weapons result in either 'cutting' or 'stabbing' depending upon whether the instrument has been used in a swiping manner or in a thrusting manner. Information of great importance can be derived from the careful examination of a 'cut' or a 'stab' that can be helpful in reconstructing the events. Therefore, either deserves to be dealt with separately.

Incision/Cut/Slash

Accidental incise wounds can occur and usually are not so severe in nature. Home, especially kitchen, or workplace/industry is the typical place of occurrence of such wounds. Casual use (slippage of blade while sharpening a pencil), misjudgement, lack of skill/inclination, etc. may be the factors leading to an accidental injury by some sharp instrument/tool. Another instance may be seen in the case of burglars who receive injuries from broken window glass while housebreaking. Sheet glass or mirror glass may also prove hazardous in innocent circumstances. Death may result from cut-throat injury or involvement of area of the groin, or from falling or impalement on a large sliver of glass.

There have been occasions when slashes have been deliberately aimed to cause disfigurement and humiliation. Face is the main target for such type of injuries. The ensuing scar may form an important factor towards legal outcome. Wounds may be discontinuous where they cross a natural fold or trench, as may be the case when a single slash involves eyebrow and the summit of cheek. Such disconnected injuries may be misinterpreted as separate passes of the weapon. As a general rule, incisions inflicted by an assailant tend to have a sweeping quality. They usually show rapid deepening. There is seldom any repetition in the same plane, although they may criss-cross.

FEATURES OF INCISED WOUNDS

Margins

As stated above, they are clean cut, regular and well-defined. They are free from any bruising when caused by light cutting weapons, but bruising and some irregularity of the margins may be present when heavy and not so sharp-edged weapons like shovel, *kassi*, axe, tangi, etc. have been used. The usual incised wound is linear with everted margins but in the areas where the skin is loosely applied to the body as in the scrotum or neck, the margins might appear inverted and jagged because the skin is pushed in front of the blade before it is divided. Even then their true nature can be determined by careful observation or examining under a magnifying glass. Such situations are usually encountered in scrotum, neck, axilla, palm of the hand, and so on. Further, a single strike over the areas where skin is folded and puckered usually produces a series of incised wounds, one separated from the other by a bridge of normal skin.

Length

Being produced by a sweep of blade, the wounds have 'length' as their greatest dimension. It does not bear any relationship with the length of the blade of the weapon.

Breadth

The breadth of the wound will depend upon the extent of 'gaping' of the margins because of the retraction of the divided tissue associated with eversion of the margins (see details under 'Stab Wounds').

Depth and Direction

Usually, the incised wounds are deeper at the commencement (as is expected from the process of drawing or sweeping

through which they are produced) except in case of suicidal cut-throat injuries with hesitation cuts at the beginning. Towards the termination, the cut becomes progressively shallow, known as **'tailing of the wound'**. Consequently, the depth of the incised wound with the 'tailing' will suggest the direction in which the force was applied.

Shape

Usually, the incised wound is fusiform or spindle shaped due to comparatively more retraction of the edges in the centre. When inflicted upon the convex surface of the body like the occipital region or buttocks, the wound may be crescentic or semilunar in appearance, so also when the blade of the weapon is curved. The shape may be 'V' or 'A' or wound may even assume any bizarre appearance depending upon the changing attitudes of the victim and the assailant as is often expected during struggle.

Haemorrhage

As the vessels are divided cleanly, profuse haemorrhage is frequently a feature of incised wounds. Even minor incision of a vascular part, say a finger tip, may occasion troublesome bleeding. An artery 'nicked' or 'incompletely severed' will bleed more freely than one that is cut through and through, because of its inability to contract or retract. Bleeding, however, being external is more amenable to prompt treatment than the hidden internal bleeding in case of a stab wound.

Bevelled Cuts

In case of an oblique strike by a sharp-edged weapon, bevelling or undermining of the edges may be present indicating the direction from which the slashing stroke was imparted. Occasionally, a 'flap wound' may be produced when the stroke is nearly horizontal to the body, particularly when some heavy cutting weapon has been employed.

Hesitation Cuts

The situation and circumstances of incised wounds of the throat deserve separate mention. They are rarely the result of an accident, and therefore differentiation between suicidal and homicidal needs to be established (Table 15.1).

The self-inflicted wound, when made by a right-handed person, normally begins high on the left side and passes downwards across the front to end on the right side of the neck. Sometimes, the wound may lie horizontally across the front of the neck. There may be present multiple superficial cuts around the commencement of the main wound. These superficial 'tentative' or 'hesitational cuts' indicate divided state of mind of a person as it is usual human instinct to preserve life. The throat wounds can be accompanied by incised wounds of the wrist or tentative cuts elsewhere. Suicidal cut-throat wounds may lead to instantaneous rigor, when death ensues. Therefore, if the weapon is found firmly grasped in the hands, it is strongly in favour of suicide.

Homicidal cuts lack the presence of tentative cuts and may be accompanied by other wounds upon the body. It tends to lie lower in the neck and assume horizontal position. There may be bevelling of the margins and they are likely to be toothed or may show some irregularity of the margins at places because the skin is thrown into folds before severance. Major blood vessels may be involved in homicidal cut-throat wounds, but they are usually protected by the sternomastoid muscle, when the head is extended by the suicide.

Chopping Wounds

Chopping wounds result from hacking or chopping motion made with a fairly sharp and relatively heavy weapon like axe, hatchet, cleaver, saber, bayonet, etc. The nature of the wound depends upon the sharpness of its blade, direction and angle of the blow, portion of the instrument that actually operated in inflicting the injuries and differences in curvatures of the tissues involved.

There is often a spectrum of injuries lying between typical lacerations and incisions, which are determined by the sharpness or otherwise of the blade of the weapon/instrument. It is important to make distinction, because it may provide an indication as to the weapon used and may prove crucial to the legal outcome of an attack. Hatchets, axes, spade, etc. are mostly kept unattended—ground but not honed. All such ill-maintained weapons have ground edges and often present some nicks and/or firm rusty deposits on their blade including the edge, preventing the weapon to produce pure incision. When sharpened, such weapons produce devastating incised wounds. Even bone may be transected with comparatively little effort. The action of chopping may produce injuries that provide some indication as to the shape and stoutness of the blade employed. (Homicidal axe blows are usually aimed at head, producing trench-like skull fractures with comminuted margins. Repeated blows may produce virtually complete fragmentation of the cranium. A near tangential sweep to the head may lift a slice from the outer table of the skull—a circumstance speaking for itself.)

At times, blunt pole at the back of the blade or wooden handle of an axe may be used depending upon the circumstances. In Babu Loshi vs. State of Uttar Pradesh [(1987) 2SCC, 352], the Honourable Supreme Court held, "When three persons beset themselves on a victim and attack him in quick succession, it is not possible for a witness standing at a distance to say where each cut or stab fell on the body and whether the blade or the handle caused the one or the other of the injuries. It is quite likely that the handle of the axe also came in contact with the body of the victim and likewise the spears had also been used now and then as a *lathi* to inflict blows. Thus, abrasions and contusions are also possible from axe or spear where more than one person uses them on the victim in quick succession".

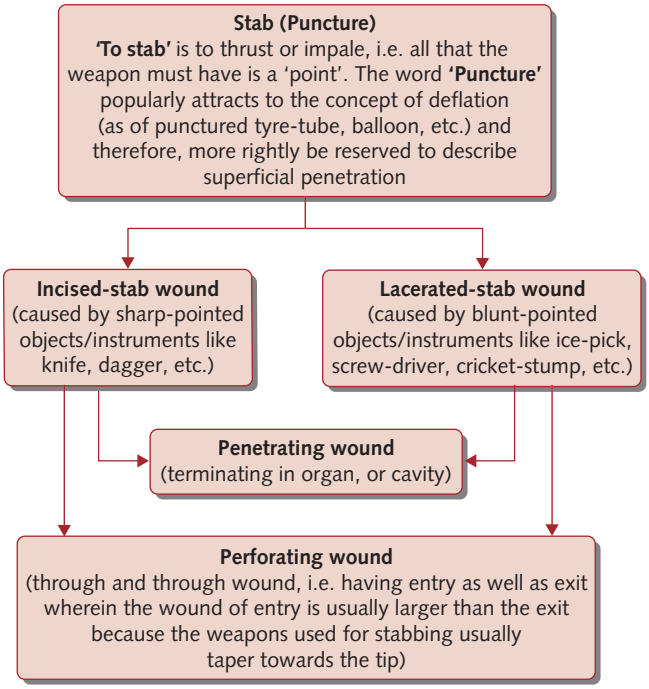
Table 15.1 Elementary Distinctions between Suicidal and Homicidal Cut Throat

Features/points	Suicidal cut throat	Homicidal cut throat
Site	Mostly on the left side and front and partly on the right side of neck (in case of right-handed person) and placed high up on the neck	Mostly on the sides of the neck and placed at a lower level
Severity of the wound	One or two are severe, others are superficial, the so-called hesitation cuts	Many of equal severity may be present
Secondary wounds	Self-inflicted incised wounds may be present on other approachable parts of the body	Other homicidal wounds, defence cuts, marks of resistance usually present on other parts of the body
Direction	From left to right and above to downwards in a right-handed person	Transverse/horizontal and from right to left when the assailant's position is below the level of the neck of the victim (a homicidal wound on the throat may resemble a suicidal one if the assailant has inflected it from behind the victim or by standing on the right side while the victim is lying)
Tailing	Gradual deepening and shallowing with tailing at the right end of the wound (in a right handed person)	Usually exhibits bold cutting at the commencement
Hesitation cuts	Marked by tentative hesitation cuts either at the beginning, above or below the main wound	No such cuts
Defence cuts	Absent	Frequent 'protective cuts/defence wounds' on the grasping surfaces of hands or on back of forearms
Vessels	Carotid arteries are usually spared because, before injuring, the victim stretches his/her neck upwards, when these vessels shift behind the sternomastoid muscles	The vessels remain vulnerable due to lack of this manoeuvre
Bleeding	As the wound is usually inflicted during standing or sitting position, a good amount of blood trickles down in front of the chest and abdomen	No particular pattern
Weapon	Usually present, sometimes firmly grasped by cadaveric spasm	Usually absent. Rarely, an ultracriminal mind may manage to place the weapon in the hand of the victim so as to mimic suicide
Foreign materials like hair, etc.	Substances like foreign hair, button, etc. are not found in the hands of the victim	May be present in the hands of the victim, gripped in a state of cadaveric spasm while exercising resistance
Corresponding cuts on clothes	Absent as the person cautiously removes the clothes to get a clear field of the area	Cuts may be present on clothes as the assailant, being in haste, is unmindful about the clothes
Circumstances	Closed room bolted from inside or bedroom or secluded place, which appears undisturbed. Sometimes, the body may be found in front of a mirror, which may show blood splashes	Considerable disturbance at the scene and even elsewhere is usually found
Suicidal note	May be present	Absent

Stab/Punctured Wounds

The word 'stab' means 'to wound or pierce with a pointed weapon' and the word 'puncture' also means 'to pierce with a pointed object/weapon/instrument'. Therefore, these words are often used interchangeably. However, the term 'stab wound', in the popular sense, is confined to the wound that is caused by thrusting a sharp-edged and pointed weapon like knife, dagger, etc. Categorisation of the puncture wounds depending upon the nature of weapon, as described ahead, further lends support to this aspect. Since the wound is produced by a thrusting or stabbing motion, the depth of the wound is greater than the dimensions of the surface wound. Depending upon the severity, they may be grouped as follows (Flowchart 15.1):

- **Penetrating wound:** Here, the wound into the body cavity or viscus may be a joint cavity or scrotal sac.
- **Perforating wound:** The wound is called perforating when the weapon pierces through the whole thickness of any part



Flowchart 15.1 Categorisation of stab wounds.

of the body thus producing two surface wounds, i.e. wound of entrance and wound of exit. The wound of entrance is bigger than the wound of exit due to tapering of the blade of the weapon and its margins will be inverted, whereas the margins of exit wound will be everted. The track of the wound, starting from the entrance towards its termination, is convergent in contradiction to the track of a wound produced by a missile at short ranges where it is divergent.

Depending upon the nature of weapon/instrument (i.e. whether some sharp pointed or blunt pointed weapon or instrument has been used), they may be grouped into:

- **Incised puncture/penetrating/perforating:** Examples may include wounds produced by knife, dagger, *kirpan*, etc. In the common usage, such wounds are designated as stab wounds as described earlier.
- **Lacerated puncture/penetrating/perforating:** Examples may include wounds produced by icepick, screwdriver, pointed end of the cricket wicket, etc. (Table 15.2).

FEATURES OF A STAB WOUND

When a sharp-edged weapon (like a clasp knife, dagger, *ballam*, etc.) is used, the wound is usually produced by the combined action of the tip and the cutting edge. Various features may be as follows:

Length

The length of the surface wound will be slightly smaller than the width of the weapon up to which it has been driven in because of gaping of the wound margins, unless rocking of the weapon or lateral movement of the victim or assailant enlarges it. Repetition of stabbing without complete withdrawal may double the entry wound or may produce an erratic surface wound. The length of the wound should be measured by gently opposing the skin margins, which more accurately approximates to the length when the blade of the weapon was in situation (Fig. 15.1). The component of gaping may be exaggerated depending upon the plane in which the muscle fibres have been cut and modifying the length accordingly. This has been discussed in detail under heading 'Shape'.

Breadth

The breadth of the surface wound usually does not have any approximation to the thickness of the blade of the weapon, again due to gaping of the wound margins (Fig. 15.1). In the dead bodies, putrefaction may cause eversion of the edges and increase in breadth of the surface wound.

Depth and Direction

The depth of wound (i.e., the length of the track) is a guide to the length of the blade in operation, but this is not by any means infallible as multiple factors are involved.

Actually, the depth of wound may frequently exceed the length of the blade that caused it. Essentially, this is due to the indentation or caving-in of the body surface as a result of thrust. Abdominal wall is the most glaring example in this concern where the yielding anterior abdominal wall during the exercise of bearing the impact of the force and the pressure of the hand or fist may cave-in considerably so that the tip of the weapon (though relatively small length of the blade of the weapon has actually been in operation), can reach quite deep into the abdominal cavity. The abdominal wall will come to its original state after withdrawal of the weapon. Even the chest wall is no exception as the rib cage is amenable to considerable caving-in. The movements of chest, i.e. expansion and retraction during the process of breathing, should also be taken into account. Stab wounds of 6 inches and 7 inches depth have been reported with a 4-inch pocket knife. Further, the lung, if punctured, may collapse and be drawn upwards and backwards when the impact is from the front side of the chest, thus giving increased measurements at autopsy. Conversely, if the stabbing is from the back of the chest resulting in puncture and collapse of the lung, then due to shift of the lung posteriorly to the point of its attachment, diminished measurements result from the wound of entrance to the wound of termination in the lung as compared with the length of the blade of the weapon actually in operation.

An additional factor that may also be given due consideration is that the relationship of intra-abdominal organs at the time of assault in a living individual in erect or bent posture is not same as is observed during postmortem on the autopsy table (Grandwhol's *Legal Medicine,* 1968, 2nd ed.).

Table 15.2 Differences between Lacerated and Incised Wounds

Traits	Lacerated wound	Incised wound
Weapon/ agent	Caused by blunt weapons or fall against hard objects/surface	Caused by weapons with sharp cutting edges
Situation	Usually against bony prominences or projecting areas	Anywhere on the body
Shape	Usually irregular (varies as per causative agent)	Usually linear or fusiform/spindle shaped
Margins	Ragged, irregular, uneven and often accompanied with bruising and abrasions. Retraction and eversion of margins are not usual	Always regular, even clean cut and everted with no bruising or abrasions when caused by light sharp cutting weapons (bruising usually present when caused by heavy cutting weapon)
Dimensions	Varies with the nature of offending weapon/object/ surface, or the mechanism of infliction. Usually shallow in depth	Usually length is greater than the depth and breadth (breadth is greater than cutting edge of the weapon due to gaping)
Condition of surrounding or underneath tissues	Soft tissues underneath show evidence of tearing and splitting with bruising in and around. There may be some loss of tissues with exposed hair and hair bulbs crushed or torn	Soft tissues underneath clean cut. Hair and hair bulbs clean cut. No loss of body tissues unless a 'flap' is sliced off
Bleeding	Not pronounced, spurting of blood is uncommon, except in case of temporal arteries because of anatomical disposition	Bleeding is usually profuse (incompletely cut or 'nicked' artery will bleed more freely because of its comparative inability to contract or retract)
Foreign matter	Trace evidence often left in the wound will help to connect the crime with the offending object and place of occurrence	These are not commonly noticed
Wearing apparels	Coverings over the affected site may be torn	Coverings over the affected site usually show corresponding cuts (when garment is loose or folded, there may be multiple cuts on the clothing)
Multiplicity	Multiple lacerated wounds with bruises and abrasions over different parts of the body indicate traffic accident or fall from height. Bevelling of margins is not noticed (though tissues may get lacerated in a flap like fashion)	Multiple superficial incised wounds over accessible body parts suggest self-infliction. Presence of 'tentative/hesitation' cuts at the neck region with one or more deep wound(s) having distinct tailing indicate suicidal phenomenon. Multiple deep incised wounds over different body parts with defence wounds indicate homicidal phenomenon

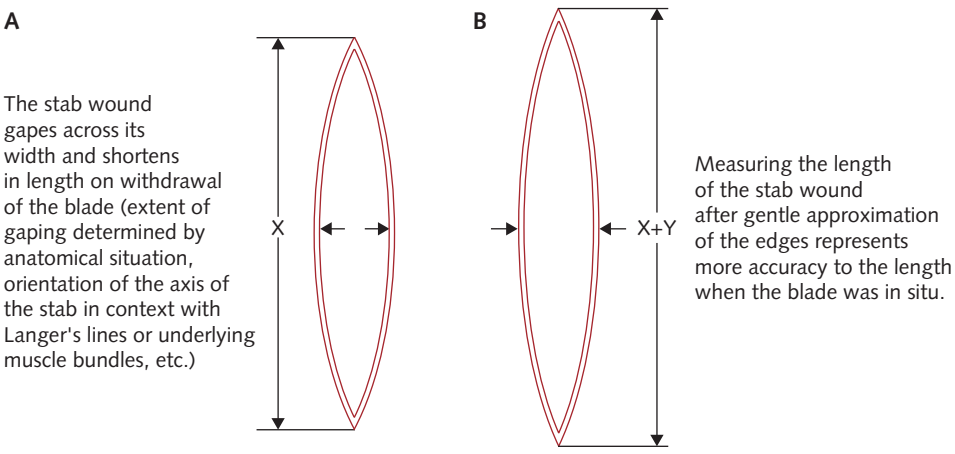


Fig. 15.1 Medicolegal aspects of stab wound: **(A)** phenomenon of gaping of a stab wound; **(B)** need for measuring the length of the stab wound after gentle approximation of the edges.

Direction is indicated by the track of the wound but evidence of undercutting, if present, will also give an idea about its direction. The track can be well-demonstrable when it has passed through a solid organ like liver or hard tissues like bones. Care must be exercised in interpreting the circumstances from the direction of the wound. Factors like lateral deviations of the weapon, left or right handedness of the victim as well as the assailant, the stature of the individual and, above all, the constantly

changing postures of the victim and the assailant (as is often expected during the stabbing incidence) need to be evaluated carefully.

Probing of the stab wound is not advisable as it may lead to fatal haemorrhage by dislodging some clot or creating false passages or injuring some organ. The depth should be determined during operation or during the postmortem examination by dissecting the track of the wound in layers. In the living, injection of dyes or radiopaque material into the wounds to enable the demonstration of the wound track by X-rays has been attempted and claimed to be advantageous by some.

The operating surgeon should not disturb the surface wound and should simply stitch the same while taking other necessary measures so that it can be evaluated in its proper perspective by the autopsy surgeon in the unfortunate event of death of the victim.

Margins

When a sharp cutting weapon has been thrust, the margins will be clean cut and without any bruising. Margins of the wound are inverted. The exit wound, if present, will show everted margins. The margins of the wound of entry can be everted when the wound is situated over the fatty area such as protuberant abdomen or the gluteal region.

Abrasion or bruising of the margins of a stab wound suggests that the blade had been completely inserted. In such cases, the suspected weapon, if available, must be examined to observe the compatibility of the shape of abrasions/contusions around the wound with the handle of the weapon in question. In this context, it may be kept in mind that unless the blade penetrates the body at right angle, all sides of the hilt may not be equally imprinted upon the skin. Therefore, depending upon whether the weapon has been inflicted in a downward or upward direction, the impressions of the hilt will be conveyed accordingly.

Shape (Configuration)

Although ellipse is the most common shape, there is a wide range of shapes of these wounds. Strangely shaped surface as well as the visceral wounds may be observed such as 'A' or 'V' or 'Λ' or some may be square or diamond shaped and still others like cruciate, stellate, etc. **The size, shape and configuration of these wounds are influenced by a number of endogenous and exogenous factors as under:**

Factors Attributable to the Weapon

The external appearance of stab wound does not depend solely upon the cross-sectional configuration of the weapon, as already described; it is influenced by the following also:

- The site of injury and the plane in which the weapon struck the target.
- Whether the weapon is single-edged or double-edged, etc.

To discuss these one by one, there is no denying the fact that the **site of the wound in relation to tissue planes** may have a modifying effect on its pattern. Traction of the tissues may cause the wound to appear to have been caused by a blade having a thick back, when it was in fact a thin sharp blade that would have produced an elliptical wound elsewhere. Further, the elastic tissue of the dermis, the deeper layer of the skin, has a considerable bearing upon the shape of the wound. It should be realised that the dermal collagen and elastic fibres are arranged in a definite pattern and are flowing in all areas of the body. The pattern of fibre arrangement is called the lines of cleavage of the skin, and their linear representations on the skin are generally known as **'Langer's lines'**. These cleavage lines correspond to the creases of the body surface. Surgeons do take into consideration the orientation of these lines because an incision parallel to these lines heal with a fine linear scar, whereas an incision across these lines may result in an ugly looking scar. A stab wound with long axis at right angles to the cleavage lines of Langer will gape open with the edges pulled apart, whereas one that runs parallel to these lines will appear slit like or wedge shaped and will represent with fair degree of accuracy the dimensions of the blade with which it was produced. If the weapon is inserted in an oblique fashion, the skin gap may be of the dimensions intermediate of those described earlier. In some instances where the skin wounds are gaping widely, injuries in the underlying viscera like heart, liver, etc. may be slit like. The converse may also be true, i.e. skin wound on the anterior thorax may be narrow slit if its long axis run parallel to the lines of Langer, but the defect in the underlying intercostal musculature may gape widely due to the retractive effect of the muscular fibres.

In context with the **single-edged or double-edged blade**, the opinion is divided—whether or not this instrumental characteristic can be ascertained with accuracy depending upon the configuration of injuries. Most observers feel that skin wounds have two well-defined marginated acute angles even when produced by a single-edged weapon. However, a skin stab wound, which closely parallels the cleavage lines, may exhibit one well-defined and another blunt extremity corresponding to the sharp and dull edges of a single-edged weapon. To be more critical, the blade of the weapon may be double-edged along its entire length or it may be double-edged for a variable distance or it may have one cutting edge.

One explanation for the presence of bilateral acute angles in the wounds inflicted by a single-edged weapon is that the initial penetration by the extreme tip of the blade of the weapon creates a dermal defect with acute angles at each extremity, and the cutting edge of the blade continues to operate while the opposite extremity of the wound which is in contact with the blunt side of the blade of the weapon remains sharply angulated as was created initially as the dull side of the instrument hardly receives any opportunity to play its role. Exceptions may arise depending upon the manner in which the weapon is being thrust, i.e. whether the cutting edge is moving forward or the

blade is being thrust quite perpendicularly to the body surface extending due consideration to the direction of the cleavage lines of Langer as stressed earlier.

Factors Attributable to Victim and the Assailant

During the process of stabbing, there are often relative movements between the victim and the assailant. If the victim twists or turns the target portion of the body either during the in-stroke or during the out-stroke, the external wound may get irregularly distorted. Moreover, the weapon may be manipulated by the assailant in different ways. Countless possibilities exist, all capable of producing bizarre or atypical wounds. **To cite a few examples:**

- The instrument is thrust in, partially out and in again along a different track. Here the external wound may be of compound nature and more than one track may be seen in deeper tissues and organs.
- The instrument is thrust in and is withdrawn with the cutting edge dragging against one extremity of the wound so as to extend the wound superficially.
- The instrument is thrust in and then, while still in, is pulled in the direction of the cutting edge so that the track and the skin wound both get enlarged.
- The instrument may be twisted during in-stroke or during withdrawal or during both. The margins of the external wound will be irregular and varying in dimensions.

Amount of Force Required to Inflict Any Particular Stab Wound

This question may be raised in some fatal stabbings. This query cannot be answered absolutely but only in comparative terms as it is a subjective phenomenon. The medical witness may use the terms 'slight', 'moderate' or 'considerable force'. For example, it is reasonable to conclude that considerably less force is required to inflict a deep stab wound with a fresh surgical scalpel blade than is needed to produce same wound with a dull knife of equal size and similar shape. Experiments conducted by Bernard Knight and many other investigators in the animals, cadavers and in the surrogate tissues, suggest the following useful generalisations:

- The amount of force required to produce a particular wound varies inversely with the sharpness of the extreme tip of the knife and the keenness of the edge of the weapon (Fig. 15.2).
- The general character of the blade of the weapon also matters. A thin, slender double-edged knife will penetrate more deeply than an equally sharp, wide single-edged blade inserted with the same force.
- Toughness of the target area/tissue also matters. Apart from bone and calcified cartilage, the tissue offering most resistance is the skin. Once the resistance of the skin has been overcome, the knife slides through the underlying tissues with out much effort.
- Another important factor is the velocity of knife at the time of impact, as fast moving knife would penetrate the tissues with greater ease than one exercising slow steady pressure.
- Victim may contribute to the force involved in the fatal thrust by running into or hurling himself on to the blade.
- The stretched skin is easier to penetrate than the lax skin. The chest wall, where the skin is stretched over intercostal spaces, is easier to penetrate.

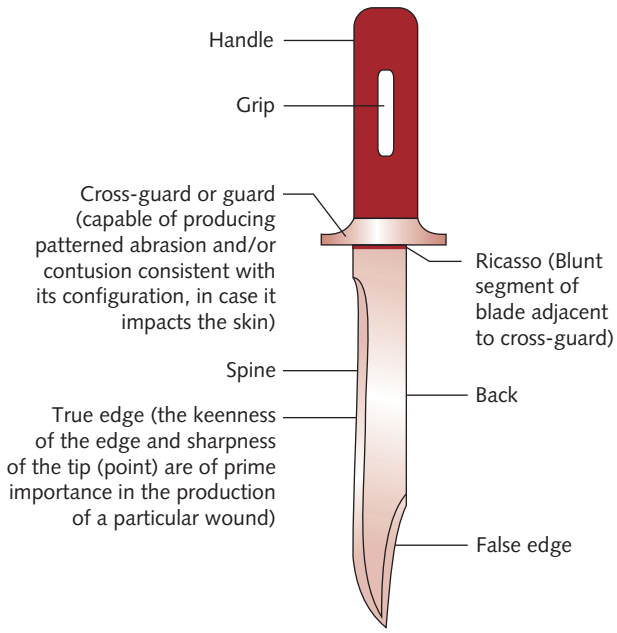


Fig. 15.2 Parts of a knife and the areas primarily influencing production of a particular wound.

WOUNDS BY BLUNT PENETRATING/DULL INSTRUMENTS

Penetrating wounds inflicted with comparatively dull instruments like scissors, screwdriver, poker, chisel, etc. frequently demonstrate lacerated marginal cuticular wound (Fig. 15.3). The skin is split as well as penetrated, and there may be marginal bruising too. The characteristic feature of wounds produced by open scissors is a pair of wounds with one wound above and somewhat oblique to the other. The distance between the two wounds differs with the angle of penetration.

WOUNDS BY GLASS

Wounds produced by glass can pose difficulty in the interpretation in absence of information concerning the circumstances. Basically, they are all lacerated wounds but if spicule of glass enters by its point, the wound is then stab-like in appearance. When applied tangentially or at a small angle to the skin, undercutting may be

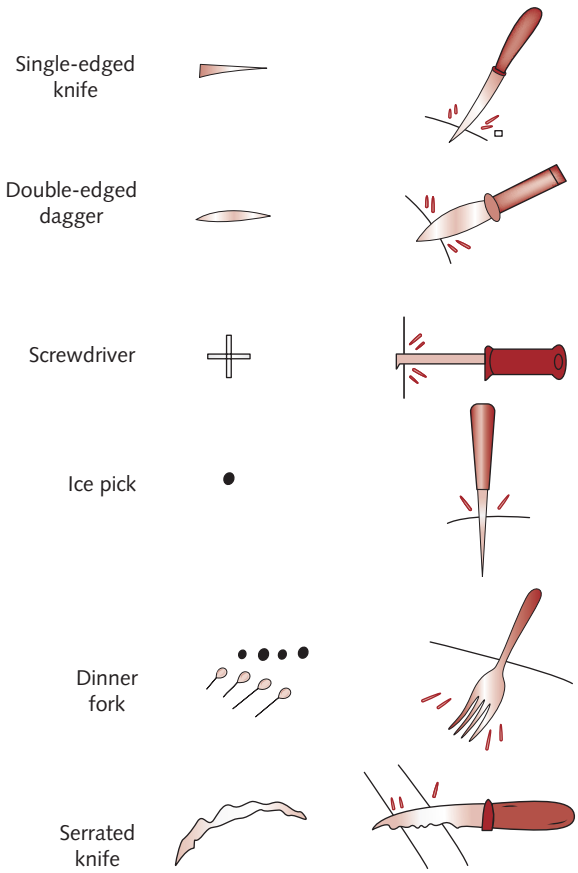


Fig. 15.3 Skin wounds produced by different weapons carrying blunt/sharp pointed blade.

a marked feature. Though the glass may be used for slashing, long slivers of glass can act as stabbing agents. The sliver may break off and remain in the wound. Side cuts are also likely to be seen and, when present, are characteristic of wound by glass. Search should be made for flakes or particles of glass in the wound.

CONCEALED/OBSCURE PUNCTURE WOUNDS

Rarely, the site of puncture wound may be relatively inconspicuous. Puncture wounds in hairy areas (scalp and/or pubes) can be located by visual and palpable search. Among other sites not anticipated as the points of occurrence of puncture wounds are the ears, nostrils, medial canthus of eye, fontanelles in the newborn, nape of the neck, axilla, underfold of female breast, vagina, rectum, etc. Extreme difficulty may be faced in locating wounds at such sites. The difficulty may get compounded because of the surface of the body being smeared with blood. The agent/instrument involved in such cases may be small in cross-section, like knitting needle, safety pin, etc. At times, pointed instrument may enter the peritoneal cavity through vagina during attempts for procuring abortion. Hendry and Stalker reported a good example where the agent was an unusual one, namely, part of an aluminium 'tail' comb.

ACCIDENT, SUICIDE OR HOMICIDE

Though the stab wounds are usually homicidal in origin, yet the issue of suicide/accident can creep up under certain circumstances. Following points may be helpful:

- Multiple stab injuries scattered over various parts of the body, accessible or inaccessible, covered or uncovered, indicate homicide. Although homicide may be accomplished by a single stab (Fig. 15.5), it is more usual to find many. It may be on these occasions that even after a lethal stab has been given, the assailant continues to stab or returns later to resume the activity, the so-called **'overkill homicide'**.
- Thrusts by homicide are all likely to penetrate deeper, and more than one may be lethal. There may be a few outlying strokes beyond the target area. On the other hand, a suicide is likely to confine his thrusts to a smaller and restricted area, and only one or about a couple of them is likely to be deeper and lethal.
- At occasions, interpretation of a single stab may pose difficulty as the usual defence against the homicidal stabbing is to allege that the deceased 'ran on to the knife'. In this context, the most important consideration is the firmness with which the weapon was held. It may be argued that the impetus of the moving body is more than enough to impale it on suitably pointed object. However, there may be occasions where some stab injuries result from the combined movements of a stab thrust and an approaching or falling victim. Direction of the track within the body may lend some help in resolving the issue, viz.,
  - a downward track might be in keeping with impalement during a head-down eventuality of the victim;
  - an upward track may suggest falling of the victim or jumping down into the fray from a height; or
  - a horizontal track may suggest turning of the victim into an unseen weapon.

  Factors like firmness of grasp of the weapon, nature of the weapon especially of its tip (blunt point will glance off the skin more readily than will a sharp one) and other subjective plus extraneous factors need appreciation in such a context.
- Suicidal injuries are commonly situated over front of body on easily accessible sites, especially over throat, precordium, abdomen or wrists and rarely found on unusual locations as cannot easily be reached by the victim.
- A couple of characteristic features of suicidal stabbing include the following: **Firstly,** presence of 'tentative wounds' that are superficial and unlikely to penetrate beyond muscular layer. The finding of **tentative wounds (hesitation cuts)** is a useful observation in helping differentiate suicide from homicide. Indeed, 'Hesitation marks' can be considered the 'trade marks' of suicide. The name is derived from the fact that cutting/stabbing oneself is painful and the would be suicide frequently makes several half-hearted, superficial cuts/stabs before he/she gathers sufficient courage to unleash the



Fig. 15.4 Multiple stab wounds on the abdomen. The case is of a suicide committed by a man after altercation with his wife over the disposing off the stereo system. Though there were 15 wounds in total, only a couple of them were of fatal character. Interestingly, blood-stained T-shirt (devoid of any cut/rent/defect, etc.) along with other things was lying on the side of the body (probably, the deceased had inflicted the injuries after removing the T-shirt—a phenomenon suggestive of suicide).

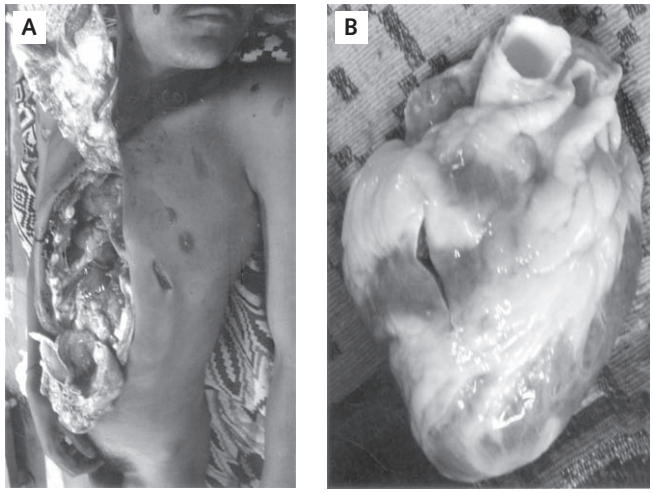


Fig. 15.5 Photograph **(A)** showing obliquely placed stab wound on left side of chest below the nipple. Photograph **(B)** showing involvement of right ventricle of the heart. Disparity between the surface wound and the ventricular wound highlights the fact that architecture of the tissue and manner of infliction are of significance in imparting ultimate configuration to the wound.

sufficiently forceful stroke, which is able to cause fatal damage. Another basis for hesitation marks may be the subject's ignorance as to how tough and resistant the tissues are and how much force is needed to produce the fatal injury with the weapon at hand. **Secondly,** suicides almost always inflict wounds over the uncovered parts of the body. Thus, wounds over the parts of the body that ordinarily remain covered by clothing, without corresponding cuts/rents upon them, are indicative of suicide. This observation is again a potentially useful factor in differentiating suicide from homicide because a homicidally inclined assailant does not ordinarily take time or trouble to expose the site of injury (Fig. 15.4).

- **Hara-kiri:** It is an unusual type of suicide, usually seen in Japan, in which the victim inflicts a single large incised penetrating wound over the abdomen with a short sword or falls upon the ceremonial sword and pulls out the intestines. The sudden evisceration results in sudden decrease in intra-abdominal pressure and cardiac return, producing sudden cardiac collapse.
- **Defence wounds,** like cuts and/or lacerations/bruises, etc. on or in-between the fingers and palms, back of hands, wrists, inner aspects of forearms or any other part of the body, if present, are strongly suggestive of homicide as these are produced during attempts by the injured to seize the weapon in instinctively defending himself or in an endeavour to ward off the attack on the head or some other vital part of the body.
- **Accidental stabbing/puncturing** is an unusual event but may be encountered in case of accidental fall over the projected ends of railings, spikes of garden walls or house walls or from falling against broken glass/earthenware fragments, which may often get embedded in the deeper parts of the wound. Difficulty may arise in cases where the injury is received during a fight, and the defence counsel tries to take shelter in arguing it to be of accidental origin. Here, factors like presence or absence of defence wounds, the angle, situation and direction of the wound, the condition of area adjoining the surface wound, detailed examination of the clothing and the circumstances of the injury will be helpful in solving the problem.

16 CHAPTER

Injuries by Firearms

After going through this chapter, the reader will be able to describe: Types of firearms | Types of ammunition | Parts of cartridge and their functions | Mechanism of bullet wound production | Characters of wounds produced by rifled and smoothbore firearms | Exit wounds by rifled and smoothbore weapons | Direction of fire | Unusual circumstances in firearm injuries | Various tests for firearm residues | Autopsy in firearm fatalities | Suicide, accident or homicide

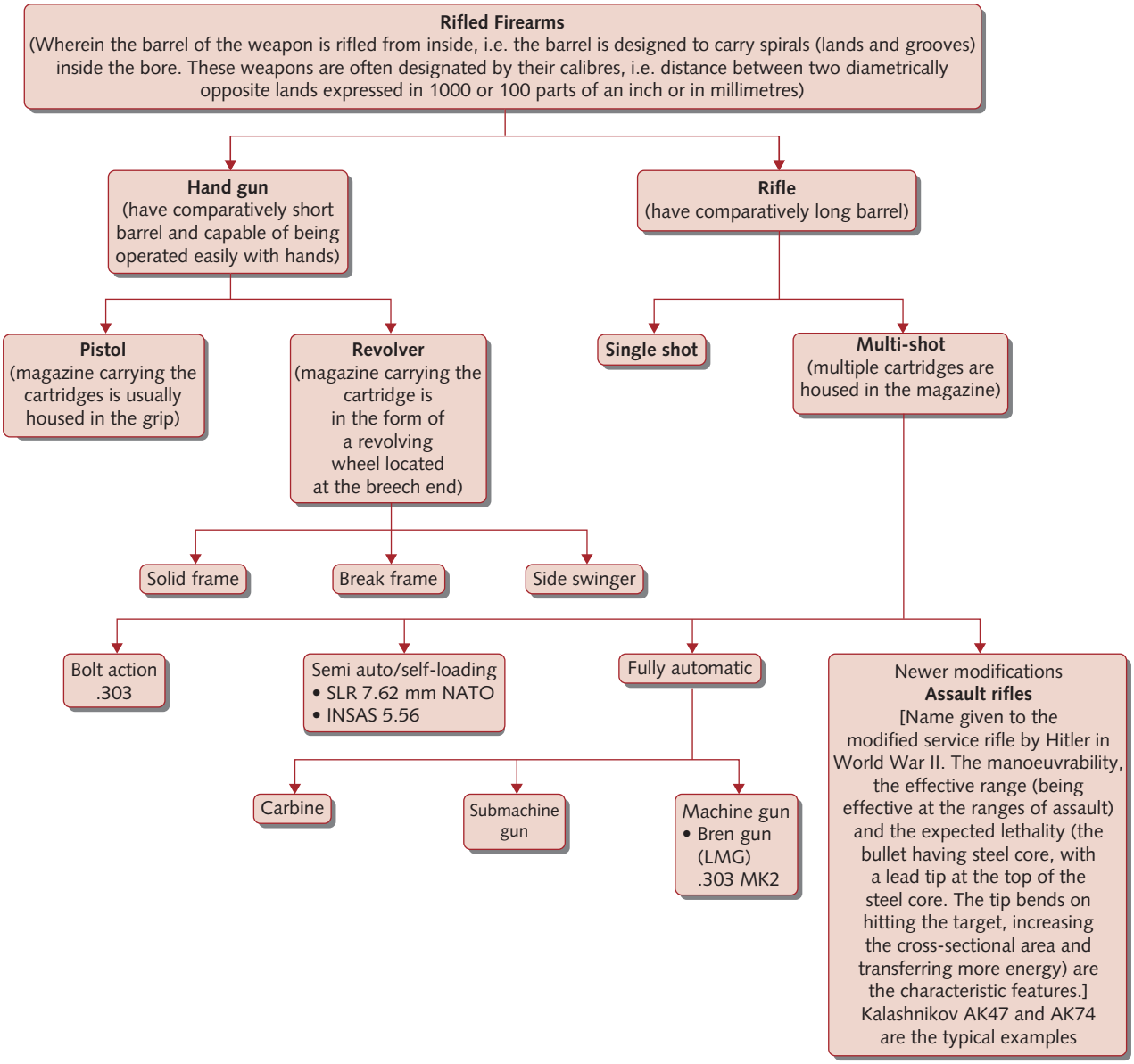
A **firearm** is any instrument or device designed to propel a projectile by means of expansive force of gases generated by combustion of an explosive substance. **Forensic ballistics** may be considered as the science dealing with investigations of firearms, ammunition and the problems attending their use. For a doctor, elementary knowledge of structure of a firearm, composition of ammunitions and mechanism of discharge of a firearm is necessary for proper understanding and interpretation of the injuries produced by them. However, at the very outset, I must convey that the **medical experts are not ballistic experts** and, therefore, should confine themselves while giving evidence in the court to the interpretation of injuries upon the body and that too in broad generalisation. It is for the ballistic expert to opine about the precise range of fire, the nature of weapon involved in the crime, the nature of ammunition, etc.

Types of Firearms

RIFLED FIREARMS

These weapons discharge a single projectile or missile through a 'rifled' bore and hence the name. **Rifling** means the inner surface of bore of the weapon from breech to the muzzle end is thrown into spiral grooves, varying from 2 to 22 or more (usually 4–7), which run parallel to each other but are twisted spirally. These **'grooves'** are called 'rifling' and the projecting ridges between these grooves are called **'lands'**. Riflings vary in number, direction, depth and width. The purpose of rifling is to impart rotational spin to the bullet along its long axis. The gyroscopic effect stabilises the flight of the bullet through the air, preventing it from tumbling end over end. This spin does not, however, stabilise the bullet after it enters the body due to great density of the tissues as compared to the air. When a bullet is fired down a rifled barrel, the rifling imparts a number of markings to the bullet that are called **'class characteristics'**. These markings may indicate the make and model of the gun from which the bullet has been fired. They result from the specifications of the rifling, as laid down by the individual manufacturer. These characteristics include (*i*) number of lands and grooves, (*ii*) diameter of lands and grooves, (*iii*) width of lands and grooves, (*iv*) depth of grooves, (*v*) direction of rifling twist and (*vi*) degree of twist. In addition to these class characteristics, imperfections on the surfaces of the lands and grooves score the bullets, producing **'individual characteristics'**. For lead bullets, these individual characteristics are more pronounced where the grooves score the bullet. In contrast, for jacketed bullets, the land markings are the most pronounced. These individual characteristics are peculiar to the particular firearm that fired the bullet and not to any other. They are as individual as fingerprints. No two barrels, even those made consecutively by the same tools, will produce the same markings on a bullet. Different types of rifled firearms have been described in Flowchart 16.1.

Calibre of a rifled firearm is measured between a pair of diametrically opposite 'lands' across the bore. In the United States and all English-speaking countries, the diameter of the bore of rifled small arms is designated in hundredths or thousandths of an inch. Thus, we have rifles, carbines, pistols and revolvers of .22, .30, .32, .38 and .45 calibre or .220, .257, .357 and .405 calibre. The European system of cartridge designation is more thorough and logical than the US system. It uses the metric system. For example, the Russian rimmed-service round is designated as $7.62 \times 54\,mm^2$ R, i.e. 7.62 refers to the diameter of the bullet, 54 mm indicates the length of the cartridge case and R indicates that the round is rimmed. The letters SR are used for semi-rimmed cases, RB for rebated cartridge cases and B for belted cases. No letter is used to describe rimless



Flowchart 16.1 Categorisation of rifled firearms.

cartridge cases. The term 'magnum' is used to describe a cartridge that is larger, carries more propellant and produces higher velocity than the standard cartridges.

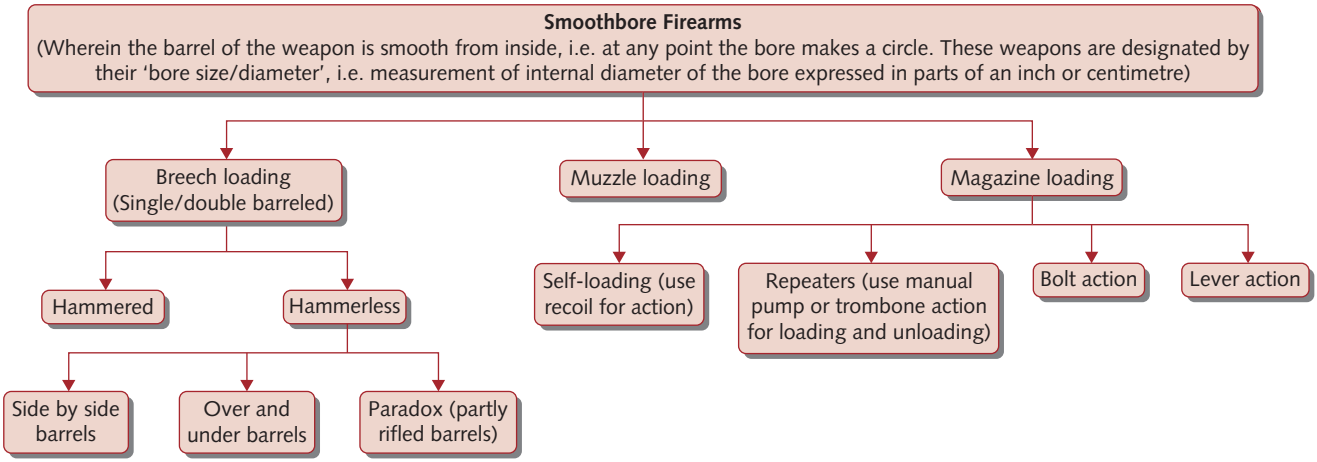
SMOOTHBORE FIREARMS

These belong to the category of shoulder-arm, having smoothbore barrel and are intended for firing lead shots (pellets) or sometimes a single ball (slug), etc. Some shotguns having small part of their bore rifled near the muzzle end are known as **paradox guns**. Different types of smoothbore firearms have been described in Flowchart 16.2.

Bore/Diameter

Diameter of the barrel of a shotgun can be expressed in any of the three systems. In the United States, the most commonly employed system is that of **gauge**. The origin of this system is archaic. It refers to the number of lead balls, each fitting the bore, which can be made from 1 pound of lead. Thus, a 12-gauge shotgun has a bore diameter such that 12 balls of lead, each fitting the bore, can be made from a pound of lead. The smaller the gauge designation, the larger the bore. **The second system** is the expression of bore diameter in inches. The modern .410 bore shotgun is the only shotgun to be so designated. **The third is the metric system,** where the bore diameter is expressed in millimetres. A 10-mm shotgun has a bore diameter of 10 mm.

Choking is a device employed in smoothbore firearms where the terminal few centimetres of the bore near the muzzle end is partially constricted so as to control shot pattern. This device, therefore, holds the shot column together for a better distance as it moves away from the muzzle. The choke may be



Flowchart 16.2 Categorisation of smoothbore firearms.

permanent and built into the barrel or the barrel may accept choke tubes of different degrees. The degree of choke is based upon the percentage of pellets that will stay inside a 30 inch circle at 40 yards as given in Table 16.1.

MISCELLANEOUS TYPES

Air-Powered Weapons

In these weapons, expanding force of compressed air or gas is used to propel a projectile down a rifled or smoothbore barrel. Pellets/projectiles, because of their extremely light weight, loose velocity rapidly, becoming harmless in less than 100 yards. Weapons include air rifle, air pistol, air gun, etc. These are used for target shooting, sport activities, firearm training, etc. (Austrian armies used air rifles against the French during the Napoleonic wars from 1799 to 1809).

Cattle Guns/Humane Guns (Captive Bolt Devices)

Specially made firearms used to kill animals. Discharge of blank cartridge drives a captive bolt, 5 to 10 cm in length, out of the muzzle. The end of the bolt is usually circular (7–12 mm diameter). It enters into the head of the animal against which the muzzle is kept pressed. In Western countries, butchers and farmers used them to slaughter animals.

Stud Guns

These are industrial tools wherein special blank cartridges are used to push metal nails or studs into the wood, concrete, or steel (accidental deaths have been reported after the nails or studs have either perforated walls or ricocheted off a hard surface, striking and killing workers).

Bang Sticks

A device used by skin divers and fishermen to kill sharks, large fish or alligators. Also called 'fish popper'/shark stick. Bang sticks may be acquired in various calibres including centrefire handgun. A number of suicides have been reported using bang sticks.

Table 16.1 Dependence of Choking on Pellets

Choke	Percentage of pellets falling in 30-inch circle at 40 yards	
Full choke	65–75	In determining the spread of shot pattern, one must exclude fliers, i.e. pellets deformed in the bore that stray from the main pattern
Modified choke	45–55	
Improved cylinder	35–45	
Cylinder	25–35	

Improvised/Country-Made Firearms

Called by different names, these are somewhat similar to 'zip guns'; the term 'zip gun' usually indicates either a crude home-made firearm or a conversion of a blank pistol to a firearm. The calibre, shape and size of the firearm depends upon the availability of the ammunition, the barrel tube and the skill of the blacksmith (even household plumbing pipes have been used for the barrel in some cases). Consequently, the range, the wounding power and the reliability vary so much that no generalisations about the nature of the effect can be made safely.

Ammunition

The ammunition for the various types of firearms is spoken of in several different ways. The word 'ammunition' means any unfired assembly of primer, powder and ball, but today the word is generally used when referring to a supply of assembled cartridges in bulk. The term 'round' refers to a single cartridge. With the coming up of wrapped powder-ball assembly, the

word 'cartridge' came into use. The 'bullet' is only the solid portion of the loaded rifled weapon. The other common designations for the bullet are projectile, missile and ball, etc. The term 'ball' is a relic of old muzzle-loading days when all the projectiles were round lead balls.

In case of a smoothbore firearm, the mass of small round lead projectiles is referred to as 'shot' or 'charge' or the 'load', while the individual round projectile is termed as 'pellet'. Although the use of round single lead ball in shotguns has been restricted, but is still peculiar to a degree in some countries, where such loads are often locally referred to as 'punkins' or 'punkin balls'.

CARTRIDGE

The cartridge, as stated earlier, denotes an assembled complete round of ammunition comprising (*i*) cartridge case; (*ii*) percussion cap, containing primer; (*iii*) powder or propellant charge; (*iv*) projectile (which may be a bullet in case of rifled weapons and pellets in case of smoothbore weapons); and (*v*) wads (only in case of smoothbore weapons) (Fig. 16.1A and B).

Cartridge Case

It is the outer covering of the cartridge that contains and keeps the inner components in position, prevents the backward escape of gases and also provides protection to the contents. After firing, empty casing is left behind, which is called empty shell.

In case of rifled weapons, this casing is composed of metallic case, usually of brass. Although brass is the traditional metal, steel is also used. However, steel must be coated to prevent it from rusting. Plastic varnish is the most usual method today. Brass is preferred for commercial ammunition and steel is almost entirely confined to military ammunition. The cartridge cases are classified into five types according to the configuration of their bases, namely: rimmed, semi-rimmed, rimless, rebated and belted. Virtually all cartridge cases have head stamps on their bases. The head stamp consists of a series of letters, numbers, symbols and/or trade names. They are either imprinted or embossed on the cartridge case-head and help in identification.

Caseless cartridge is one in which the conventional metal case is not employed. Here, the propellant is mixed with a binder to make it a hard paste, which can be shaped as required. A cap, made up of combustible material, is fitted into the base and a bullet is recessed into the other end. The object is to eliminate the extraction and ejection of the empty case after firing and therefore to speed up the action of the weapon. It also has the advantage of lightening the round (brass or steel cases are heavy). However, the disadvantage is that it does not seal the breech and it does not provide an insulating 'heat sink' between the propellant and the hot chamber of the gun. Germans developed 4.7 mm G11 Hecker and Koch rifle and the $4.73 \times 33\ mm^2$ DM 11 caseless cartridge. The first production weapons were probably issued to German Special Forces in 1990. The end of Cold War, the unification of Germany, defence cuts and the availability of a large number of arms at cheaper rates has apparently led to the demise of such cartridges.

In cartridge case of a shotgun, the longer anterior part is made up of cardboard or plastic and the posterior part is composed of brass. Usually this posterior part (base) is rimmed to position the cartridge correctly in the chamber and to help its extraction after firing. The base has the percussion cap at its centre.

Percussion Cap

It is formed of either zinc or copper or a compound of both, so as to be malleable and easily deformed under the blow of the firing pin. Beneath the metal is a layer of sensitive cap-composition, which is sealed in place by a layer of varnish that also waterproofs the composition. The impact of firing pin nips the composition against anvil and the resulting flash travels into the body of the cartridge case and ignites the propellant charge. Presently, **the primers are composed of** lead styphnate, barium nitrate and antimony sulphide. Most centrefire primers are composed of all the three compounds. The detection of these compounds constitutes the basis for the tests to determine whether an individual has fired a firearm. In case of rimfire ammunition, the primer composition is spun into the rim of the cartridge case with the propellant in intimate contact with this composition. On firing, the firing pin strikes the rim of the cartridge case, compressing the primer composition and initiating its detonation.

Propellant Charge

The principal requirement of propellant is that it should explode rapidly generating a mass of gas, but it should not detonate because this would damage the weapon. It may be of following types:

Black Powder (Gunpowder)

The black powder or the gunpowder was the first propellant and remained the only one until the discovery of smokeless powder in the later half of the nineteenth century. Average composition of black powder is:

Potassium nitrate (saltpeter)	75%	Charcoal is the fuel, potassium nitrate is the oxygen supplier, while sulphur gives the mixture more density and makes it more readily ignitable
Charcoal	15%	
Sulphur	10%	

The speed of burning of black powder is regulated by the size of granulation. As the size of the granules decreases, the strength of the powder increases. However, when a powder is made very fine like dust, the speed of burning is reduced as all the spaces between the grains are filled up and there is no way

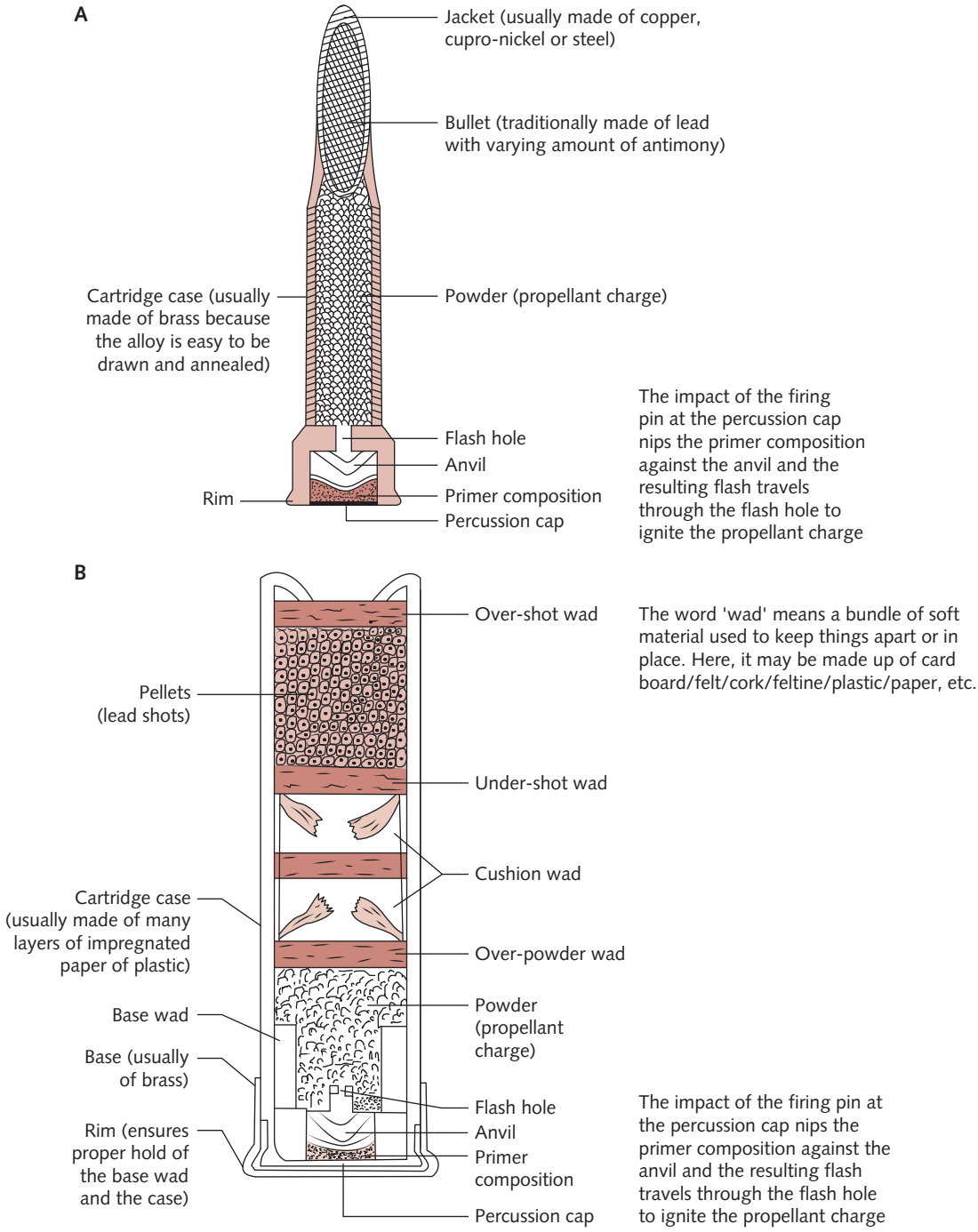


Fig. 16.1 Structure of cartridge: **(A)** small arms cartridge, **(B)** shotgun cartridge.

for the flame to communicate and ignite the whole charge rapidly. Black powder is made and sold in the form of irregular shiny metallic-looking black grains designated as to the size by the letters FG, FFG, FFFG, etc. The more the number of Fs, the finer are the grains. One gram of powder produces 3000–4500 cc of gas.

Smokeless Powder

Smokeless propellants come in a variety of shapes and sizes. Most pistol propellants are in the form of thin flakes; rifle propellants may be in short cylindrical or tubular form or may be composed of small spheroids, known as 'ball powder'. The shape and size has a bearing on the rate of burning. Simple shapes like flakes, cylindrical grains or balls, etc. burn with a gradually decreasing surface. A tubular grain, burning inside and outside, has a burning surface that remains almost same since the decreasing external surface is almost balanced by the increasing internal surface and thus can develop a near constant pressure. One gram of powder produces 12,000–13,000 cc of gas. It may be of following types:

- **Single base powder:** The term used to describe propellant powders that are made from nitrocellulose with the addition of small amounts of chemicals to promote chemical stability. It is probably the most common type of commercial powder because of its simplicity, adequate power, good keeping properties and a low flame temperature that does not cause excessive erosion in the barrel of the weapon. However, single base powder is more susceptible to damp and therefore needs to be adequately protected during storage.
- **Double base powder:** In this, the principal constituents are nitrocellulose and nitroglycerine. It is more powerful than single base because of the presence of nitroglycerine but for the same reason, it is also much hotter and has a flame temperature that melts away the steel of the barrel of the weapon more rapidly. Double base propellants are no longer used these days having been replaced by triple base propellants.
- **Triple base powder:** This is a type of propellant in which three principal ingredients are used nitrocellulose, nitroglycerine and nitroguanidine. It was devised in an attempt to compromise between the low power of single base powders and the high power (but excessive heat) of the double base powders. Therefore, the quantity of nitroglycerine is small but sufficient to give added power; the nitroguanidine lowers the flame temperature while still adding an active explosive constituent.

Projectile

Rifled Weapons

In case of rifled weapons, there is a single projectile or the bullet. **The traditional bullet** is made up of soft metal and has a rounded nose. The metal is lead with varying amount of antimony added to provide hardness. This missile (bullet) is generally known as round-nosed soft bullet and is frequently used in small arms. Rifle bullets are usually ogival and may be streamlined. There are some variations in the size and shape of the bullets:

- It may be square-nosed soft metal bullet known as 'wad cutter'.
- The second may be hollow point, which has a depression in the nose of the soft metal. This bullet is designed to expand or 'mushroom' upon impact.

All these soft metal bullets cause 'leading' of the bore of the firearm. This wiping of lead on to the bore causes a decrease in the accuracy of the firearm; to overcome this, the bullet may be lubricated. The lubricant may be applied over the entire surface except the base or may be applied in the small grooves or cannelures cut circumferentially into the bullet near the base. The second way to overcome this shedding of lead from the surface of the soft metal bullet is to cover the bullet with a jacket. **The jacketed bullets are of two types:**

- Full metal jacket bullet in which tough heavy jacket covers all except the base where the soft metal interior is exposed. Such bullets were designed for military purposes. The tough jacket may be made up of steel, copper, nickel and zinc.
- Semi-jacketed bullets are provided with relatively thin but tough jacket, covering the base and the cylindrical portion of the bullet, leaving the nose partially or fully exposed. This type of bullet was designed to expand or 'mushroom' like the soft metal hollow-point type.

Having considered the basic types, some specific types need to be discussed, which are as follows.

Dum Dum Bullets

A type of bullet developed at Dum Dum Arsenal in India in 1890s by Captain Bertie Clay. They were first used at the battle in 1898, but displayed a serious defect in that because the jacket did not cover the base, there was tendency for the core to blow and leave the jacket in the rifling of the barrel of the weapon that hindered the loading of the next round. The design was therefore neglected and replaced by the 'Ball Mark III' bullet that had a full jacket with a hole bored in the nose and filled with a short metal tube. In 1899, the Hague Convention outlawed the use of any expanding bullets in military service and the Dum Dum bullet and Ball Mark III were abandoned.

Explosive Bullets

These bullets, apart from causing extensive damage to the victim, pose considerable potential danger to the surgeon and doctor conducting autopsy, because the bullet may explode during emergency surgery or might detonate during diagnostic techniques involving ultrasonography, if it had failed to detonate in the body. In the assassination attempt on the President, Mr. Ronald Reagan, who had been shot with an exploding bullet that failed to detonate, though some of the lead azide from the charge had spilled into the surrounding tissue of the lung and was removed surgically. Such bullets are modified by drilling out bullet tip and inserting a tiny canister containing lead azide charge and the aim is to impart the missile greater stopping power so as to enable the bullet to transfer massive kinetic energy to the tissues. In suspected cases involving such ammunition, the autopsy surgeons wear goggles and use long-handled instruments to manipulate the missile during autopsy. The recovered bullet should be kept in a padded container to protect it from extra impact, vibration and heat, etc. The Hague Convention of 1899 forbade the use of all such bullets and they fell into disuse.

Frangible Balls

A type of ball bullet made from compressed particles of metal and paint. Used in US Army and Air Force as a training bullet for aerial gunners. When fired at an aircraft, the bullet will get disintegrated to dust like particles on impact, causing no damage but leaving a paint mark so that the trainee's gunnery could be evaluated.

Baton Round

Popularly known as 'rubber bullet'. It is riot control projectile, usually a cylinder of rubber or plastic of a size to suit 12-bore, 26-mm or 37-mm gun and fired by a low powered charge to attain a muzzle velocity of about 60 metres per second and a range of about 100 metres. The first baton rounds were developed for Hong Kong police and were of wood but proved to be liable to splinter on impact. It was therefore replaced by rubber batons. Rubber batons, however, were found to bounce indiscriminately and were superseded by PVC type of plastic batons that are more predictable in their behaviour.

Armour-Piercing Bullet

It is a type of military bullet designed to penetrate light steel armour. It is formed of a hard steel core surrounded by a lead sleeve, both carried in the usual type of jacket. On impact, the lead sleeve and the jacket are arrested, while the piercing core continues to penetrate through the target. Today, it is mainly used against light armoured vehicles.

Incendiary Bullets

A type of army bullet used to cause fire in the target. Usually confined to use in the aircraft armament in order to ignite fuel tanks. Commonly, it is in the form of a jacketed bullet with the front half of the core removed and the space filled with white phosphorus, which possesses property of ignition on coming in contact with air. During 1939–1945, incendiary composition of barium nitrate and powdered aluminium and magnesium were developed because phosphorus was disliked due to its tendency towards leakage and causing fires in ammunition dumps.

Tracer Bullet

This type of bullet leaves a visible mark or 'trace' while in flight so that gunner can observe the strike of the shot. They resemble ball but have the rear portion of the core removed and the space filled with a chemical compound, a mixture of barium nitrate and powdered magnesium with strontium nitrate added to give it a red colouring. The mixture is ignited by the flash of the propellant and burns during the flight of the bullet shedding red sparks.

Smoothbore Weapons

There are usually multiple projectiles in the form of spherical pellets, called the 'shot'. The shot consists of up to several hundred small lead pellets, the number depending upon the size of the individual pellet. Three general classes of shots are used in the shotguns—bird shot, buck shot and individual projectiles (usually termed as rifled slugs) with the frequency of use in the given order:

Bird shot is generally used for hunting fowl and small animals. The shots are small ranging in diameter from 1 to 3.5 mm. A 12-bore shotgun shell will contain 200–400 shots depending upon their size.

Buck shot is larger than bird shot being 6–8 mm in diameter, and in a 12-bore shotgun they are nine in number. It derives its name from its original use against large game such as deer. Terminology differs according to the country of origin.

Rifled slug is lead or steel and lead projectile for a shotgun with wing-like helical ribs on its outer surface that, due to the passage of air during flight, give it a rotational movement and so produce a spinning projectile from a smoothbore weapon. The foster type of rifled slug is usually used in the United States. The rifled slug is intended for large games such as deer. A number of devices are available. One of the older ones is solid round 'pumpkin ball' or the 'Brenneke rifled slug', but more recent types include the Foster rifled slug and the French Blondeau, which is dumb-bell shaped.

Wad

It is present only in cartridge of smoothbore weapons. The wad is made of some soft material like felt, cork, plastic, straw or rug. The cardboard disc behind the shot charge prevents the pellets from getting lodged in the felt wad, separates the propellant from the projectiles, seals the bore effectively and prevents the escape of gas from the breech end. Some wads are disc-shaped, others cup-shaped and still others having bizarre shapes. Certain modern modifications that may be seen in the imported weapons include the increased use of plastic especially to replace traditional wads. Some of these may be **'power piston'** where the shot-mass is contained inside the polythene cup, which may also be responsible for producing injuries. In some modern cartridges, plastic granules may be used as filler between the lead pellets, and this highly coloured material may also be found within the wound or upon the skin. Deliberate tampering with the cartridge contents may also include removing the top closure card and fusing the shot with melted paraffin wax or even black pitch in order to prevent dispersion. This may be called as **'balling or welding of shot'**. The balling of shot may also result from faulty manufacture or deterioration of old ammunition but mostly it is due to deliberate interference with the intention to increase the lethal power. Balling of shot may sometimes lead to complex injury, i.e. a part of picture may resemble that of a shotgun injury at distant range (caused by individual pellets that have not fused) and the other showing circular or oval wound (caused by the welded/balled shot-mass) resembling that of a rifled weapon.

Such a situation came to be seen in a case wherein during an assault, one party successively fired upon the other. The victim received lacerated puncture wounds upon the right forearm (caused by pellets, the so-called '*kachi goli*' in the language of police) and distinct wounds of entry and exit on the front and back of lower abdomen measuring 1.5 × 1.5 cm and 1.75 × 1.5 cm, respectively (caused by welded/balled shot-mass, the so-called '*pakki goli*' in police language). It was being argued

that two persons were involved carrying different weapons. However, the police asserted that there was only one person carrying some country-made weapon. And, it was possible to produce two types of wounds by deliberate tampering with the cartridge contents.

BLANK CARTRIDGE

A cartridge without a bullet/missile contains a charge meant for generating a loud report on firing. Employed for theatrical performances, training game dogs and for military training, etc. The cartridge often contains black powder, as it produces gas at an extremely rapid rate so as to generate the noise. In the military, special grading of smokeless powder is used in such cartridges in order to produce the desired effect. The powder is retained in the cartridge case by means of wadding; the mouth of the case being folded or crimped to retain the wad. This wadding therefore can cause injuries, when ejected, up to 5 metres from the gun. Military blank cartridges have the mouth of the case extended and folded so as to mimic a missile, but so designed as to split open on firing. More recently, blank cartridges are being made of plastic material with the nose weakened so that it splits under pressure but does not eject any solid material.

General Makeup of a Firearm (Fig. 16.2)

STOCK OR BUTT

This is the rear part of gun, which is held in hand (in case of short barrelled or hand rest gun) while firing one round of shot.

BARREL

It is the hollow cylindrical length of gun, which has the following functioning parts:

- **Chamber:** It is the posterior part of the barrel that accommodates the cartridge to be fired. The posterior wall of the chamber has a metallic plate (breach plate), which has a central hole for the percussion pin of the hammer, to strike the percussion cap at the centre of the back of the cartridge, which initiates the process of firing of a round.
- **Taper/leed:** The inner diameter of the chamber part of the barrel is wider than the rest. The part of the barrel, anterior to the chamber, tapers anteriorly. This part of the barrel is known as taper or leed. In a smoothbore gun, this part of the barrel is termed as the chamber cone.
- **Bore of the barrel:** The vast length of the hollow barrel anterior to the chamber cone or the taper is called the bore.
- **Muzzle:** The anterior end of the bore is the muzzle end of the barrel.

BREACH MECHANISM

- **Hammer:** Behind the chamber, there is a hammer with a pointed pin (percussion pin) at the centre of the anterior surface of the hammer.
- **Trigger:** Below the chamber there is the trigger that, when pulled, cause the hammer to advance and the percussion pin of the hammer to pass through the central hole of the breach plate of the chamber to strike the centre of the posterior surface of the cartridge (percussion cap).
- **Trigger guard:** The trigger is surrounded by a metallic trigger guard, so that the trigger will not be pulled accidentally.

Mechanism of Bullet Wound Production

Leaving the decelerating injuries, all mechanical trauma is caused by transference of energy from an externally moving object to the tissues, and the injuries produced by missiles are the classical examples. Bullet wound production depends upon the combined action of different factors. Some are inherent in the missile itself—speed, size, shape and character of its motion in flight at the time of hitting the target. Other factors depend upon the nature of the target—the density, cohesion and character of tissues struck and the rate of energy transmission from the missile to the tissues.

SHAPE, SIZE AND VELOCITY OF THE MISSILE

Large bullets cause greater damage than the small ones. Round bullets produce larger wounds than conical ones. Round bullets may show deflection in their course by coming across some solid object, may be some body structure or some object being worn or carried by the victim. Berg reports a case in which a metal trouser button was hit by a bullet and while the bullet itself after hitting the button fell to the ground, the button was drawn into the abdomen.

Modern steel-jacketed bullets used in army weapons have the shape of an elongated cone and owing to their great velocity, usually pass straight and direct through the body tissues without any deflection and without causing much damage. The wounds of entry and exit may be similar in appearance in such cases.

Velocity of the missile is a significantly important factor as the wounding power of the missile is directly related to its kinetic energy (KE), i.e. $\mathbf{E = mv^2/2gm}$. Therefore, it is obvious that the KE increases in direct proportion to weight (mass) and square of the velocity of the missile. A bullet, travelling at twice the speed of another bullet of equal weight and similar shape possesses four times as much energy (wounding power) as compared to the other. **Modern experts dealing with the production of ammunition** take advantage of squaring of the velocity and prefer to develop weapons having missile of small mass but exceedingly high velocity to provide the maximum kinetic energy for tissue damage.

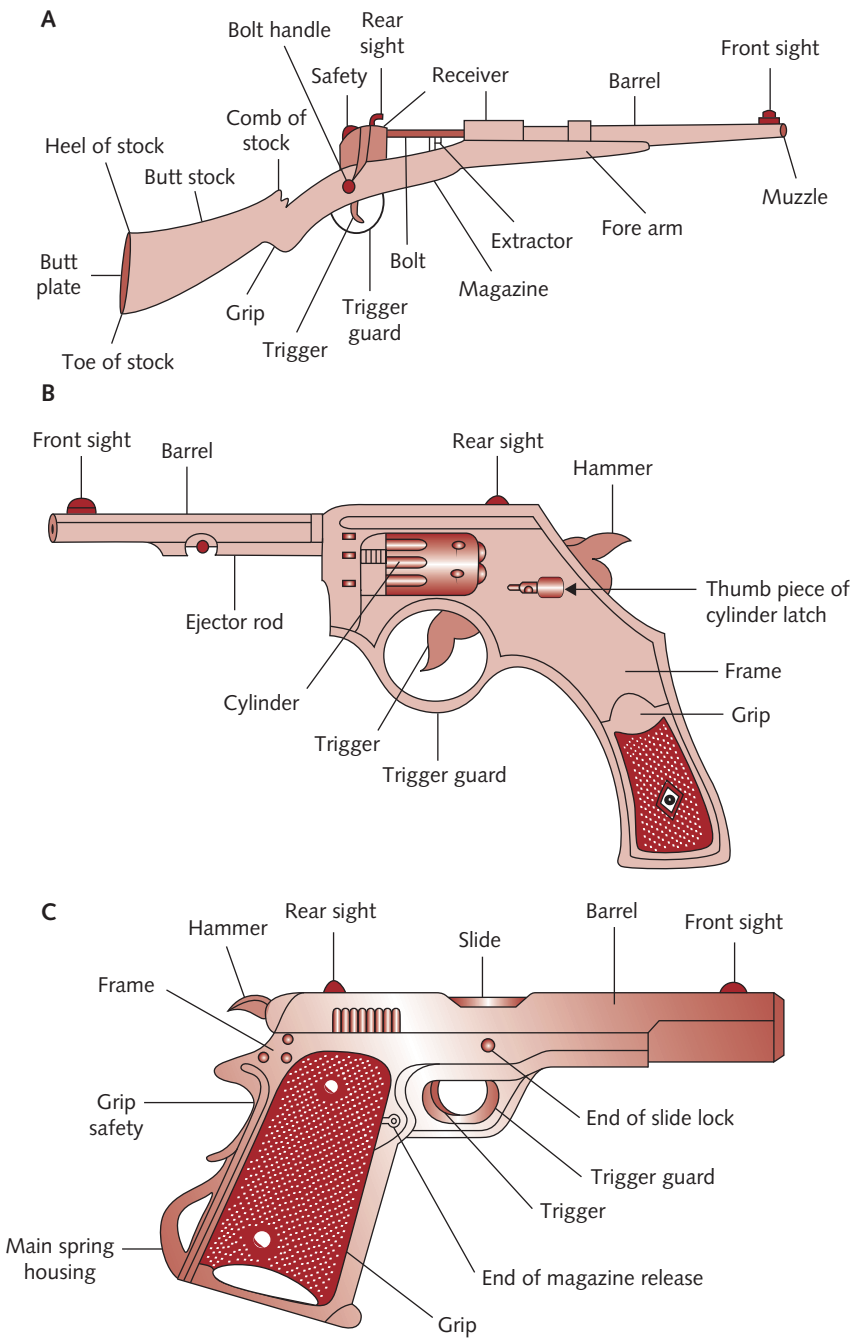


Fig. 16.2 Diagram showing general characters of **(A)** rifle, **(B)** revolver and **(C)** pistol.

Velocities are usually classified as **'low'** when below the speed of sound in air (340 metres per second or 1100 feet per second) and **'high'** when above this speed. **Missiles having 'low muzzle velocity'** like handguns mechanically thrust the tissues along with track that is only slightly wider than the missile and secondary damage occurs from rupture of blood vessels and other structures. **Missiles with 'high muzzle velocity',** i.e. above the speed of the sound in air, transmit a 'shock wave' of compression, ahead of the laceration track—this wave being propagated at about the speed of the sound in water (1500 metres per second or 4500 feet per second). Though this wave lasts for a brief period, it raises tissue pressure up to thousand of kilopascals. It can, therefore, cause damage within a wide zone of bullet track and can be propagated through hollow fluid containing vessels to cause distant vascular damage.

High-velocity projectiles also produce cavitation. It accelerates the molecules of the tissues close to the bullet track so that they continue to move centrifugally onwards even when the projectile has gone further. Thus, due to this radial displacement of the soft tissues, a cavity is produced around the track of the bullet. It reaches its maximum size within milliseconds and then decreases in size, ultimately leaving a fusiform cavity in the wake

of the bullet, even when the bullet has left the body, i.e. the track of damage persists that is wider than the actual missile. This may be termed as **permanent cavity**, while the initial cavitation may be called as **temporary cavity**. High-velocity bullets, therefore, create so much expansion and tearing of the tissues incident to the temporary cavity formation that they give rise to explosive type of changes. The term 'explosive' here is descriptive of appearance of the wound and not indicative of its origin. Rapid and tremendous transmission of energy by high velocity projectiles can fracture bones and can cause severe soft tissue damage, at considerable distances from the direct bullet path.

Passage of projectile through the chest produces comparatively little cavity formation, because the thorax is fundamentally an air-filled enclosure. In case of head, the situation is different. Here the brain is surrounded by unyielding bony framework, i.e. the cavity expansion is confined within the skull with resultant development of markedly increased intracranial pressure. Therefore, in case of high-velocity missiles, even brain pulpefaction can occur and the cranium may literally be **'blown apart'**.

CHARACTER OF MOTION IN THE FLIGHT

The trajectory of the missile also determines how much and how fast its energy is being distributed to the target area. With lead shots of smoothbore firearms being spherical, the orientation of impact is immaterial; the bullets from rifled firearms may assume an erratic course and may 'wag' or 'yaw' from side to side or may even tumble end on end, or may 'mutate' resulting in unexpected complex movements about the axis. Obviously, such movements will provide more contact with the tissues, allowing more transference of energy and thereby more tissue damage.

DENSITY OF THE TISSUES

Tissue density can be critical in evaluating the degree of damage occasioned by the missile. The greater the tissue density, the greater the amount of energy discharged by the bullet while passing through that structure. A bullet passing through soft tissues may affect relatively little damage, but the same bullet travelling at the same speed can produce extensive comminution while striking some bone. This variation in destruction and disorganisation is attributed to the fact that considerably more energy is liberated by the projectile in penetrating firm or solid tissues than it is liberated in traversing soft tissues. Soft tissues tend to dissipate the transmitted energy in radial waves, whereas solid bone shatters as the energy radiates from the site of impact.

HYDROSTATIC FORCES

Hydrostatic forces are responsible for apparently extensive degrees of damage observed in some visceral injuries due to blunt force including the bullet injuries. When a missile travels through a fluid-filled hollow organ such as food-filled stomach, urine-filled bladder, cerebrospinal fluid-filled cerebral ventricle or a cardiac chamber distended with blood in diastole, the liquid contents within these organs are displaced violently in all directions away from the bullet path, producing extensive lacerations.

ENERGY TRANSMISSION RATE

Rate of energy transmission from missile to tissues is another important factor in influencing the extent of damage produced by the missile. To ensure effective transference of energy to the tissues, some bullets are especially designed or modified to slow down or abruptly stop within the body. Soft-nose bullets get flattened on impact and some may be designed to fragment just to increase the rate of transference of energy. Dum-Dum bullets are more destructive as they burst into the tissues because of crossed-grooves at their tips. Cupronickel or other jacketed bullets may produce mushrooming effect from the exposed lead-core at the tip of the bullet, which expands on striking and thus producing enormous lacerations. Some military missiles have an air-cavity within the tip that, therefore, is intended to splay open on impact to multiply the deceleration effect, thereby transferring greater energy for disruption.

Wounds Produced by Rifled Firearms

The injuries produced by these weapons depend upon multiple factors as detailed above. However, there are some characteristics common to all. Therefore, they may be considered together.

It must be kept in mind that **when a firearm is discharged**, a tongue of flame and hot gases follows the missile/lead shots. The gases are at high pressure and temperature at the muzzle (the gas may be heated to 5200° F and the pressure exerted may vary from several thousand to 50,000–60,000 pounds per square inch), but rapidly expand and get cooled, producing the **'report'** or **'noise'** associated with the discharge. The flame is of significance in contact or near-contact wounds where it may sear the skin around the entrance wound. The 'ball of fire' emerging from the muzzle consists of oxygen-deprived gases like oxides of nitrogen, carbon dioxide, carbon monoxide and other compounds. When they emerge from the barrel at extremely high temperature, they react with the oxygen in the atmosphere producing what is commonly known as **'muzzle flash'**, particularly well appreciated at night or in a dark room. Other effects that are expelled are the **'soot'** from the combustion of the propellant along with some semi-burnt or unburnt flakes or grains of the propellant. **The gas,** with its contained soot, is very light and therefore travels a short distance measurable in inches. **The powder grains** being heavier travel further; the distance travelled depends upon the type of powder and kind of weapon. Here, it may be reminded that the amount of unburned or partially burned powder exiting depends largely upon the burning properties of the powder and the length of the barrel. The rate of burning can be controlled by the manufacturer by means of varying the size and shape of the powder grains as well as by coating them with substances that retard combustion. The size

and shape affect the burning rate by controlling the amount of surface area exposed to the flame. As far as the length of the barrel is concerned, it is obvious that shortening the barrel will cause more unburned powder to emerge and lengthening of the barrel will cause the reverse. **Chemical traces of elements in the detonator or the priming mixture** also accompany the discharge but are not visible. However, they can be demonstrable in the laboratory, in case the samples of tissues/clothing(s) from the wound/defect of entrance are sent to the laboratory. **Hangfire** may be caused by contamination and/or degradation of either the primer or the propellant. In a series of experiments attempting to induce hangfires, Haag was unable to do so by contamination or degradation of primers. The primers either discharged or misfired, i.e. failed to fire. Contamination of propellant, however, resulted in both misfires and hangfires. Haag concluded that with the modern ammunition, hangfires were rare.

The wounds may be categorised on the basis of muzzle–victim distance because this factor is the most critical feature, primarily responsible for the production of their characteristic appearances. A useful and simple classification of entrance wounds is as follows:

- Contact wounds
- Close-range wounds
- Intermediate/short-range wounds
- Medium-range wounds
- Extreme-range wounds

CONTACT WOUNDS

The following three types of contact situations may be described:

- **Firm contact with skin over shallowly situated bone:** The prime example of such a situation is the contact wound of the head. Here, the gas and the other effects are forced through the scalp, but the shallowly situated bone serves as a barrier to the rapid deep expansion of gases. Therefore, the skull tends to turn back the ever expanding cone of gases that then tends to blast out around the muzzle of the firearm, everting the tissue and imparting it an **'explosive'** or **'eruptive' appearance**. When the gas volume is large, the dome may then split, resulting in a cruciate, stellate, triradiate or ragged wound with skin flaps.

  As the contact with skin is firm, an effective seal is formed between the muzzle and the skin that prevents much escape of gases, soot and powder, so that soiling, burning and powder deposition around the margins of the entrance wound will be minimal or absent. However, if the blast is powerful enough, it usually shatters the bone and hence most of the effects of discharge will be blown into the track of the missile. The entrance wound will, however, usually show searing and powder blackening of the immediate edge of the wound and in case of death, the autopsy will reveal soot and unburnt and/or semi-burnt powder particles in the wound track. Drying haemolysed blood and decomposition can simulate or mask soot. Generally, blood can be removed by running or spraying hot water over the wound. Clots resistant to hot water can be dissolved with hydrogen peroxide. Neither hot water nor hydrogen peroxide will remove the soot. In case of any discrepancy as to the entrance wound and where no powder particles can be identified, the use of energy dispersive X-ray (EDX) or scanning electron microscope-energy dispersive X-ray (SEM-EDX) should be employed. These devices can enable one to analyse for the vaporised metals from the bullet, cartridge case and the primer.

  If the skull gets fractured, the wound of entrance shows a **punched-in (clean) hole** in the outer table, and inner table shows a bevelled crater; whereas reverse will be the effects at the exit (if present), i.e. **punched-out opening** is produced at the inner table and bevelled opening at the outer table. The crater effect is produced when the unsupported diploe everts and fragments on the side where the bullet leaves, this is in contrast to the approach side where the rim of the defect is supported by the underlying bone. Associated linear fractures are common, radiating from the bullet holes. Sometimes, bullets entering the cranial cavity have insufficient energy to make their exit on the other side, may ricochet from inside of the skull and cause secondary track of damage or even skid around the inside of the skull in a circumferential manner. Occasionally, in the bullet wounds of the head, it may be possible to ascertain the sequence of shots by the pattern of fracture lines that radiate from each defect. The fractures that extend from second bullet hole are arrested by those which originated from the first **(Puppe's rule)**. This rule is applicable to any multiple blunt force impact causing skull fractures.

  High-velocity projectiles may cause gross damage to the head by virtue of total release of their kinetic energy. The effects may be so destructive as to cause virtual decapitation, and the injuries may sometimes be confused with those due to explosives rather than due to simple projectile. The facial features may be distorted to a considerable extent. Restoration of skull by replacement of bone fragments may be helpful in localising the entrance defect created by the missile and direction of discharge from the direction of bevelling.
- **Firm contact with skin but not over shallowly situated bone:** Here there is no layer of bone to divert the expanding cone of gases and therefore the various effects of discharge continue to penetrate deeper and get dissipated in the surrounding soft tissues. As the contact is firm, there will be little or no sideways escape of flame, gas, smoke and powder particles, etc. There may be abraded-bruised area in the immediate vicinity of the wound edges. The wound will not be of eruptive or explosive type but will usually be circular or oval.
- **Loose contact with skin:** Here some of the gases escape with the resultant scattering of the muzzle blast and an unusual arrangement of soot is seen on the skin surrounding the entrance wound. This is known as **'corona'**. The corona consists of a circular zone of soot deposit surrounding the

missile defect but separated from it by a band of normal skin. This is due to the fact that the gas expanding about the muzzle, initially is at too high a velocity to allow for the settling of the soot, but at a short distance away, as the velocity gets reduced allowing the soot to be deposited at a short distance away from the missile defect. The blast effect is not as pronounced as in case of firm or tight contact, and splitting of the wound margins usually does not occur. A few unburnt and/or semi-burnt grains of powder may also escape out of this gap and be deposited on the skin interspersed in the band of soot. Particles of powder, vaporised metals and soot will be deposited in the track along with carbon monoxide.

Under all these contact situations, carbon monoxide combines with the haemoglobin and myoglobin of the tissues of the track and thereby imparts pinkish colouration to the interior of the wound track and the adjacent tissues. This concentration goes on diminishing along the track. Presence of carboxy haemoglobin and myoglobin has been suggested as a test for distinguishing entry wound from that of exit, particularly where the decomposition has affected the morphological characters of the wounds. Even if the cherry-red hue of the muscles in the wound track is not appreciable, elevated levels of carbon monoxide may be detected on chemical analysis. Control samples of muscle should always be taken from another area of the body if such determinations are to be undertaken. By using gas chromatography, carbon monoxide has been detected in wounds inflicted up to 30 cm from the muzzle. The presence of both powder particles and carbon monoxide in a gunshot wound would seem to leave no doubt as to the fact that one is dealing with the entrance wound. Very rarely, both the carbon monoxide and powder may be found at the exit, particularly where ball powder is involved that possesses more penetrating capability as compared to flake or cylindrical powder.

Muzzle Imprint

There may be muzzle impression in tight contact wounds, most frequently, when the shot is in the area overlying shallowly placed bone but sometimes may also be seen over the area having no shallowly placed bone. Such an impression or imprint results when the skin is slapped against the muzzle due to rapid and extensive expansion of the gases within the tissues deep to the skin. Many muzzle impressions fail to be recorded because of the rapid removal of the weapon by recoil. The imprint may be incomplete and indistinct and rarely may be a perfect impression of the muzzle in the form of abraded-contused area. In rifled weapons, especially hand guns, there are more features at the end of the barrel than the shotguns. Therefore, complex imprints may be made of foresights and the mechanisms for self-loading in the automatic weapons. Similar patterns may occasionally be noticed upon clothing especially when viewed through special techniques such as infrared photography. The interposition of clothing may, of course, prevent any distinct skin mark being made in a contact wound.

Blow Back into the Barrel

Contact wounds may cause blood, fragments of tissues, hair and fibres of clothing to enter the muzzle, sometimes penetrating for several centimetres. This is due to negative pressure created following the discharge and is known as **back spatter**. Occasionally, pieces of skin and/or adipose tissue have been found inside the weapon. The occurrence and degree of back spatter depends upon the anatomical location of the wound and range and calibre of the weapon. A contact wound of the head from a large calibre weapon is more likely to produce back spatter than a wound of the trunk from a small calibre weapon. Back spatter is important because the resultant stains may be found on the weapon, the shooter and the objects in the vicinity.

CLOSE-RANGE WOUNDS

Entrance bullet wounds are characterised as close range when the muzzle to target distances are such that the target surface is still within the range of flame and muzzle blast (which is usually not more than 1–2 inches in case of handguns). The entrance wound is usually circular or oval with inverted margins, but the rebounding gases may sometimes get levelled up and evert the margins. **All the effects of a discharge will be appreciated at such ranges, namely:**

Skin Burning and Hair Singeing

Within a few centimetres of the bare skin, there is likely to be burning of the skin and singeing of the hair, if no clothing are interposed. The surface hair, if present, may be either completely removed by burning down to skin level or may be blown away by the gases or may be shrivelled, blackened and clubbed owing to burning and melting of the keratin. The skin may show scorching of the epidermis and if the victim survives for any length of time, reactive hyperaemia, swelling and probably blistering may also be seen.

Soot or Smoke Soiling/Blackening

This is also called 'smudging'. Deposition of soot is far more marked with the black powder than with the modern nitrocellulose type of propellants.

Powder Tattooing

Alternatively it may be termed as 'stippling' or 'peppering'. These marks are due to semi-burnt or unburnt powder particles and again far more common with black powder than with modern propellants. Individual tattoos are caused by individual semi-burnt or unburnt powder particles/grains being blown into the skin of the victim. Physical forms of propellant powder exert their influence in the production of powder tattooing. Usually, there are four forms of propellant: flake, spherical, ball and cylindrical powder. Ball powder is favoured in high-pressure

loadings and provides consistent homogenous ignition. Flake powder usually is in the form of discs. The sphere has a better aerodynamic form than a flake, thus ball powder can travel farther retaining more velocity and enabling it to mark the skin at a greater distance. Due to the same reason, ball powder can readily perforate hair and clothing at close and medium range. In contrast (except at close range), flake powder usually does not produce powder tattooing through clothing or dense hair.

The presence/absence of powder tattooing and also of smoke effects can be more readily demonstrated by infrared photography. If the firearm is discharged on the clothed part of the body, clothing may filter out soot and powder particles so that none may be seen on the skin. Measurement of spread of particles on the target is necessary for the subsequent comparison with the patterns obtained by test shots to evaluate the range of firing. Scaled photography, if available, is recommended. In case of survival, evidence of powder tattooing becomes accentuated due to inflammation associated with the process of healing. This is in contrast to soot, which is easily removed by improper handling.

Fouling

It refers to tiny lesions around the entry wound, caused by metal fragments expelled by the discharge. These fragments may come either from the surface of missile or from the interior of the barrel. Friction between the bullet and the rifling may scrape pieces of lead or steel barrel and hurl them on to the skin where they become embedded. These particles, like powder particles, will not be wiped off whereas the soot soiling can easily be removed with a wet sponge.

As in case of contact wounds, the carboxy haemoglobin and myoglobin will be present in the wound track in diminishing concentration as the range increases.

Abraded collar and grease or dirt ring may be present though not distinctly appreciable due to dominance of other effects of the discharge. Therefore, these will be described in detail under medium-range wounds.

INTERMEDIATE/SHORT-RANGE WOUNDS

This term may be applied when the victim is within the range of powder disposition but outside the range of flame and muzzle blast. When the discharge occurs at a distance of few centimetres, the lacerating and burning effects of gases and flame are usually lost due to dispersive cooling of gases before they reach the skin. The entrance hole is seen as round or oval about the size of the missile with abraded-bruised margins and with a distinct zone of blackening and tattooing but absence of burning/singeing, etc. The production of **'powder tattooing' is the sine qua non** of the intermediate-range gunshot wounds. In addition to the powder tattooing, there may also occur blackening of the skin or material around the entrance site from soot produced by combustion of the propellant. The size and density of the area of powder tattooing vary with the calibre of the weapon, the barrel length, the type of the propellant and the distance from the muzzle to the target. As the distance increases, the intensity of the powder blackening and tattooing decreases and the size of the soot or powder pattern increases. For virtually all handgun cartridges, soot is absent beyond 12 inches (30 cm). Although soot can be easily wiped away either by copious haemorrhage or by intentional wiping, but powder tattooing cannot. Powder tattooing consists of numerous reddish-brown to orange-red punctate lesions surrounding the wound of entrance. The distribution around the entrance site may be either symmetrical or eccentric, depending upon the angle of gun to the target at the time of discharge, the nature of the target (flat or otherwise) and any covering of the skin, e.g. hair or clothing that may prevent the powder grains from reaching the skin. **Powder tattooing is an antemortem phenomenon** indicating that the individual was alive at the time of discharge of the weapon. If the individual was dead before being shot, although the powder may produce marks upon the skin, these marks will show moist grey or yellow appearance rather than reddish-brown to orange-red colouration of antemortem tattooing and moreover, they are usually less numerous than markings produced in the living subject at the same range. Punctate abrasions of tattooing usually heal completely if the victim survives. In the region of palms and soles, the vital reaction involving punctate abrasions due to penetration of the powder particles may not be easily appreciable and this is probably due to the fact that the thickness of stratum corneum protects the dermis from the impact of powder grains at such sites. It may be stressed again that the maximum range at which the tattooing occurs as well as the size and density of the powder tattoo pattern depends not only upon the form of the powder but on a number of other factors, including barrel length, calibre, individual weapon and the presence of intermediary objects such as hair, or clothing, etc. Silencers will filter out a great proportion of soot and powder particles, thus making the range appear greater than it actually was. The size and density of the powder tattoo pattern on the body around the wound of entrance can be used to determine the range at which the weapon was discharged by the replication of this pattern on the test material, using the same weapon, the ammunition identical to that of the fired round and the similar background. Although isolated powder grains may travel much farther, patterns of tattooing are usually not to be seen at a range of over 4 feet.

In addition to soot and powder grains, **other materials are also deposited on the body at such a range. These materials include** antimony, barium and lead from the primer; copper and zinc vaporised from the cartridge case; copper, aluminium or lead stripped or vaporised from the bullet that was fired and the grease and oil coating the barrel or the bullet before discharge. The metallic elements can be detected on the body or clothing(s) by soft X-ray if they are sufficiently large. Trace metal deposits can be detected by EDX and SEM-EDX.

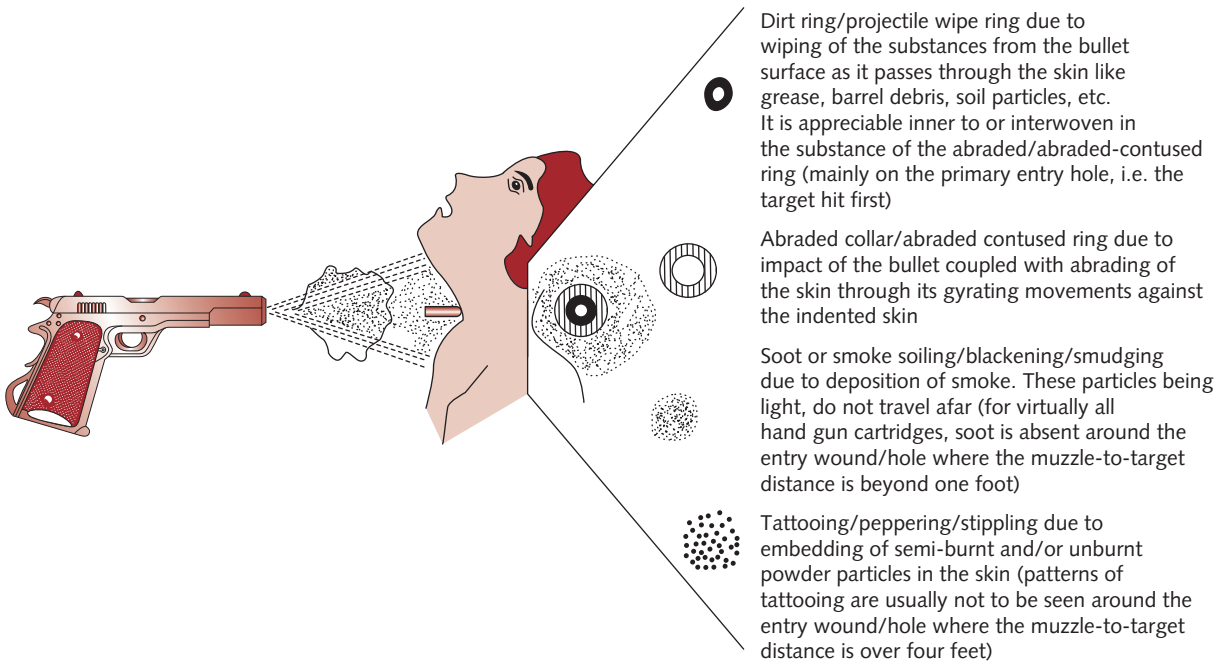


Fig. 16.3 Diagrammatic as well as descriptive representation of formation of various effects and deposition of extraneous deposits around the entry wound of a bullet at appropriate range(s). The extent of such deposit depends upon the weapon, the ammunition, the range plus angle of fire, and the target characteristics.

MEDIUM-RANGE WOUNDS

Once the discharge of rifled weapon is greater than 5–6 feet, there is nothing to indicate increasing range. Consequently, the appearance of the wound inflicted from a distance of 5 feet or 50 feet will be similar unless at extreme ranges when the erratic appearance of the wound may occur due to instability of the bullet.

The entrance wound at such ranges is usually circular or oval, and the margins are driven inwards by the passage of missile. The size of the hole is rarely equal to the size of the diameter of the missile and therefore the calibre of the weapon cannot be determined from the inspection of the wound. The reason for noncoincidence of skin puncture and the missile size is that the projectile indents the skin before penetrating it, so that perforation is effected with the skin under tension. After the bullet passes through the skin, the skin tends to return to its former size and edges of the wound contract and the resulting hole is smaller than the diameter of the missile.

Abrasion Collar (Marginal Abrasion)

The skin immediately around the central aperture shows abrasions and even bruising. It is, therefore, also called as abraded–contused collar (Fig. 16.3). This results from abrading of the skin around the entrance wound due to rubbing of the gyrating body of the bullet against the indented epidermis. This abraded zone is reddish at its commencement but becomes brown and then brownish-black as it dries. This dried and discoloured abrasion collar may not be confused with the blackening or marks of powder because it provides no indication as to the range (Fig. 16.3).

Apart from being a proof of an entrance wound, its distribution around the margins of the wound is also helpful in determining the direction of fire. If the weapon has been discharged perpendicular to the body surface, the abrasion ring is circular and uniform but if the weapon has been discharged from the side (i.e. obliquely), it presents an elliptical shape, the longer axis pointing towards the direction of approach of the missile (Fig. 16.4A and B). This, however, assumes that the body surface, where the bullet strikes, is flat. However, where the body surface shows curve, depression or projection, the results will need cautious interpretations. Occasionally, an entrance wound may not have an abrasion ring. This can be due to the nature of the bullet or the location of the entrance wound. Wounds from high-velocity centrefire rifle bullets may not show abrasion ring but exhibit small splits or tears radiating outwards from the edges of perforation. These usually involve the complete circumference of the entrance wound though like abrasion ring, they may only involve a part of the circumference. If the projectile approaches at a very small angle to the skin, there may be a furrow ploughed through the epidermis before complete entry occurs. Occasionally, a bullet may strike the body tangentially and never actually enter, so that an elongated furrow is left.

Grease or Dirt Ring

In addition to the abrasion, a coating of foreign material may be found around the margins of the wound. It is due to removal

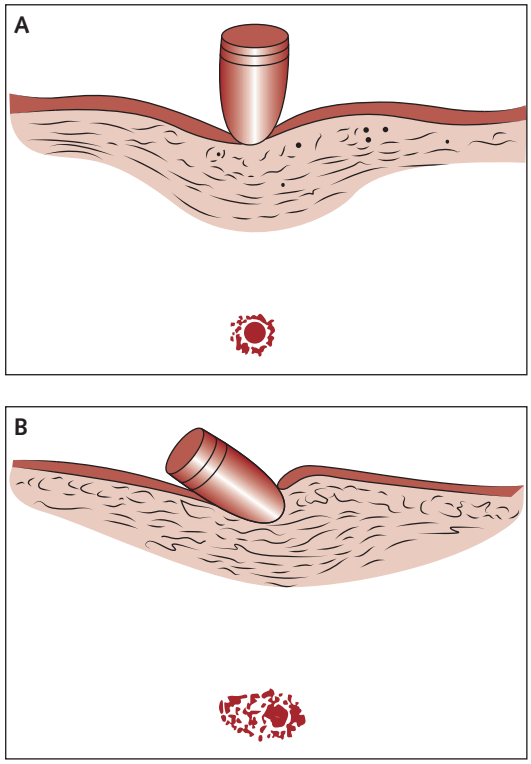


Fig. 16.4 Sketch showing different configurations of the marginal abrasion around an entrance 'missile wound', depending upon the angle at which it strikes the body: **(A)** missile perpendicular to the body and **(B)** missile at an acute angle to the body.

of the substances from the bullet as it passes through the skin, i.e. bullet lubrication, gun oil from the interior of the barrel, barrel debris and the dirt carried on the surface of the bullet as it travels through the atmosphere. This ring is present in to or interwoven in the substance of the abrasion collar (Fig. 16.3). With the jacketed bullets of hard metal, this soiling is less common. The ring is not usually appreciable on gross inspection but may be demonstrable on microscopic examination or other tests in the laboratory.

EXTREME-RANGE WOUNDS

For most of the distance of travel, the bullet remains steady with only minor variations from axial stability. When the extreme range is achieved, the reduced velocity may result in instability of the flight path. The bullet may begin to wobble and yaw and may even tumble, i.e. turn end-over-end. If the bullet strikes the body during this phase, the impact may be sideways or even backwards. The wound accordingly will be irregular and may present difficulty in its differentiation from a laceration by other means.

CONCEALED ENTRANCE WOUNDS

Bullets striking at the unusual locations may cause injury and death but the wound of entry may be extremely difficult to locate. Among the areas not anticipated as the sites of entrance are the nostrils, ears, mouth, axilla, vagina, anus and the perianal/perineal areas. Difficulty may further be accentuated if the area is smeared with blood and thereby obscuring the wound. Entrance wounds in the hairy areas such as scalp, pubis, etc. can be located by careful visual search coupled with gentle palpation. Autopsy surgeon may sometimes discover gunshot wounds missed by the police or the doctor in the emergency room. The likelihood of missing head wounds (particularly on the back of the head) is more because of thick growth of hair concealing the wound and lack of diligent search. In the emergency room, there may even occur confusion over the entrance and exit wounds. This may be partly due to massive bleeding and partly the chaos created by the relatives/attendants.

EXIT WOUNDS

The bullets that pass through the body cause exit wounds, sometimes known as **'outshoot wounds'**. The exit wound commonly presents a larger and more ragged appearance than the corresponding entrance wound, though exceptions are numerous, particularly depending upon the range of fire and other factors described in the beginning (Table 16.2).

Outshoot wounds may be of varying shapes and can be described as slit-like, stellate, cruciate, irregular or gaping. The reasons for marked variations in the shape of exit wounds are following:

- Deformation of the bullet during its passage through the body and thereby presenting an irregular wound at exiting.
- Tumbling of the bullet in the body and therefore it may not be able to exit with nose end first.
- Breaking up of the bullet in the body after striking bone. The bullet therefore exits not as a single mass but as many pieces. If jacketed, the jacket may get separated completely or partially. Conversely, the bone may get fragmented and the pieces **(secondary missiles)** may be imparted sufficient velocity so that they make individual exits.
- The bullet on leaving the body on farther side tends to produce an everted wound and there may well be enlargement and tearing of the margins due to disturbance of the steady gyroscopic path of the bullet at the exit. Because the unsupported skin is struck from within, the wound tends to burst outwards and frequently, fragments and tags of tissues may be seen either at the margins or actually extruded. An important exception to this, which is of considerable medicolegal importance, is the exit of a bullet at a point which is well supported. Such exit wounds are called **'shored or supported gunshot wounds of exit'**. The support to the skin necessary to cause an exiting bullet to make a 'shored outshoot wound' may be afforded by the following:
  - Certain items of clothing such as the waistband of trousers, the side-panels of brassiere or a man's collar and tie.
  - Certain anatomical portions of the body, i.e. the bullet exiting from the side of the chest at a point where the

Table 16.2 Distinguishing Features of Entry and Exit in Rifled Firearm Wounds

Entry wound	Exit wound
The wound is usually smaller except in 'contact' discharge when the skin is torn erratically. May also happen at 'close range' too, where rebounding gases may get levelled up and produce larger, ragged wound. Ricochetting, wobbling of the bullet may be the other reasons for producing larger wound of entrance	The wound is generally bigger than the corresponding entry wound except in the situations as described here
Edges are usually inverted as the missile penetrates the body from the outside. Deviation may be seen in 'contact' wounds.	Edges are usually everted as the missile forces its way out of the body
Abrasion collar/abrasion-contusion ring is an essential feature of the wound of entrance	Abrasion collar/abrasion-contusion is absent except in 'shored' exit wounds
Burning, singeing, blackening and tattooing may be seen at appropriate distances	Burning, singeing, blackening and tattooing cannot be there. (Blackening and/or tattooing may rarely be seen where the exit wound is present in proximity with the wound of entrance)
Grease or dirt ring may be present inner to or interwoven in the substance of abrasion collar, which may be demonstrable on micro-chemical or other tests in the laboratory	Not so
In 'contact' and 'close-range' wounds, the track near the entry wound may be bright pink due to carboxy haemoglobin. (Diminishing concentration of carbon monoxide along the track has been suggested as a test for distinguishing entry from that of exit wound, particularly where the decomposition has affected the morphological characters of the wounds)	Not so
Clothing may be turned 'in' and shreds carried into the wound to a varying distance	Clothing may be turned 'out'

inner surface of the arm is being held closely against the chest wall.

- Position of the body in relation to the substances in the surroundings, i.e. when the victim is leaning against some firm object like a wall.

In these situations, the margins of the exit wound may not be everted and there may even be a spurious 'abrasion collar' that may be produced by the emerging bullet slamming the wound margins against the resisting surface and the wound may sometimes be indistinguishable from the entrance wound. Examination of the clothing and the circumstances may provide the answer.

Wounds Produced by Smoothbore Weapons

With the discharge of a shotgun, the following constituents emerge and all or a varying combination may contribute to the production of wound depending upon the range of fire:

- Lead pellets/shots
- Flame and hot gases under pressure
- Soot in the form of smoke and debris
- Unburnt and semi-burnt propellant particles
- Wads (cardboard, felt or plastic)
- Constituents of detonator
- Fragments of cartridge case

Resting on the same justification, as described under 'Wounds Produced by Rifled Weapons', **it is convenient to describe them in terms of increasing range of discharge** (Fig. 16.5). (It must be kept in mind that factors like gauge of the weapon, degree of 'choke' size plus number of pellets and muzzle-victim distance, all play their role in determining characters of a shotgun injury.)

CONTACT WOUNDS

As written under 'Wounds Produced by Rifled Firearms', three situations may arise:

- **Firm contact with skin over shallowly situated bone:** The characters already described under 'rifled weapons', particularly those of contact and close-range wounds, are also observed in shotgun wounding. In this situation, where the gases have restricted space for expansion, extreme mutilation due to explosive effect may occur. Destruction of the entire contour of the face and head may occur and actual point of muzzle impact or entry may be difficult to locate. There is

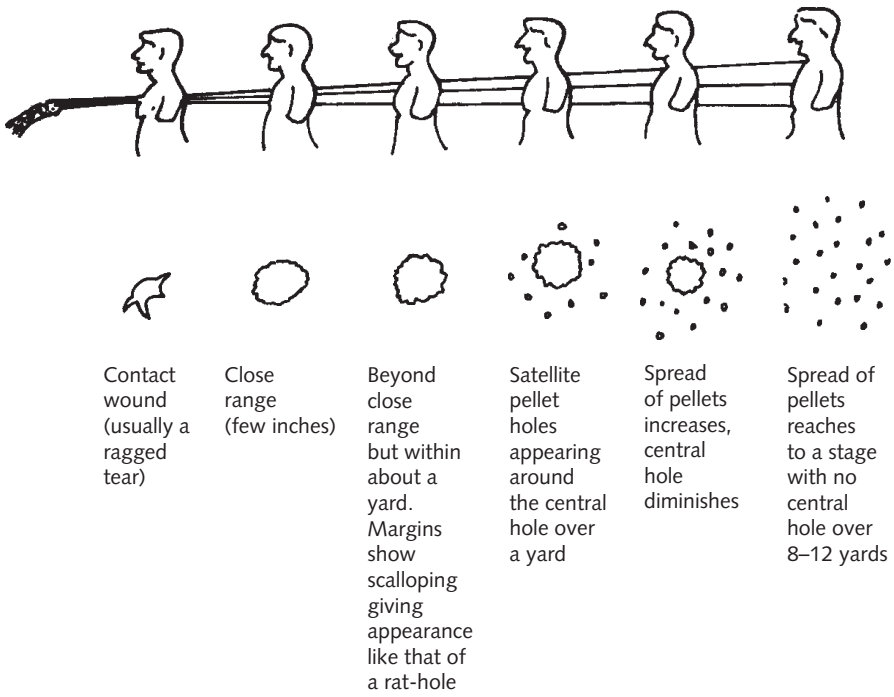


Fig. 16.5 Characteristics of shotgun wounds at various ranges (all these effects vary with the degree of choke, type of weapon and ammunition).

usually greater disruption of the margins that may often show subsidiary linear tears in the skin extending from the margins of the main wound. Skull shows large irregular hole with fissured fractures running from its margins. The margins of the skin wound may show minimal degree of soiling, burning and tattooing as the firm contact prevents the sideway escape of the effects of discharge and therefore the tissues of the track and its vicinity show burning, blackening and tattooing, etc. as the various effects are blown into the track.

- **Firm contact over an area without shallowly situated bone (like abdomen, thorax, etc.):** The consequent wound will be single and circular or oval. The edges of the wound may be crenated by the individual shot. Here there is no bone to divert the expanding cone of gases and therefore various effects of discharge will continue to penetrate deeper and get dissipated in and around the tissues of the track. Soiling, burning and tattooing, etc. on the margins of the skin wound will be minimal or absent.
- **Loose contact' with skin:** Here there will be effective deposition of various effects of discharge around the margins of the skin wound, though the tissues in and around the track will also exhibit blackening, burning and tattooing. Typical deposition of smoke in the form of 'corona' may also be present.

Other features like muzzle imprint, back spatter and absorption of carbon monoxide deserve the same mention as described under 'rifled weapons'.

CLOSE-RANGE DISCHARGE OF A SHOTGUN

When the muzzle is held close to the body, i.e. one between actual contact and about 15 cm (6″), is likely to show the following features:

- **Burning of the skin and singeing of the hair:** There is burning of the skin and singeing of the hair by the flame and hot gases, within this range. Hair may be completely burnt away. Some may get blackened, shrivelled and clubbed, due to melting of keratin and later on solidifying on cooling.
- **Soot or smoke soiling/blackening:** There is usually a wide zone of carbon deposition surrounding the wound. This spreads more widely than the powder tattooing. This effect is much less to be seen with the modern propellants.
- **Powder tattooing:** Also called 'stippling' or 'peppering' is the deposition of unburnt and semi-burnt powder particles surrounding the wound, as described in detail earlier.

The wound usually appears largely on the pattern of contact wound, though there is more extensive smoke soiling and powder tattooing. **'Nibbling'** or **'Crenation'** of the edges of the wound may be seen. There may also be annular abrasions and bruising. The tissues within and around the wound may be cherry-red due to absorption of carbon monoxide.

Any felt or cardboard overshot wads or plastic cups from the cartridge are usually encountered within the depths of the wound at such a range.

INTERMEDIATE/SHORT-RANGE DISCHARGE OF A SHOTGUN (Within about a Yard or so)

The shorter range nearing the distance of close range will provide the similar picture as described under 'close range'. If the wound inflicted is outside the range of flame and gases but within one yard, the effects like burning/singeing will be absent but others will be present. Up to about 1 yard, the wound is likely to be single as the shotgun charge enters the body as a single conglomerate mass producing an irregularly circular or oval defect and contused/lacerated, inverted margins, somewhat larger in diameter than the bore of the barrel. The margins of such wounds may also exhibit some scalloping. A zone of blackening and tattooing will be present around the wound. The overshot cards may be seen within the depth or just underneath the entrance of the skin wound. Carbon monoxide may be present in the blood and tissues damaged near the entry wound.

MEDIUM-RANGE DISCHARGE OF A SHOTGUN (1–5 Yards or so)

Beyond about a yard or so, satellite pellet holes begin to appear around the main entrance wound. This spread of pellets increases progressively, the central principal wound diminishing at the same speed. The approximate formula of 'total diameter of spread in inches is roughly equal to the range in yards' may be applied very loosely (one-third of the spread in centimetres equals the range in metres). **As a rough rule,** the hair singeing occurs over the first 30 cm; soot staining can be seen for the first half metre and powder tattooing rarely to be seen beyond 2 metres. However, the only reliable method of determining range is to secure the actual weapon and the same brand of ammunition used and to conduct a series of test shots so as to reproduce the pattern of fatal wound on the body. It need not be stressed again that ammunition plays a great role in the size and pattern of wounding. Different brands of ammunition, even when loaded with the same shot size, produce different patterns at the same range. Another factor that can lead to erroneous results of range determination involves the measurement of shot pattern upon the body. Here, it may be reminded that occasional 'flier' may be ignored and only the main mass of pellet-pattern should be considered.

The wads may still be present in the wound up to a variable distance, usually up to about a couple of yards and occasionally as far as several feet. Therefore, the presence of a wad in the depth of the wound or even embedded in the tissues underneath the entrance wound will indicate something about the range of discharge. Often the wad assumes a lower trajectory and may strike the body below the shotgun wound. It may penetrate the skin causing a separate wound or may only bruise the skin.

DISTANT-RANGE DISCHARGE OF A SHOTGUN

Beyond about 5 yards or so, the spread of pellets becomes greater depending upon the choke of the barrel and other factors until the central (principal) hole vanishes at around 8–12 yards, and is replaced by holes created by individual pellets. At still longer ranges, the pellets begin to fail to penetrate the skin, leaving small abrasions with occasional pellets embedded in the skin. Obviously, there will be no wad injuries, no smoke blackening and no tattooing, etc., and no way of ascertaining range except to say that it is beyond the distance from which the complete dispersion of pellets usually occurs.

Regarding dispersion of shots, one misconception may be kept in mind, i.e. at close ranges, when the shots are bunched, they strike one another upon hitting the skin/clothing of the victim and may fan out in a wide pattern as they continue to travel into the body. This may lead to erroneous conclusion. The same holds true if the shot happens to strike any intermediary object, such as a door or a window, before reaching the victim. This occurs secondary to the **'billiard ball'** effect described by Breitenecker (Fig. 16.6A and B). Here, the foregoing pellets striking the intermediary object are delayed allowing the following pellets to catch up and impact the preceding pellets, thus causing dispersion of the pellets. This phenomenon has been thought to occur with intermediary targets as thin as a pane of glass or a window screen. Coe and Autin, however, demonstrated that for the dispersion to take place, the intermediary targets should have sufficient thickness and tensile strength to slow down the motion of the foregoing pellets striking the target. However, **the practicality lies in the fact** that if the intermediary target is of sufficient thickness as to cause dispersion of pellets prior to striking the victim, estimates of range from the pattern on the body may be erroneous unless the effects of dispersion are taken into account. The only way to determine the range correctly is to interpose a similar intermediary object during test

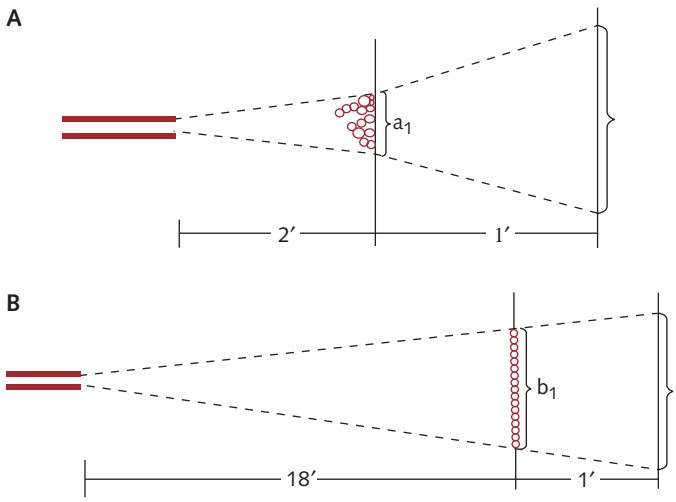


Fig. 16.6 Types of ricochet effect: **(A)** billiard ball ricochet effect at close range of firing. Note the bunched shots at primary target (a_1) tending to fan out by striking with one another upon hitting the target. **(B)** No billiard ball ricochet effect as range of firing increases. Note the spread-out shots at primary target (b_1).

firings. At occasions, attempts have been made to estimate the range of the shotgun pattern within the body by X-rays when the body has been burnt or markedly decomposed. Experiments have revealed that this method is not reliable. Both close-range wounds and wounds of several yards' distance can give rise to similar patterns on X-rays because of billiard ball effect of the pellets on entering the body in close-range shotgun wounds. However, old poorly constructed shotguns with degraded ammunition may not show such variations where the corroded pellets often adhere to each other and may not get separated on striking an intermediary target.

SHOTGUN EXIT WOUNDS

Shotgun exit wounds are uncommon, especially when the areas involved are the wider ones, i.e. chest, abdomen, etc. This is due to the fact that the residual energy of independent pellets is usually insufficient for them to emerge through such wider parts, owing to their meagre mass and relatively low velocity. Large shot sizes tend to pass through the body somewhat more frequently due to the greater mass and energy.

Practically speaking, the appearance of shotgun exit wounds is absolutely random, depending upon the part involved and the nature of tissues encountered during the passage in the body. Usually, when present, it is in the form of jagged irregular laceration with everted margins through which some tissues or bone fragments may sometimes be seen protruding. However, it is not uncommon to observe accumulation of pellets immediately beneath the skin opposite the wound of entrance after they have travelled through the body and have been trapped by the skin. The best way to trace them is acute visual observation and gentle palpation upon the area described. (**Tangential wounds** can pose considerable difficulty in the interpretation. Some may erroneously be attributed to knives or blunt injuries. The common situations may be the side of the chest, side of the face, etc. In the chest, severe damage may occur to the contents of chest, even if the penetration is minimal and death may ensue occasionally. When the side of the head is hit tangentially, shattering of skull may occur and intracranial damage is usually present, even though no metallic shots enter the cranium. Rifled weapons can also produce tangential wounds, and the lateral transference of energy can cause severe internal damage in both skull and chest, even though the missile does not enter the cavity.)

Unusual Circumstances Encountered in Firearm Injuries

There are many misconceptions concerning the trauma inflicted by the firearms. (Space does not permit the inclusion of an exhaustive catalogue of circumstances resulting in unexpected/unusual outcome.) Some frequently encountered situations may be listed as follows:

ATYPICAL WOUND OF ENTRANCE

There is a general belief that the entrance wound is always circular or oval with inverted margins and smaller than the wound of exit. This may not hold true under the following situations:

- At close range, the entrance wound may become atypically large and margins may even get everted especially when the wound is upon the area lying against shallowly situated bone. This is due to the presence of **'tail wag'** or **'tail wobble'** at such close range, before the bullet settled down into a steady gyroscopic progression. The tip of the bullet may follow the axis of the trajectory but the tail of the bullet may describe a circle or spiral, around the line of flight. If the bullet strikes the body during this phase, i.e. during the first few microseconds after leaving the muzzle, the entrance wound may be larger than the exit and the wound margins may even be everted as said earlier.
- Such a phenomenon of a large, irregular and jagged inlet wound may also be seen at extreme ranges where steady gyroscopic spin of the bullet is being lost and the bullet may begin to pursue an erratic path with tail-wag and even tumbling, the bullet turning head over heels and striking the target. In this situation, it may cause an irregular wound.
- Entry wound of a ricochet bullet is often ragged and larger as the bullet gets distorted and loses its initial velocity after the primary impact.
- Bullet while grazing the skin may produce a longitudinal furrow known as bullet slap.
- Firing the blank cartridge close to the body can also cause fatalities, when the wadding or gun powder cause irregular entry wound and laceration of the tissues.
- In case of duplex or tandem cartridge, a given round contains two bullets that will enter the body at two different places, thus causing two wounds from one fire.

RICOCHETTING OF BULLET

A ricochet bullet is one that gets deflected or deviated from its course by striking an intervening object in its way before striking the body. It may even ricochet inside the body by striking some hard/firm tissue. This deflection of the bullet on encountering slight obstacles has been ascribed partly to the obliquity with which it strikes and partly to the rotary motion on its axis (Fig. 16.7A).

A ricochetted bullet may only glance or gutter the body surface producing abrasions and/or bruising and sometimes may fall to the ground without entering the body. Bullet may also simply strike the body with 'side-on' and produce an elongated wound of entrance looking like **'key hole'**.

Particles of paint, mud, fibres, etc. may get deposited on the surface of the bullet when it strikes any object bearing these things. Such things may occasionally be carried into the track of the wound. Such trace evidence on the surface of the projectile may be identifiable with the help of SEM. In the case

described by Di Maio et al. (1987) limestone was detected that had originated from the stone surface from which the bullet had ricochetted. Another interesting case has been cited in the literature where an individual shot himself while lying next to his wife. The bullet passed through his body, entering his wife's body where it was subsequently recovered. Tissue of his blood group, which was different from that of his wife's, was recovered from the tip of the bullet. This authenticates the proposition that bullets may carry materials from an intermediary object into a body as well as material from a body to the outside (atmosphere) while exiting.

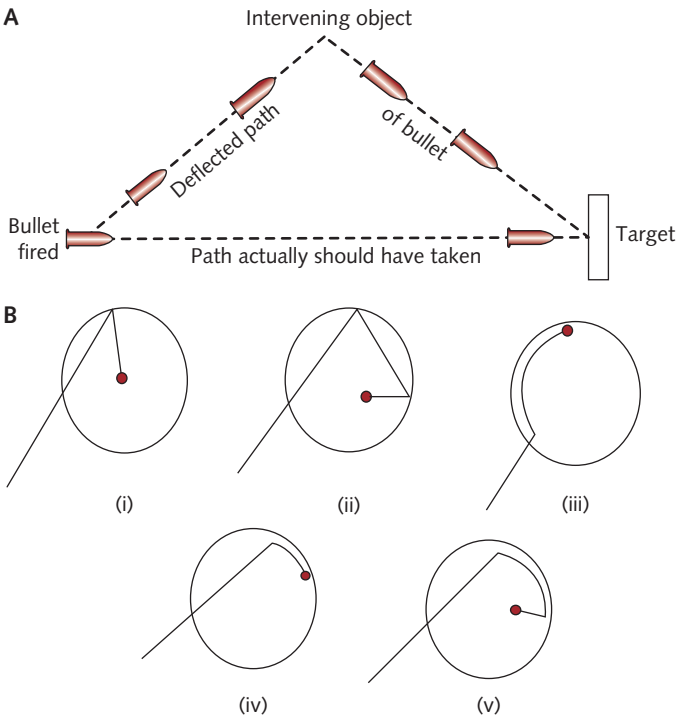


Fig. 16.7 Peculiar effects in the path of a bullet after meeting some resistance through an intervening object **(A)**, or some hindrance/obstacle within the body (here, skull) **(B)**.

While ricochetting inside the body, the bullet may somersault in the tissues when the nose of the bullet may even face the entrance wound. The phenomenon of 'billiard ball ricochet effect' in case of internal ricochetting in the cranial cavity has already been described. Therefore, a ricochetted bullet may assume a devious and circuitous course inside the body and may ultimately be found in an unexpected situation.

A rare effect of ricochetting may be observed in the impacts of the head where the bullet striking the skull obliquely gets deflected and may travel under the scalp without penetration into the cranial cavity, following the curvature of the skull for some considerable distance (Fig. 16.7B). Similarly, the bullet striking the rib at an angle may sometimes travel under the musculature of chest following the curvature of chest and may exit through some point on the opposite side without entering the pleural cavity.

More atypical wounds caused by ricochet may be produced by bullets having passed through one part of the body before proceeding into the other. For instance, a missile that has passed upper limb may cause an unusually ragged and irregularly abraded wound on its re-entry into the chest or abdomen due to tumbling and also, possibly, due to percussive effect of the limb against the area of re-entry. Such considerations may be kept in mind in interpreting the position of the victim and other derivations.

In medicolegal parlance, one cannot escape the fact that every case is unique. A police party was traveling in the train while escorting an under-trial for bringing him to the court. While climbing down the train with their heads leaning forward (as is usual with everyone), some unidentified person fired bullets upon them from behind. Three got injured. In one, a bullet grazing the scalp, entered the chest (making atypical entrance wound) and got lodged in the chest wall (Fig. 16.8).

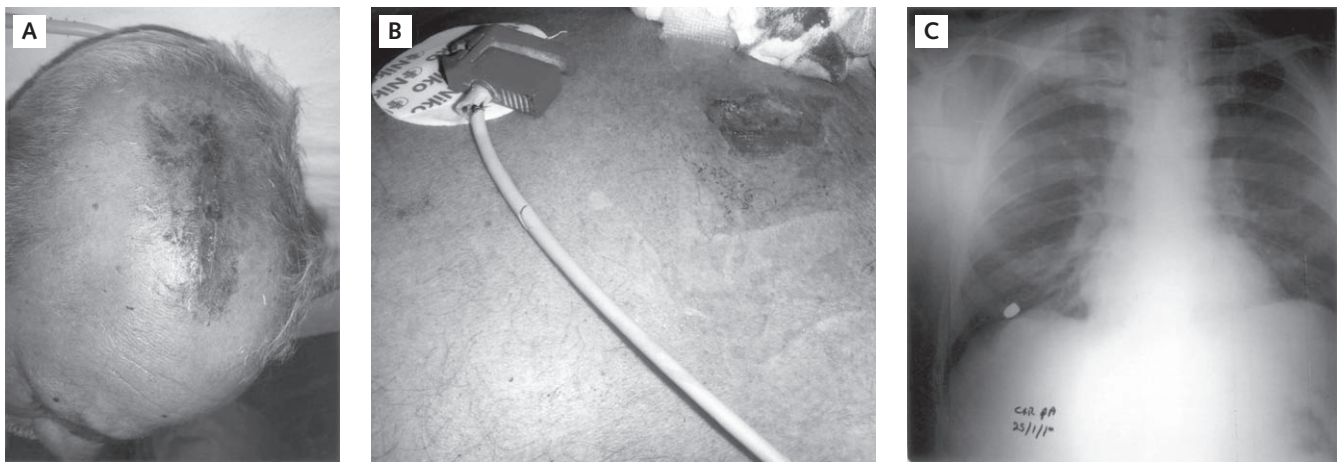


Fig. 16.8 Photographs **A**, **B** and **C** showing grazing of the scalp by a bullet, entrance into the chest (by making an atypical wound of entrance) and lodgement into the chest wall, respectively.

He was managed conservatively and discharged. Here, one gets reminded of a news report wherein it was reported that a victim, who was standing at some '*paan shop*', was attacked by some assailants by firing indiscriminately. The only bullet that hit the victim happened to strike against the 'five rupee coin', which the victim was carrying in the left pocket of his shirt. The bullet fell apart, but the coin got badly deformed leaving some bruised skin underneath (Contributed by Dr. Parmod Goyal, Associate Professor of Forensic Medicine at AIMSR, Bathinda).

SINGLE ENTRANCE AND MULTIPLE EXITS

It may result in the following situations:

- The bullet, on hitting some hard object like bone, may get fragmented and each fragment making its own way out of the body. In the jacketed bullets, fragmentation may not be ordinarily expected unless it strikes some hard object. In case of head, metal fragments may be found scattered in the brain and on X-ray may occasionally resemble a shotgun injury. Bullet may even disintegrate from the effects of its own centrifugal force. A striking case has been cited in Taylor's *Principles and Practice of Medical Jurisprudence*, 12th ed. where a deserting soldier was shot by a service rifle at a range of about 15 yards. The bullet travelled through the fleshy part of the left thigh making its exit on the opposite side and then entering the inner side of the right thigh, shattering the right femur, destroying the femoral artery on this side and made its exit on the outer side. Numerous fragments of bullet were demonstrable in the tissues showing that the bullet had disintegrated in the muscles before it smashed the bone.
- Sometimes the bullet striking the bone may break it into fragments, each fragment being imparted sufficient velocity to make its own exit. These are termed as **'secondary missiles'**.
- Sometimes the jacket of the missile may get separated and adopt its independent path and exit. Sometimes, the core may leave the body but the jacket may not. Recovery of the jacket is of importance as it carries marks/striations upon its surface, which help in identification of the suspect-weapon.

MULTIPLE WOUNDS OF ENTRANCE AND EXIT FROM A SINGLE SHOT

This occurs when the person is running or sitting or leaning in an unusual position so that several re-entries and exits can take place. Examination of clothing is important in such cases. Production of six wounds by the same bullet has been reported in a man who was bending 'on his haunches' at the time of being hit. The bullet traversing through the chest, thigh and lower leg produced the said wounds. Dupuytren cited an instance in which a bullet, after striking the tibial ridge, divided into two parts that traversed the calf of one leg and penetrated the calf of the opposite leg. Thus, five wounds were produced by a single bullet—three of entrance and two of exit.

ENTRANCE WOUND PRESENT, BULLET ABSENT

In some cases, despite the presence of entrance wound, the bullet is not traceable in the body. This may occur in the following situations:

- The bullet entering the stomach but may be vomited out and similarly entering the wind pipe may be coughed out. A case has been reported wherein an individual sustained gunshot wound of the chest. During hospitalisation, bullet was seen on X-ray apparently lodged in the right lung. The victim, however, expired after some days. At autopsy, the bullet was found in the bronchus of the left lung. Apparently, the bullet entered the bronchial tree on the right side and subsequently was coughed up and aspirated into the left bronchial tree.
- The bullet entering the gastrointestinal tract may be passed in the faeces.
- When it is so deviated, it passes out through the same wound of entrance.

TANDEM (PIGGY BACK) BULLET

Occasionally, more bullets are found than the entrance wounds. This may be on account of some defect in the weapon or due to faulty ammunition or where the loaded firearm has not been used for a considerable period or prolonged exposure to high environmental temperature or humidity. On firing, the bullet may fail to be ejected and on firing second time, the second bullet may go off carrying the lodged bullet with it and both may enter the body through the same entrance wound. This is called 'tandem' or 'piggy back' bullet. The bullets may get separated inside the body or before they hit the target. Unless it is realised, the presence of two bullets in the body of the victim may cast doubt on the version of firer that he had fired but once. The ballistic expert can evaluate that the bullets had travelled in tandem. Michaux and Thiodet (1960) reported the case of a woman who had been shot in the right breast. A bullet was recovered from the base of right side of her chest. However, subsequent X-ray examination revealed the presence of another bullet that was extracted through the same incision. The examination of the bullets showed that they had travelled in tandem.

SOUVENIR BULLETS

Long presence of bullet inside the body may be accompanied by encapsulation, and the body surface may show the original entrance wound in the form of a tiny scar that offers no clues as to the agency that produced it. The long time presence of the bullet is indicated by absence of fresh haemorrhage in the vicinity and inability to locate the recent track from the site of lodgement of the bullet to the entrance wound upon the surface of the body.

Lead poisoning from a retained bullet is very rare. As of 1994, there were 35 laboratory-documented cases of lead toxicity

from a retained lead bullet in the English literature. Onset of symptoms has occurred from months up to 27 years after being shot. It has been recognised that synovial fluid is capable of dissolving lead. A rich vascular supply to the tissues surrounding the bullet and prolonged bathing of the bullet with synovial fluid makes the development of acute lead intoxication more likely.

BULLET EMBOLISM

Bullets entering large blood vessels may behave as emboli and be carried to distant places and, therefore, may be found far from their expected sites of lodgement. Bullets entering lung and penetrating large pulmonary vein may be carried in embolic fashion back to the heart and be swayed peripherally as an arterial embolus. The most common sites of entrance of bullet into the arterial system are the aorta and the heart. In a review of 153 cases of bullet emboli in the English literature, 100 cases of embolism involved the arterial system and 53 were attributed to the venous. The source of embolism to the arterial circulation was the thoracic aorta in 37.9% of cases, the heart in 34.4% cases and the abdominal aorta in 15.5% cases. The sources of emboli to the venous circulation were the vena cavae, the iliac veins and the heart. Although the embolisation often occurs immediately following entrance of the bullet into the circulation, delays as long as 26 days have been reported.

Firearm Residues

The term 'residue' simply means something 'left over'. The term has several meanings as applicable to firearm residues. For example, the law enforcement agencies may be interested in the residues left on the hands of the suspect/assailant in firearm assaults, the ballistic expert may be looking for the residues in the firearm itself and the doctor may be craving for the residues on the victim's body in association with the firearm injuries upon the body.

The residues of a discharge of a firearm have traditionally been described as powder particles and soot produced by burning of the powder. There are actually many more residues left after the discharge of a firearm, namely residues found on the bullet, in and on the cartridge case and on the firearm itself. For a doctor, residues upon the target carry special significance. Some of these are visible but some, comprising elemental components of cartridge, primer and bullet, may be invisible as they are deposited in very minute quantities. **A potpourri of residues may be expected at shorter ranges.** However, a detailed analysis of whatever present is usually rewarding in determining range of fire and in distinguishing inlet from outlet. In determining range of fire, Forensic Laboratory Testing includes test firing the suspected weapon into cloth or paper or like material at known distances using same type of ammunition as was used in inflicting the original injury and comparing the results with the characteristics of the pattern upon the clothing or skin of the victim. This calls for the imperative necessity of having accurate documentation of wound pattern including the distribution of various elements of discharge as early as possible and prior to any major alteration resulting from any medical/surgical intervention.

FIREARM RESIDUES ON SUSPECT'S HANDS

Residues on the hands may be visible, in which case their presence needs to be observed and described. More frequently, the residue is not visible to the unaided eye. Special techniques should be employed to demonstrate invisible residues. The first such test was the **'paraffin test'** also known as the **dermal nitrate test/diphenylamine test**. This was introduced in the United States in 1933 by Teodoro Gonzalez of the Criminal Identification Laboratory, Mexico City Police Headquarters. In this test, the hands were coated with a layer of paraffin. After cooling, the casts were removed and treated with an acid solution of diphenylamine, a reagent used to detect nitrates and nitrites that originate from the gun powder and may be deposited on the skin of the person who has fired the weapon. A positive test was indicated by the presence of blue flecks in the paraffin. However, false positive results were also obtained on the hands of the individuals who had not fired the weapon because of widespread distribution of nitrates and nitrites in the atmosphere. Therefore, this test was discarded.

In 1959, **Harrison and Gilroy** introduced a qualitative colourimetric chemical test to detect the presence of barium, antimony and lead on the hands of the individuals who fired the firearms. These metals, originating from the primer, get deposited on the back of the firing hand. In revolvers, these metals come primarily from the cylinder–barrel gap and in automatic pistols from the ejection port. In this test, a square of white cotton cloth was moistened with hydrochloric acid, and the hand was swabbed with it. The swab was treated with triphenylmethylarsonium iodide for the detection of antimony and sodium rhodizonate for the detection of barium and lead. The limited sensitivity of this test prevented its widespread adoption.

By the mid-1980s, **there were three generally accepted methods of analysing gunshot residues**, namely (*i*) neutron activation, (*ii*) flameless atomic absorption spectrometry (FAAS) and (*iii*) SEM-EDX. All the three methods were based on the detection of metallic elements, chiefly barium, antimony and lead.

Neutron Activation Analysis

It was launched during the 1960s. A sample is obtained from the hands by the use of paraffin or by washing the hands with dilute acid. It is then exposed to radiation from a nuclear

reactor emitting neutrons. Secondary radioactivity is induced in the materials removed from the hands and by making appropriate counts at different energy levels, the elemental composition of the residues can be determined with precision. The technique is extremely sensitive and very minute quantities can be estimated.

By the 1990s, neutron activation had been discarded as a method of analysis. This was due to its limitation that it could analyse only for antimony and barium but not lead and thus had to be used with FAAS and secondly, it was expensive too because of involvement of nuclear reactor to perform this test.

FAAS

This method is easy for analysis, carries adequate sensitivity and low cost. FAAS will detect antimony, barium and lead from the primer as well as copper vaporised from either the cartridge case or the bullet jacketing. In this method, palms and backs of the hands are swabbed with four cotton swabs moistened with hydrochloric acid. A fifth swab is moistened with acid and acts as a control. The metallic elements are then detected. Based on distribution and amounts of antimony, barium and lead detected on the four surfaces of the hands, one may conclude whether the deposits are consistent or inconsistent with gunshot residues and thereby firing of a weapon. Typically, the residue is deposited on the back of the firing hand of the suspect who fired the gun. Detection of primer residues on the palm of the hands is suggestive of defensive gesture rather than of firing a gun. In suicides with handguns, primer residue on the palm may be due to cradling the gun with this hand at the time of firing. With rifles and shotguns, residue is often detected on the non-firing hand that has been used to steady the muzzle against the body. For correct interpretation of the result, one must take into account the surface area of the hand which is positive, the quantity of metals deposited on different areas, the nature of the weapon, etc. In living individuals as the time interval between firing and taking of samples increases, there may occur loss of residue from the hands. This can be produced not only by washing of hands but also just by rubbing them against different materials.

SEM-EDX

Here, the gunshot residues are removed from the hands using adhesive lifts. The material removed is scanned with SEM for the gunshot residue particles. The X-ray analysis capability is used to identify the chemical elements in each of the particles. The analysis by this method is not as time-dependent as FAAS and neutron activation analysis. Analysis of the hands of the firers by SEM has been positive up to 12 hours after they fired the weapon. However, the weakness of this method was reflected in the intensive labour required for the analysis and inability to quantitate.

FIREARM RESIDUE ON THE VICTIM ASSOCIATED WITH THE WOUND OF ENTRANCE

The visible residue, as noted earlier, consists of soot deposits, bullet lubricant, powder tattooing and occasionally lead stippling. The invisible residue consists of primer constituents and vaporised metal from the bullet, its jacket (if any).

Ordinarily, the visible residue is noted in the immediate vicinity of the entrance wound. Detection of such residue is best accomplished by removing the skin (2.5 cm × 2.5 cm × 5 mm) surrounding the defect and searching for powder grains, preferably with the dissecting microscope. The wounds of inlet and outlet can be distinguished in this manner, particularly in the decomposing bodies or whenever there is an issue whether the wound is of contact type or distant range. When searching for the invisible residues, in and surrounding the wound of entrance, the examination of the tissue by energy dispersive X-ray apparatus is rewarding.

CARBON MONOXIDE

Carbon monoxide also needs to be mentioned as a firearm residue. At contact and close-range wounds, carbon monoxide combines with haemoglobin of the blood and myoglobin of the muscle to form carboxy haemoglobin and carboxy myoglobin, respectively. Demonstration of these in the tissues surrounding the surface wound and the track of the wound has been advocated as a helping criterion in distinguishing inshoot wounds from the outshoot ones. This has been stressed at appropriate places.

Direction of Fire

RIFLED WEAPONS

The direction of discharge of a firearm may be derived from the relationship of entrance to the exit wounds, though due consideration to internal deviation, if present, be given. Distinguishing entry wound from the exit wound may not pose any difficulty in contact or short-range wounds because of geometry of effects like blackening, tattooing, etc. surrounding the wound of entrance. However, in case of distant shot, this distinction may be difficult and the characteristic feature of circumferential marginal abrasion around the wound of entrance may come to help. This results from scraping of the wound margins by the gyrating motion of the penetrating missile. This abrasion becomes darker and consequently more conspicuous when drying of the area sets in. The width of this abraded area surrounding the wound is uniform, if the bullet strikes the body perpendicularly and if it strikes the body at an angle, the wound of entrance may be round or oval but the marginal abrasion will be of unequal distribution, measuring more on the side of approach of the missile (indicating direction of discharge of firearm). Sometimes, the bullet may be lodged in the body itself without exiting; in that case, the path from the

entrance towards the lodgement of bullet inside the body is to be evaluated for determining the direction of discharge. Further, bullet may undergo disintegration or, occasionally, the jacket of the bullet may get separated from the core on impact over the hard object like bone and may assume a separate track. The core may leave the body but the jacket seldom does so and may be recovered from the body itself. All these possibilities **emphasise the need of X-raying the corpse** in order to avoid unnecessary dissection and to have better interpretation of the path of the missile/fragments, etc. (Table 16.2).

Bizarre or very rarely patterned abrasions adjacent to the wound of entrance may be caused by the coarse articles of clothing scraping on the skin, which probably is the outcome of percussion wave produced by the missile upon striking the target. Pieces of fabric may be driven into the wound track and found in proximity of the entrance wound. Careful examination of the clothing will facilitate the proper interpretation of the findings under such circumstances. Marginal abrasion of an exit wound is rare and occurs only under those circumstances as described under 'shored exit wounds'. Hence, circumstantial evidence will come to the rescue in such cases. In questionable cases, laboratory examination will resolve the issue.

Once the differentiation between an entry and exit wound has been effected, the trajectory between an entry and exit establishes the direction. However, again a caution is to be exercised providing due weightage to the phenomenon of ricochetting, if there seems any possibility and, secondly, the attitude of the victim's body at the time of impact also deserves evaluation. For example, a horizontal wound track usually results if the victim is standing upright when confronted by the assailant or if the victim is lying on the ground and the assailant is standing over him. Many bizarre examples can occur and, therefore, it is advisable to be conservative in extending opinions.

SMOOTHBORE WEAPONS

It has already been stressed that when the discharge has been at right angles to the body surface, the wound is almost circular and symmetrical; in all other situations, an elliptical wound will be traced out, its elongation increased as the angle between them diminishes. This pattern also applies to the spread of soot and powder, giving an easy indication to the direction of fire.

Wound margins may be shelved, the tissues being acutely lacerated below the margins distal to the origin of discharge. This may be better appreciated in the injuries from rifled weapons where the wound has been produced by a single projectile than from the more diffuse mass of pellets. The track of the wound may be traced, which will help in determining the direction of discharge of fire. Here again, a missile from a rifled weapon provides a more clear picture than the shot-mass from the shotgun. However, a general idea can be gathered from the knowledge of the position of the surface wound and the mass of pellets. **Examination by X-rays will avoid the laborious search** for pellets at autopsy (Fig. 16.9).



Fig. 16.9 X-ray of right arm of a lady showing comminuted fracture of humerus and lodgement of some pellets in the adjoining chest cavity. It pertains to a case of a married lady who had an extra-marital affair. One day the paramour visited the lady in absence of her husband. Incidentally, the husband reached home earlier than schedule. On seeing his wife having intimacy with another man, the husband became furious. Without losing time, he (the husband) snatched the shotgun from the paramour and attacked his wife. On autopsy, multiple fractures of skull (produced by the butt of the weapon) and comminuted fracture of humerus (produced by the pellets) were revealed.

Autopsy

Postmortem examination of a firearm victim presents some peculiar problems over and above those of the usual medicolegal autopsy. Needless to say that careful and detailed examination is invited in all such cases with particular attention to collection, preservation and dispatch of certain evidence for the forensic science laboratory. Decomposition of the body, which usually poses problems, will not prevent the recovery of bullet/pellets and also powder residues on the skin or clothing of the victim. In case of decomposition or bodies recovered from water, though it is likely that surface details may have deteriorated, effects of powder or carbon monoxide into the deeper tissues may survive much longer. A case has been reported by Taylor in his *Principles and Practice of Medical Jurisprudence* where the evidence of carbon monoxide colour changes over a radius of some 4–5 inches in the subcutaneous muscles around an entry wound over the mid-frontal chest could be demonstrable in the body of a Pole recovered from a pond several weeks after death. Spitz and Fisher reported that they had observed deposits of soot on the bone in a young woman who had survived 3 months after she had shot herself in the temple.

The examination should include:

- Clothing
- X-ray examination
- Pertinent findings regarding injuries showing:

  - External evidence of injuries
  - Internal evidence of injuries
- Collection, preservation and dispatch of exhibits
- Cause of death

CLOTHING

Examination of a victim of firearm injury is grossly incomplete without a detailed scrutiny of clothing for any defect made by the missile/missiles and for deposition of any firearm residue. The doctor must be attentive to the possibility of finding bullet or some other residues on the clothing. Any distortion of clothing, extent and manner of blood staining, or their smearing with mud/grease, etc. should be noted. Number and location of defects produced by missiles need detailed description. The location of these defects may be described in relation to the distance from collar, seams, pockets, buttons, etc. Several holes may result by a single bullet due to presence of creases in the garments, thereby simulating more than one shot. The defects in the clothing ordinarily correspond with the wounds upon the body, but this may not necessarily be so since the clothing often get disarranged in the process of struggle or in the process of fleeing, leaning or tossing, etc. during the defence or escape, usually seen in acts of firing.

There may be the following objectives for the proper scrutiny of clothing:

- **Helping to establish the range of discharge of firearm:** The extent and manner of distribution of soot and/or powder is obviously indicative of range of fire as already explained. It should be measured to enable the laboratory people to compare with the test-shot patterns. Since the garments may totally filter out these residues of discharge of a firearm, their relation with the body surface is essential so as to have an understanding of the range at which the firearm was discharged.
- **Helping to determine about the wounds of inlet and outlet upon the body:** This may be possible because of deposition of various residues/bullet-wipe surrounding the entrance defect at appropriate ranges. Further, the direction of the bullet travel may be suggested by insertion of the fabric surrounding the inlet wound and eversion at the outlet wound. This may, however, be altered under numerous circumstances.
- **Helping to locate the bullet/missile:** If no exit defect exists in the clothing while it is present upon the body, either the clothing did not cover the area of the exit or the bullet left the body with insufficient velocity to pass through the clothing. The bullet may, therefore, be either lying loose in the clothing or might have dropped during transport or during management in the emergency wing of the hospital where the victim is usually supported/handled by many attendants/relations, etc.

The importance of clothing in cases of firearm victims need not be stressed in cases where unambiguous identification of entry and exit wounds is not possible. Indeed, the task of carrying out an autopsy is lightened by an adequate examination of the clothing before the autopsy is commenced. However, there may be situations outside the control of an autopsy surgeon that may interfere with the proper interpretation of the findings upon the clothing, e.g.:

- The defects made by the firearm may be in the line of the cuts made to open the garments.
- Fragile residues may be flipped off the clothing.
- The area of the defects may be soaked with blood, body fluids, intravenous fluids and the like.

All this, therefore, calls for the examination of clothing by several different techniques. Infrared photography can be used to reveal soot deposits on the dark-coloured or black fabrics. Ordinary X-rays can be applied to search for larger metallic fragments of the bullets and other missiles. Soft X-rays may be employed to demonstrate only mildly radiopaque materials like powder grains, etc. Energy dispersive X-ray techniques can be used to analyse metallic fragments for elemental control.

X-RAY EXAMINATION

Usefulness of X-ray examination of the dead body of a gunshot victim is undeniable, since the missile or more often the pellets may lodge in the most unlikely and distant places. Instances are not uncommon where the bullet entering the shoulder region has been eventually recovered from the pelvis and the like. Therefore, subjecting the body to X-ray examination prior to autopsy will prevent undesirable mutilation and also save the time.

Importance of X-rays is depicted from the following:

- It helps in locating the missiles/pellets, fragments or jackets, etc.
- It helps in determining the track of wound as stressed earlier under 'Direction of Fire'.
- It helps to determine defects in bones in the areas not easily approachable on direct examination.
- It helps to delineate air embolism accompanying large vessel damage by the missile.
- It helps to scan the body in instances of bullet embolism or where the missile has been propelled along the gastrointestinal tract through peristaltic movements.
- It helps to provide documentation that the body was examined.

Use of X-rays to locate a bullet will save valuable time at autopsy. In instances of bullet embolism, X-rays are invaluable in locating the bullet. X-rays should always be conducted even when there apparently exists an exit wound too, because an exit wound does not necessarily indicate that the bullet did indeed exit. A bullet making an exit in the skin can rebound back into the body through the same wound after meeting resistance from the overlying clothing. Moreover, exit can also be due to

a fragment of bone being expelled through the skin while the bullet itself remaining inside the body. A particular situation can arise in case of partial metal jacketed bullets. Here, separation of jacket and the missile can occur as the missile moves through the body. This jacket carries valuable evidence in the form of markings upon its surface and will be available for bullet comparison. Sometimes, both the jacket and core after separation in the body may remain inside the body. These two can be identified on X-rays where they will be distinguishable by different densities.

In through-and-through gunshot wounds, small fragments of metal from the bullet may be deposited along the wound track or in the bone fractured by the bullet. These metallic traces, otherwise invisible, can be analysed by SEM-EDX. If the fragments are large enough, they can be submitted for quantitative compositional analysis by inductively coupled plasma atomic emission spectroscopy. A comparison can then be performed with the missile recovered at the scene and suspected to be the lethal missile.

Occasionally, routine X-rays in deaths from gunshot wounds may reveal old bullet(s)/pellet(s) or bullet fragment unrelated to the death of the victim. Such old bullets are encapsulated in fibrous scar tissue and usually have black colour due to oxidation of lead. Black discolouration can occur in recent bullet, if the bullet has been exposed to the contents of the gastrointestinal tract.

Lesser information can be had by X-rays in case of shotgun wounds. Determination of range cannot be made from the spread of pellets on X-rays because both close-range wounds and wounds of several yards' distance can give similar patterns on X-ray because of billiard ball effect of the pellets on entering the body in close-range shotgun wounds.

However, X-rays have some limitations. The exact calibre of the bullet cannot be determined by use of X-rays. This is due to magnification of the bullet image depending upon its distance from the source of X-rays. Bullets close to origin of X-rays will appear larger and have fainter appearance than those close to the film. Secondly, there may be situations where some artefacts can be misconstrued as bullet. Dislodged crown from a tooth may appear as flattened bullet. X-rays should always be taken while the deceased is fully clothed. This practice will be helpful in revealing bullet(s) that exited the body but got entangled in the clothing.

PERTINENT FINDINGS REGARDING INJURIES

The body should be thoroughly examined to look for the wounds of entrance and exit. If they are multiple, it is advisable to assign them number and describe the wound of entry, the track on dissection and the wound of exit in one section so as to avoid confusion.

Location of each wound should be described in relation to its distance from the top of the head or from the heel as well as from some recognised and fixed landmark upon the body. Hairy areas such as scalp may be shaved to appreciate the wound. Each wound should be described with measurements in respect of size, shape and location. In case of wound of entry, entry hole should be measured first and then the marginal abrasion. The difference in width of the abraded collar at different parts around the wound of entrance is very significant and helps in determining the direction of fire, as detailed already. However, where the wounds have been debrided or extended or otherwise interfered with, the medicolegal evaluation may not be possible. Where there is dispute between entry and exit, the skin and the subcutaneous tissue measuring 2.5 cm × 2.5 cm × 5 mm around the wound of entry and exit, may be excised for examination and packed separately in rectified spirit, labelled properly and sent to the forensic science laboratory under sealed cover. The examination of the tissues at the entrance and from the track of the wound for the evidence of carbon monoxide may be fruitful in some cases. This will almost certainly be higher in concentration near the entrance wound, and this phenomenon may even be recognised after putrefaction or immersion in water.

How to describe the wound? The wound of inlet is usually described as a 'lacerated puncture/penetrating wound' with inverted margins, measuring ($1 \times 3/4\,cm^2$), oval in appearance present on left side of chest, 2 cm below the left nipple. It is surrounded by a rim of abrasion collar measuring ($3 \times 2\,mm^2$), the greater width being on medial/lateral aspect. The presence or absence of blackening/tattooing, etc. should be specially measured and mentioned. Other effects, like muzzle imprint, singeing/burning of the hair, etc., if present, need to be described. The wound of outlet will be written as 'lacerated wound' measuring ($2 \times 1.5\,cm^2$) with everted margins present on the right lateral aspect of front of chest, 4 cm below the 2nd space in the anterior axillary line. However, it is not necessary that the wound of outlet is always greater than the wound of inlet. The reverse may be there under a handful of circumstances that have been described at appropriate places.

Internal Evidence of Injuries (Track of Wound)

Each track must be described separately by layerwise dissection of the tissues. Probes should not be introduced as there is every likelihood of creating false passages and thereby drawing erroneous interpretation as to the direction of firing. The path may be traced from entry to exit or to the lodgement of missile/pellets, etc. Here again may be stressed the importance of X-ray examination of the body prior to conduction of autopsy. Ricochetting of the bullet/shot mass may be kept in mind for proper evaluation. The distance of entry and exit wounds from the respective heels will provide inclination of the track and will help in knowing the attitude of the victim at the time of firing. The path of the missile through the body should be described in relation to the planes of the body, i.e. 'the track passes from front to back or from left to right and somewhat downwards'. Angular estimates with respect to the horizontal, vertical or sagittal planes of the body are also useful in completing the description.

COLLECTION, PRESERVATION AND DISPATCH OF EXHIBITS

Clothing, as described earlier, carry importance and must be handled with particular care because of the possibility of projectiles, powder residues or similar materials being lost by mishandling or rough handling. They must be retained as described and sent to laboratory as detailed earlier.

The bullets/fragments should be recovered as complete and intact as possible, either with the gloved fingers or with rubber-tipped forceps to avoid any scratching or defacing done inadvertently during handling. In the present scenario, the risk is enhanced and made more serious by dangers of blood borne pathogens, viruses and human immunodeficiency virus (HIV), which may occur in blood or body fluids that are present on the bullets. Therefore, in addition to usual precautions, following guidelines may be kept in mind while extracting the bullet/fragments, etc.:

- Double heavy duty gloves should be worn while handling projectiles or other foreign objects.
- Prior radiography should be conducted in order to localise bullets/pellets and to evaluate likely ensuing hazards.
- Rubber-tipped extractor for recovery and handling of the bullets/fragments should be employed.
- Projectile should be examined for any trace evidence, such as fibres, glass pieces, paint, etc. Then it may be dried in open air, if need be.
- Pack the bullet/fragments, etc. into a hard plastic container padded with any soft material like tissue paper or paper towels rather than an envelope to prevent accidental puncture through the envelope and consequent injury.
- Before packing, bullet should be marked on its base for future identification.
- The container should bear the particulars of the case and the warning, 'Biohazard' may be written upon it.

Similarly the pellets, in case of shotgun injuries, should be recovered as many as possible. The collection may present a tedious job of recovery, where again X-raying the body before examination is of paramount help. Cards and wads from shotgun cartridges should be retained and sent in envelopes after drying them in open air and wrapping them in cotton or gauze. The envelope must bear the particulars of the case including the site of location of these exhibits.

Collection of exhibits may also include the analysis of blood for alcohol or some other drug and blood for blood grouping, etc.

CAUSE OF DEATH

The cause of death is usually relatively straightforward, and haemorrhage is by far the most common cause of death in victims of firearm injuries. Total amount of tissue damaged and vascular damage should be considered in evaluation. Here, it may be added that gaping tears of heart may occur where the missile track simply passes through the chest and does not directly involve the heart. Further, death may sometime ensue merely because of concussive effects of the impact, though the missile/slug never penetrates the cranial/chest cavity. At this juncture, **some estimate of the rapidity of death may be made**. Depending upon the organ and blood vessel involved, death from bleeding may occur within a few minutes to several hours.

The limiting factor for consciousness is the oxygen supply to the brain. When the oxygen in the brain gets consumed, unconsciousness supervenes. Experiments have shown that an individual can retain consciousness for at least 10–15 seconds after complete occlusion of the carotid arteries. Thus, if blood supply to the brain is prevented because of extensive gunshot wounds of the heart, an individual may remain conscious for at least 10 seconds before collapsing. Sudden blood loss causes interference with activity when it exceeds 20–30% of the total blood supply. Loss over 40% is assumed to be life-threatening. The rate of bleeding, the amount of blood loss, the nature and extent of injury, individual's prior physical status and, of course, individual's physiological response determines the time from wounding to incapacitation and death. Here, the degree of vulnerability of cells to the lack of oxygen and their potential for recovery needs some mention. As documented, nerve cells are highly sensitive to oxygen and ischaemia; further, there are regional differences within the central nervous system. In total ischaemia, cessation of nerve cell function has been reported to commence in the cerebral cortex after 8–15 seconds and in the brain stem ganglia after 25–35 seconds. Irreparable structural damage occurs in the cells of the cortex after about 3 minutes, in the basal ganglia after 6–7 minutes, and after about 9–10 minutes in the vagal centre. In contrast, myocardial cells have a considerably higher tolerance for oxygen deficiency (this accounts for the sustenance of heart beat for some minutes after complete ischaemia of the brain occasioned through hanging or some other cause).

Suicide, Accident or Homicide?

Traditional autopsy findings ordinarily do not provide enough information to arrive at the conclusion regarding the manner of death. A comprehensive medicolegal investigation including autopsy assessment, however, allows some reliable conclusion to be drawn. It must always be borne in mind that most extraordinary events can occur in medicolegal field and therefore, one must avoid making dogmatic statements. However, there may be certain patterns of injuries including firearm injuries that may either indicate or be consistent with certain methods of causation. A medicolegist must be equipped with adequate knowledge, experience, common sense and, of course, obtuse mind ever ready to consider various possibilities apart from one by which he/she is initially impressed. The distinction between suicide, accident or homicide can be assisted by the following.

CIRCUMSTANCES SHOWING DESIGN (Need to be Examined by the Officials of FSL)

In suicide, there is usually some strong evidence of design; in an accident, it is wanting. Note(s) of farewell or indicating the reason for suicide is/are common and a quiet/solitary spot is often chosen. Some would go into the woods or riverside or to a favourite rendezvous of the past. If some evidence in the form of attempted disposal of the body, tying of limbs or any other such evidence or activity is available, the possibility of homicide gets enhanced. Use of the uncommon weapon or any peculiar contravention applied in an unusual manner will usually suggest suicide. At times, the victim may undertake some unusual, rather flashy and elaborate devices. At an inquest at Southwark, a remarkable wooden frame made by a suicide was produced in the court. Deceased had spent some weeks preparing the frame to hold a rifle steady and a system of levers by which it could be discharged. He fired it through the back of the neck—a rare site of election indeed.

Mostly, the scene of death in firearm injuries is quite bloody. However, this observation is not immutable because at occasions, haemorrhage may be internal (into the chest or abdominal cavity) and, moreover, the clothing(s) may act as a pressure bandage. Furthermore, when the deceased is wearing multiple clothing, blood may be absorbed by the internal layers of clothing so that there is no/minimal evidence of bleeding on the outer clothing. Gunshot wounds of abdomen may get plugged with some omental/mesenteric tissue and thus prevent the bleeding to appear external. Similarly, the wounds of head may get concealed by a bushy haircut and thus escape showing external evidence of haemorrhage.

Photographs of the place where the body is found should be taken with particular emphasis on the position of the body, attitude of the body, position of the weapon relative to the body (if present), any spent cartridge case/bullet(s), presence or absence of signs of struggle, condition of the doors, windows and flooring, etc.; any blood or other stain, footprints, fingerprints on the doors/windows/weapon, etc. If the body is found in the open, a search for footmarks and marks of struggle must be made. Presence of deposition of soot or powder burning/tattooing, etc. upon the hand(s) must be looked at the scene itself and washings/swabs from the skin be obtained to seek propellant residues indicating that the weapon was held by the deceased. Quite rarely, such effects may be recorded on the hands of the individual trying to push the assailant away while he was holding the weapon and thus resulted during a scuffle. The urgency of examining and collecting samples at the scene resides in the fact that any interference with the dead body for transporting it to the mortuary can vitiate/mask/obliterate the findings upon the hand(s). Here, it may be worth mentioning that poorly constructed weapons are more likely to deposit effects of discharge on the firing hand than weapons of good quality, maintained in good condition. Occasionally, gun smoke deposits may be noted on both the hands, especially on the one that was used to steady the weapon while the other pulled the trigger. However, absence of visible effects of discharge of a firearm on the hand(s) of the victim does not necessarily imply that the weapon was fired by someone else. Laboratory tests including neutron activation analysis and scanning electron microscopy-energy dispersive X-ray for propellant and detonator residues are capable of detecting such effects even when none can be seen on gross examination.

EVIDENCE FROM WEAPON'S POSITION RELATIVE TO THE BODY (Need to be Examined by the Officials of FSL)

The weapon, in most of the cases of suicides, is present at the scene usually near or on the body. It may be firmly grasped in the hand through the development of instant rigor, a rare event but sufficiently striking to suggest suicide. The wounds of the brain are thought to be more liable to be followed by instantaneous rigidity. However, rarely, an ultracriminal-minded assailant may leave the weapon at the scene and arrange it so as to mimic suicide. The weapon may be placed in the victim's hand but it is highly improbable that it would be held by the same firm grip as to be expected to develop in an instantaneous rigor following a suicide. Contrarily, sometime the suicide may survive for sufficient period to remove himself from the vicinity of the shooting or to dispose off the weapon. There are numerous examples on the record, as mentioned earlier in this chapter, where even the considerable destruction of brain has been consistent with survival and volitional activity for sometime after the shooting. Thus, absence of weapon at the scene may not totally exclude a suicide. A relative or a friend may remove the weapon in an attempt to escape the stigma of suicide in the family. Rarely, if the suicide is committed on the bank of a river or pond, the weapon may have fallen into water. In a case reported in the Taylor's *Principles and Practice of Medical Jurisprudence*, 12th ed., a soldier was found dead on his back on the bank of a pond, the feet dangling. Initial impression appeared that the back of the head had been swept away by some heavy blunt instrument, but later it transpired to be blown off by a rifle discharge through the mouth. The weapon had slid between the legs to sink out-of-sight into the pond.

EVIDENCE FROM THE SITE OF ENTRANCE WOUND(S)

A suicide intending death will tend to aim at the area that he believes to be important to life and its destruction will lead to death. Therefore, the majority aim at the so-called 'sites of election', namely, temple, chest (precordial region), forehead, mouth or under the chin. Abdominal region is distinctly less common since an ordinary person appears to be aware of the fact that the outcome is less certain. The majority of those who choose the head aim at the temple and since the majority are right handed, the right temple is the commonest choice. However,

this proposition of right handedness or left handedness need not be strictly adhered to. Suicides rarely, if ever, shoot themselves in the eye or the back of the head/neck.

It is not, however, to be assumed at once that presence of entry wound at the popular site of election negatives homicide, for an assailant may naturally try to copy the same features. A man might be so stupefied with drink or sleep, etc. as to allow the assailant to place the muzzle of the weapon within the mouth or at any other suicidal site. Lack of knowledge or lack of resolution on the part of a suicide or accidental slipping of hand may sometimes cause a wound to be placed at a site where we least expect it.

EVIDENCE FROM THE SEVERAL WOUNDS

Though it is usual for only one shot to be fired in a suicide, yet there may be many exceptions. Even several wounds, each appearing to be immediately fatal, can be inflicted in quick succession. Sometimes, spasm may cause the trigger to be pulled again, if the weapon is self-loading or automatic. Therefore, the appearance of more than one mortal wound usually favours homicide but does not categorically exclude suicide, particularly in the present set-up involving sophisticated automatic weapons. Further, multiple firearm wounds are hardly likely to be accidental. In this concern, it may be added that one bullet may sometimes produce several wounds either by splitting itself or making the bone to split with which it strikes or sometimes by ricochetting or because of peculiar attitude that the victim was assuming at the time of firing.

EVIDENCE FROM THE DIRECTION OF THE INTERNAL TRACK

The bullet generally has a tendency to travel in a straight line from the point of entrance to the point of exit/lodgement/deflection, so that if the internal track is straight that indicates the direction in which the barrel of the weapon was pointed when fired. High velocity bullets are more easily deflected from their original course, even by slight obstacles and remarkable damage may result from the physical forces disseminated. The deflection of the projectile may occur not merely when they come in contact with the bone, but even by meeting the skin, muscle or tendon, etc. As reported, a bullet that entered at the back of the left shoulder passed around the inside of the scapula and was found below the right ear. In judging the direction taken by the track traversing the chest from front to back, it is necessary to give due allowance to the difference that exists in the level of the same rib anteriorly and posteriorly. Tilting the body or angling the discharge may accentuate the difference. Further, the possibility of the body, being in an abnormal position/attitude at the time of discharge, may also be kept in mind. Thus, a stooping person may be shot in the back with the direction of the wound from above downwards by a person standing in front of him. Another caution to be observed in examining the track of the firearm discharge is to be taken care of in case of splitting of the bullet. The mechanism of splitting of bullet is hard to understand, particularly in the jacketed bullets barring its striking against the hard object like bone. But this may not always be necessary and the projectile may disintegrate from the effects of its own centrifugal forces.

EVIDENCE FROM RANGE OF FIRING

Range of firing is of vital importance in eliminating suicide. Thus, a contact or near-contact wound upon the 'site of election' points strongly towards suicide. If the distance or 'range' of the discharge is beyond arm's length, it raises presumption of homicide unless some peculiar steps/device had been adopted by the victim to effect suicide from a distance for which the evidence of arrangements may be available. Homicidal **contact** or **near-contact** wounds may be incurred during hand-to-hand scuffle involving a handgun or may also be encountered where the victim was incapacitated by disease, drug/drink or was asleep or under restraint (as happened in case of prisoners of the Second World War). Presence or absence of soot and powder marks upon the hand of the victim must be looked for and the swabs/washings procured at the earliest possible opportunity as stressed earlier. It is imperative, therefore, that in ascertaining the probable range of fire, account must be taken of factors like modifying factors for reducing the length of the barrel and vice versa, travelling of the shot first through some other medium (may be a door, glass, etc.) and then striking the victim, ricochetting of the projectile, balling of shot, idiosyncrasy of the weapon, the kind of ammunition and the target area of the body involved since in close-range firing of the head, dense hair may prevent the soot/powder particles to reach the scalp and this may obstruct any attempt for evaluating the distance of the fire. In case of doubt, the tissue from the margin of entrance wound(s) including hair should be sent to the FSL as such for chemical and microscopical analysis of the effects of discharge of a firearm.

EVIDENCE FROM CLOTHING

A word about involvement/noninvolvement of clothing also deserves mention. Suicides frequently avoid shooting through clothing and often pull them aside and shoot through bare skin (it would be most unusual for an assailant to open the victim's clothing and shoot him). Rarely, firearm wounds may voluntarily be inflicted for the purpose of imputing murder or extorting charity. A man intending to commit suicide by firearm but failing in the attempt may also, from shame or desire to conceal his act, attribute the wound to the hands of an assassin. Such wounds should be interpreted carefully with due consideration to the features likely to be encountered in self-inflicted/self-suffered wounds.

Some pointers towards accidental circumstances of the discharge may be furnished as under:

- As far as accident or suicide by firearm is concerned, it is lesser in women as such weapons are not easily accessible to them and they are less used to their operation.

- Sportsmen, farmers and hunters may be involved.
- Farmers/peasants are notoriously careless with firearms, often leaving them in loaded condition where they are accessible to children and others.
- Some home-made/country-made guns are extremely unsafe because of poorly constructed mechanism and may get discharged with slight stimulus.
- Accidental firearm wound may be received under the circumstances when the weapon is discharged at a distance without any possibility of human injury but the bullet hits the victim after ricochetting or the victim suddenly appears on the scene.
- Rarely, a person may accidentally shoot himself while cleaning the weapon which he may fail to check carefully.
- Another rare situation may be one where the individual may be carrying a weapon with hand(s) lying close to the trigger while travelling in a vehicle and the weapon gets discharged accidentally on sudden application of brakes of the vehicle. A case was conducted by the author where a security person, while sitting on the side seat of the open cabin of the jeep and holding a weapon in between his legs, got accidentally injured upon sudden application of brakes at some busy crossing (Fig. 16.10).
- Still another peculiar instance may be where the weapon gets discharged of its own during a bomb explosion aimed to kill some particular person. Heat and shock wave produced in an explosion may result in discharge of firearm being carried by some person in the vicinity.

Surgical Artefacts in Firearm Wounds

Surgical intervention may make interpretation of gunshot wounds difficult as a result of obliteration or alteration of wounds.



Fig. 16.10 Photograph showing wound of entrance with surrounding blackening and tattooing. (The policeman was going while sitting on the side seat of gypsy, holding the weapon in between the legs. The trigger accidentally got pressed on sudden application of brakes of the vehicle).

In gunshot wounds of chest, the surgeon may insert a chest tube or in gunshot wound of the neck, he may make incision for thoracotomy **(as happened in Kennedy's case)**. In gunshot wounds of the head, he may obliterate or alter the wound by performing craniotomy. Wide debridement of the wounds by some surgeons may cause concern for the doctor conducting autopsy. Such tissue must be sent to pathology for examination.

Here, it may be pointed out that surgeons must be cautious about the findings upon the clothing, i.e. defect(s) produced by the bullet(s), any evidence of soot and/or powder deposition and exercise utmost care in preserving/interpreting them. If the surgeon happens to recover bullet from the body, he/she must inscribe his/her initials either on the nose or base of the bullet, thus preserving the rifling characteristics. In case of shotgun injuries, wadding and representative pellet(s) should be retained by the surgeon for evidentiary purposes.

Cases

BULLET ENTERED THROUGH FOREHEAD FOUND EMBEDDED IN SCALP

On 8th June, 1998, at about 8.30 p.m., two young boys were sitting and gossiping in a car. Two other boys, having some previous enmity with them, appeared at some distance and raised a '*lalkara*'. Those in the car could not restrain themselves and reacted in a hostile manner. Surprisingly, the other party opened fire, and the bullet hit the victim on the forehead making him unconscious there and then. The friend of the victim who was sitting beside him also fired in air and in the meantime the other party escaped. The victim was rushed to a hospital, but was declared 'brought dead'. Postmortem was conducted the next day by a team of doctors led by the author. The interesting features of the case were (Fig. 16.11):

- An oval shaped typical wound of entrance measuring $0.8 \times 0.6\,cm^2$, just above and to the right of root of nose.

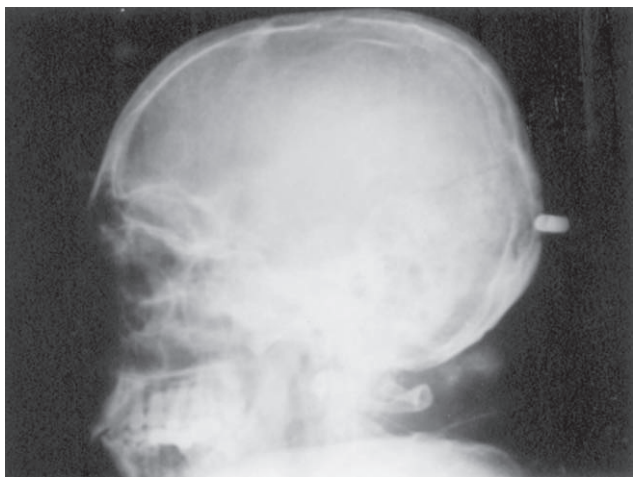


Fig. 16.11 X-ray photograph showing bullet in the scalp.

Marginal abrasion was evident. A defect in the underneath frontal bone, measuring $0.9 \times 0.7\ cm^2$ was present.

- An exit was present in the occipital bone (left side) in the form of irregular defect in the bone, measuring $2.6 \times 2.00\ cm^2$. Brain tissue was emerging out of this defect, and two chips of bone were lying loosely attached to the margin of this defect.
- The bullet was found embedded in the scalp tissue with nose projecting towards the cranium, i.e. after exiting through the skull, owing to much decreased velocity, it got reversed and lodged in the scalp tissue being incapable of making its exit through the scalp (Fig. 16.11).

ACCIDENTALLY DISCHARGED CARTRIDGE HITTING ANOTHER CARTRIDGE—DUAL EFFECTS BEING DRIVEN INTO THE BODY OF THE VICTIM

In March 2006, an unusual case relating to firearm death focused the headlines of the newspapers. As per the information gathered from news items, a security guard (SG) was sitting on a chair with another friend sitting next to him on another chair. He received a call and started responding to the same. Incidentally, he asked the friend to take hold of the weapon (shotgun—a smoothbore weapon) as he was engaged in conversation on the phone. Swayed by the usual curiosity, the friend started meddling with the weapon, which consequently went off. The discharge, after shattering the arm of the chair on which the SG was sitting, hit him at his waist on the right side, causing the discharge of another cartridge contained in the magazine file tied around the waist, and effects of both discharges were driven into the body of the SG, leading to his death shortly afterwards.

The case invites following points of medicolegal interest helping to reconstruct the theory of accidental discharge and death of the SG due to dual effects:

- Demonstration of pellets in the higher abdomen (Fig. 16.12) speaks of upward track of the pellets (in consideration to the entry wound being present on the right side of the waist of the deceased, clothes showing corresponding defects), fitting into the theory of discharge coming from the side on which the friend was sitting on another chair.
- Presence of single entrance wound showing nonspread of pellets at the time of entry and availability of two wads from the body of the deceased indicates close or near-range discharge of the firearm (wads may be recoverable from the depth of the entrance wound or from the body up to a variable distance, usually up to about a couple of yards. Often the wad assumes a lower trajectory and may strike the body below the shotgun wound. It may penetrate the skin causing a separate wound or may only bruise the skin).
- Widespread of the pellets in general appearing in the X-ray photograph (probably represents the population of pellets released from the discharge of the main/primary shot) and a separate cluster of pellets (probably represents those released through hitting the cartridge contained in the magazine file tied around the waist of the deceased) lend support to the theory of the dual effects under reference. However, dogmatic opinion as to which pellet belongs to which effect/discharge is not advisable since under such situations dispersion of pellets in an undetermined manner may occur. Further, it invites caution in interpreting 'range' of the shotgun pattern within the body through evaluating spread of pellets demonstrable in the X-rays (both close-range wounds and wounds of several yards' distance can give rise to similar patterns on X-rays).
- The following evidence collected from the site of occurrence was highly informative in helping to trace the events:
  - Blackened portion of front end of the right armrest of the chair through which the discharge had travelled,
  - Availability of broken pieces of the plastic chair,
  - Pattern of some pellets scattered on the ground under and around of the broken chair,

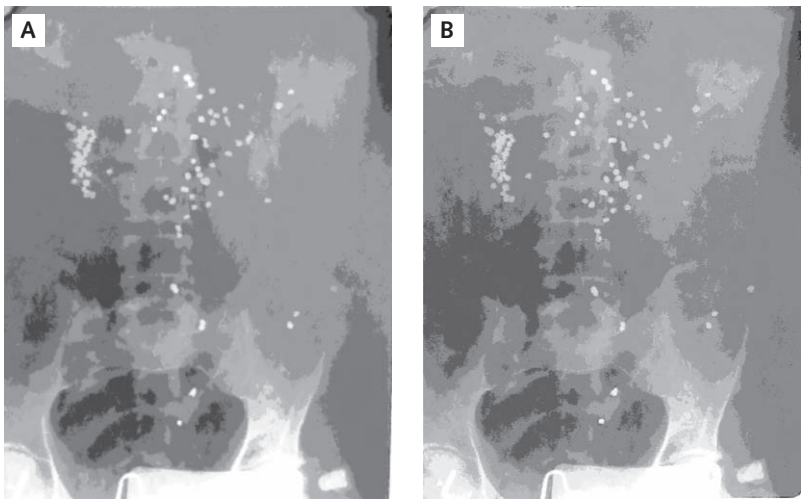


Fig. 16.12 Spread of pellets in (**A** and **B**) abdominal and adjoining region.

- Presence of shotgun in front of the chair,
- One fired 12-bore shotgun cartridge case, and
- One metal head portion of a 12-bore shotgun cartridge, etc. The belt carrying ammunition that was worn by the SG at the time of the incident showed one big hole and a few other small holes over the cartridge pockets of the belt with corresponding holes/marks on the bodies of the 12-bore shotgun cartridges.

(Communication from SS Baisoya, Scientific Officer, Central Forensic Scientific Laboratory, Chandigarh.)

SETBACK TO THE SCIENTIFIC EVIDENCE DUE TO NONCONSENT OF THE VICTIM

In February 2005, a well-publicised case flooded the newspapers. In this case, the victim (an adult male) was shot from across the road (a distance of around 6 feet) while he was parking his car. The report (DDR) was lodged in the police station which was later transformed into FIR under Section 307 IPC (attempt to murder) and Section 25 (punishment for shortening the barrel of a firearm or converting an imitation firearm into a firearm without due licensing) plus Section 27 (punishment for using arms without license or using prohibited arms/ammunition, etc.) of Arms Act. The victim was admitted to the hospital. Attending doctors of the emergency wing of the hospital handed over a sealed packet to the police containing sweater, shirt and vest smeared with blood. The same were presented by the police to the board of doctors conducting the medicolegal examination of the victim for correlating the holes in these clothing with those made by the bullet injuries on the body. The victim was identified to the board of doctors by the police and the treating surgeon. The treating surgeon also opined as to the fitness of the victim to make statement. The board of doctors after noting down various injuries advised radiography of the whole body. X-rays of the chest and abdomen showed presence of three bullets, location being like this: One lodged in left side of chest outside the bony cage, just opposite to the 6th rib. Another lodged in the abdominal region, just above the tenth rib (Fig. 16.13). Another lodged in the abdominal wall opposite lumbar 3rd vertebra (not appreciable in the photograph).

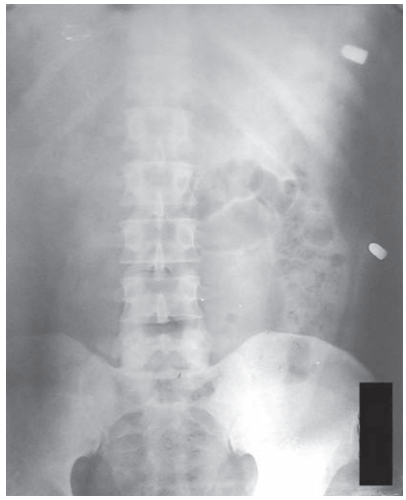


Fig. 16.13 Superficial lodgement of bullets in the chest and abdominal regions.

As per news items, the victim was brooding against the police saying that the State Agency was behind the incidence as his name was being voiced in some crime against the State. To rule out this, the nature of weapon involved in the crime assumed utmost significance and hence, the reference of the case to the laboratory.

Examination and comparison of the individual characteristic marks present on the **spent cartridge cases** (bullets remaining lodged in the body due to nonavailability of victim's consent to remove the same were not available for the examination) under comparison microscope helped in furthering the opinion as to the singularity or otherwise of the weapon(s) as far as possible.

(Communication from SS Baisoya, Scientific Officer, Central Forensic Scientific Laboratory, Chandigarh.)

17 CHAPTER

Injuries by Explosives

After going through this chapter, the reader will be able to describe: Mechanism of production of injuries by bomb blast | Autopsy in explosion fatalities | Medicolegal considerations in explosion injuries

The recent upsurge of terrorism for political and other purposes in many parts of the world has brought with it the use of explosives. It seems that in the general political unrest, which is prevalent in the world, the bomb will continue to be used to reinforce direct and indirect political objectives and therefore, a medicolegal expert needs to be conversant with some basic knowledge about the effects contributing towards injuries/death, etc. Identification of the material used in the manufacture of the bomb and mechanism of its explosion, etc. are the domains of the forensic scientists.

Most of our knowledge of explosions has been gained through wartime events. There have also been some notable explosions affecting civilians such as the one in Texas City in 1947 when a ship loaded with ammunition exploded at the docks killing about 560 people and injuring over 3000. **Following an explosion, a person can be injured/killed in a number of ways:**

- If he is quite near to the explosion, he can be blown to pieces.
- He can be injured by a wave of pressure, called the 'shock wave', which spreads concentrically from the seat of the explosion. When the explosion is in air, the pressure wave is referred to as air blast.
- He can sustain 'flash burns' from the momentary heat radiation or, if his clothing or other material is set on fire, he can sustain ordinary burns.
- He can be struck by 'flying missiles' propelled by the explosion.
- He can be injured or crushed by debris, usually of building(s) demolished by the explosion.
- He can be overcome by fumes generated as a result of the explosion.

The above factor(s) may operate solely or in varying combinations, and the relative importance of each will depend upon the type of detonation, the distance of the victim from the seat of explosion and the location of the explosion. Each factor is being discussed.

Disruptive Effects

If the victim is almost in contact with a large bomb, usually when he is carrying it or sitting with it in some vehicle, he may be blown to pieces. A premature explosion, sometimes during the act of setting the timer, may cause disruptive injuries. With smaller explosions or when the victim is a few feet away, disruption is limited to the blowing off of head or limb or the mangling of a localised area (Fig. 17.1A and B). Therefore, sometimes a part of the body may be totally destroyed, while the remainder of the victim being remarkably intact. The pieces can get scattered over an area of 100 metres or more from the seat of explosion. Many parts of the body may never be found having mixed with the masonry and other debris of the blast site.

Air Blast (Shock Wave)

A blast comprises a wave of compression, which spreads concentrically from the blast centre. The velocity of the shock wave depends upon the distance from the epicentre, being many times the speed of sound in the air at the start but rapidly decreases as it spreads out. This wave of compression/high pressure is followed by a weak wave of negative pressure (below atmospheric), so that a rapid double change in pressure is suffered by the victim. The magnitude of the blast varies with the energy released and also with the distance from the epicentre. As the distance from the explosion increases, the peak pressure falls rapidly, almost exponentially. About 100 lb/sq inch (690 kPa) is the minimum threshold for producing serious damage to human beings.

Effects of Blast Wave/Shock Wave

The high pressure shock wave generated by an explosion can knock a person down and thus cause injury but the specific injury



Fig. 17.1 Photograph of bomb blast victim showing: (**A** and **B**) damage caused by bomb blast; (**C**) X-ray photograph showing some part of device in the chest.

associated with blast is due to the shock wave being propagated through the body. **It causes most damage at an interface between tissues in contact with the atmosphere and that is why the lung is usually the worst sufferer.** The shock wave can pass through solid homogenous tissues like muscle and liver, causing little or no damage but in the lungs the damage is caused owing to marked variation in density between the alveolar walls and the contained air so that damping of shock wave occurs leading to disruptive effects. Its transit through the lungs can tear the alveolar septa and give rise to alveolar haemorrhage. Other findings in the lungs may include subpleural patchy haemorrhages (often in the line of ribs) and intrapulmonary haemorrhages. The air passages may be filled with bloody froth causing airway obstruction and hypoxia in addition to the primary damage. Later, neutrophilic reaction may develop around the haemorrhagic areas and those can progress onto bronchopneumonia. The pulmonary injury is a specific injury of the air blast and is sometimes called as **'blast lung'.** However, the lungs can also be bruised by direct blows on the chest, and haemorrhagic areas can arise by aspiration of blood or regurgitation of stomach contents down the trachea. Rarely, when the victim dies soon after the explosion of a bomb, this finding may not be seen, presumably due to relatively small amount of explosive detonated and the victim being somewhat away from the seat of explosion so that the blast wave is unable to exert any serious effect.

Blast may also cause **damage to the ears**. Its effects tend to be capricious, because the pressure on the tympanic membrane is modified by many factors but when the pressure rises excessively above the atmospheric, rupture is likely.

Gastrointestinal system often suffers from the effects of a blast because like lungs, it contains air and gases and is thus not a uniform medium for the transit of shock wave. The caecum and colon are more often hurt than the ileum, jejunum and stomach, presumably because they are larger and often contain more gases. Occasionally, ruptures of the gut can occur if the blast is violent and the victim is situated nearby.

Burns

When a bomb explodes, the temperature of explosive gases can exceed to 2000° C, and the heat radiated momentarily can cause **'flash burns'**. The amount of thermal radiation received decreases with the square of the distance from the explosion and the intensity of explosion.

The burns sustained are usually extensive and mostly affect the exposed areas of the body. Areas protected by a footwear or a brassiere tend to be spared as do areas shielded from radiation by solid objects. The body contours also exert shielding effect so that the front of the chin is burnt but the part underneath is usually spared. After death, burnt areas become reddish brown and parchmentised.

Objects in the vicinity and the clothing may be ignited and the victim is then burnt by contact with the flame. These burns usually involve irregular areas of the skin to a different degree, and this feature differentiates them from the flash burns. Other burns may be caused by ignition of building material or vehicle catching fire from the effects of bomb or from gas or petrol ignition, etc.

Flying Missiles

Although the blast is the specific hazard of an explosion, it is only important when some large explosive device has been used or the victim is virtually adjacent to the lower energy bomb. Smaller explosions usually injure and kill by propelling solid objects/materials in all directions. The fragments may originate from the bomb-casing or container or from the vehicle in which the bomb was concealed.

Fragments may vary in size, ranging from tiny splinters to large chunks, which are projected at high speed. The smaller ones may not be able to travel longer but larger, heavier fragments

can fly over considerable distances and may cause serious or fatal damage in just the same way as missiles from a firearm.

In the open, debris is scoured away, which can impinge upon the body to injure and discolour the area of the body. A more common appearance is that of a sort of **'peppering'** resulting from numerous small missiles/fragments producing varying sized/designed abrasions, bruises and puncture lacerations of varying sizes and depth, intimately mixed on the skin. Some of the puncture lacerations may contain fragments of metal, stone, wood or a piece of clothing. Metallic fragments usually are of interest to the forensic scientists because they can be pieces of the bomb mechanism. **This triad of injury is usually considered to be diagnostic.** While abrasions and bruising can occur beneath clothing, dust tattooing usually remains confined to exposed skin showing abrupt demarcation close to the areas like collar or sleeve, etc.

Sometimes, the explosion might be specifically meant to propel missiles as with the hand grenade, the casing of which is specially designated to fragment into shrapnel and the nail bomb in which many nails are bound round a stick of gel ignite.

Falling Masonry

When a bomb demolishes a building/porch, etc., the persons inside the building or underneath the porch receive multiple injuries from the collapsing structures; on many occasions, these injuries may be the only effects of the explosion on the body. The victims are often heavily soiled by blood, dirt, dust and oil, etc. In some cases, there may be signs of **crush asphyxia** (purple discolouration of upper part of body with petechial haemorrhages into the skin and the conjunctivae; some congestive haemorrhages from the ears and nose may also be present).

Fumes

Sometimes, explosion fractures a gas-main and the people injured or trapped get poisoned before they are extricated. Poisoning by fumes is most prominent in mine disasters. Gaseous products, called 'after damp', usually comprise carbon monoxide, carbon dioxide and hydrogen sulphide.

Autopsy in Explosion Deaths

Postmortem examination of an explosion victim involves the following major objectives:

- Identification of the victim(s).
- Enlisting the injuries.
- Cause of death.
- Medicolegal considerations.

IDENTIFICATION OF THE VICTIM(S)

Usually, a major initial problem is to discover how many bodies are involved and to try to allot the correct fragments to the right individuals. Where there are a number of victims and the small fragments are scattered over a wide area, the task may be extremely difficult or impossible. However, this is largely an anatomical exercise, similar to the sorting out of multiple skeletal remains. **Complete body X-rays of the victim(s) are imperative before the clothing is removed.** Fragments of the bomb may be trapped within the body tissues or the clothing (Fig. 17.1C). Clothing must be retained for chemical analysis, since this too may reveal the presence of some trace evidence with respect to the type of explosive used. However, if the victim was quite close to the explosion, his/her clothing might have been blown off by the blast and may be recovered in shreds at a considerable distance from the victim. Victim in such cases may be found partly or completely nude. Tight articles such as a belt, a buttoned collar or lace-up shoes are commonly retained on the body.

Apart from assisting in locating the trace evidence pertaining to the explosive device as detailed above, radiology will also go a long way in detecting other radio-opaque objects/findings like stone(s) or pacemaker or some old fracture/bony changes that the alleged victim was known to have. The dentition and artificial teeth can also help considerably in establishing identity if a recent dental record is available. This aspect of identity has been dealt with at length in the chapter on 'Identification'.

Finger, printing must never be omitted wherever possible, since it can prove or confirm identity in many cases. Even if the victim's prints are not available in the police records, prints can be compared with those on articles handled at work or at home once the person's identity has been suggested.

ENLISTING THE INJURIES

The external as well as internal lesions must be described in detail. If possible, photographs may also be taken. Nature and extent of external injuries has been mentioned above in detail. The diagnostic triad, i.e. varying sized/designed abrasions, bruises and puncture lacerations intimately mixed on the skin, has already been highlighted. This is produced by the flying missiles including splinters of wood, stone, dust, dirt, etc., as outlined above. Signs of crush asphyxia may be characteristically found when the death occurs due to some falling masonry. Internally, damage to the lungs, gastrointestinal tract, ears, etc. is more common. The mechanism of their production has already been described in detail.

CAUSE OF DEATH

Death may result from a variety of causes depending upon the nature and intensity of explosion, the distance of the victim from the seat of explosion and the location of explosion, i.e. whether in a confined space or in open. The body may be completely disintegrated as a result of blast effect when the victim is in

the vicinity of the blast. If the victim is at some little distance away from the explosion, death may result from burns, blunt force injuries and falling debris. Crush asphyxia may be the cause in some cases dying of being buried under falling masonry. At times, death may occur due to inhalation of toxic fumes, especially in mine disasters.

Sometimes, the victim may die within a short period after an explosion with no more than a slight injury and no contributory disease. Some of these deaths may be due to systemic air embolism from air, which has gained access to the pulmonary veins after blast-damage to the lungs. In other rapid deaths, it appears that death is due to profound circulatory changes resulting from lethal reflexes, the so-called **'blast shock'**.

MEDICOLEGAL CONSIDERATIONS

Injuries from the explosions are usually accidental. Homicidal cases are infrequent. (A time bomb may be left at some place to coincide with someone's arrival at a particular time when it may explode.) Alternatively, an impact bomb may be thrown or left at a venue of a meeting where it may explode as a result of friction. Of late, human bombs are being used for attaining specific political ends.

Reconstruction of the scene and circumstances of death can be gathered from the type, severity and distribution of the injuries upon the body. Various pointers, as given below, may help in this direction.

Explosive Force Declines Rapidly

As stressed in the beginning, the intensity of blast varies with the energy released and the distance from the seat of explosion. The velocity of the shock wave is many times the speed of sound in the air at the start but rapidly decreases as it spreads out. Therefore, for a person to be blown to pieces, he/she must be in contact with the bomb, i.e. either carrying the bomb, sitting with it or arming it. Persons can be injured by flying missiles and collapsing structures when at distances from the bomb.

Explosive Force is Extremely Directional

The parts of body directly exposed to explosive force are most often involved, i.e.:

- Explosion at ground level usually injures lower legs and feet.
- When the person is bending over the bomb, the face, chest, waist and upper limbs may be blown away.
- Legs may be blown off or the abdomen disrupted or the hands and arms torn away in a person who was implanting the bomb.
- If the bomb explodes at the back of a person sitting in a chair, injuries are likely to be distributed on the back of legs, thighs and on the back of the trunk.
- At occasions, bomb may go off prematurely whilst being made, in the transit, whilst being planted, while setting the timer or while being diffused, causing localised injuries.

Such localised severe trauma may be able to assist in the reconstruction of the events as it indicates the relative position of the bomb and the victim at the time of detonation. This was unambiguously exemplified in a sensational political killing, where the perpetrator was allegedly carrying explosive around his waist (the so-called human bomb) and thereby had undergone remarkable disruption of the upper and middle portions of the body. Only lower legs were available from the scene, which went a long way in helping towards identification (from DNA profiling).

Case: Assassination of Rajiv Gandhi and the Birth of 'Human Bomb'

Rajiv Gandhi was the ninth Prime Minister of India from 31st October 1984 until his resignation on 2nd December 1989 following defeat in the general elections. He remained Congress Party President until the elections in 1991. While campaigning, he was assassinated by Liberation Tigers of Tamil Eelam (LTTE) group. When he reached the venue, he got off his car and began to walk towards the dais to deliver the speech. Along the way, he was garlanded by many party workers and school children. At 10.10 p.m., the assassin Tanu approached him and greeted him. She then bent down to touch his feet and detonated an RDX explosive laden belt tucked below her dress. Rajiv Gandhi, along with many others, was killed in the explosion that followed. The assassination was caught on film through the lens of a local photographer whose camera and film were found at the site. The cameraman also died in the blast. Certain medicolegal aspects emanating from such scenarios may be as under:

- **Identification** is usually extremely complex in large scale explosions that cause mass casualties with dismemberment or fragmentation of the body (see text). This was a typical feature in the instant case.
- **Histopathology** may help in detecting injuries caused by shock wave being propagated through the body, lung tissue being the worst sufferer. The injury is sometimes called as blast lung (see text). Myoglobinuric renal failure resulting from crush syndrome is another entity diagnosable through histopathology.
- **Blood tests** for carboxy haemoglobin, cyanides, and phosphorus may be necessary, particularly when the blast has occurred in closed space or in fire-related blasts.
- **Explosive residues** need to be collected and dispatched for subsequent examination by experts in the field of explosives.
- The possibility of **contamination of the body** with chemical or radio-active material needs to be kept in mind at the time of conducting autopsy.

18 CHAPTER

Regional Injuries

After going through this chapter, the reader will be able to describe: Injuries of the scalp including forensic aspects of anatomy of the scalp | Fractures of the skull including forensic aspects of anatomy of the skull | Mechanism of production of skull fractures | Meningeal haemorrhages with their medicolegal aspects | Mechanism of production of cerebral injuries | Medicolegal aspects of coup and contrecoup injuries | Concussion | Head injuries in boxers | Spinal injuries with their medicolegal aspects | Facial, cervical, thoracic and abdominal trauma

Of all the regional injuries, those of head are most common and account for about one-fourth of all deaths due to violence, and responsible for 60% of fatal road accidents. Even in the author's own series, head injury cases comprised of 69.5% of all the fatal road traffic accident cases. Reasons for their dominance, as furnished by Adelson, are listed below:

- The head is the target of choice in the majority of assaults involving blunt trauma.
- On being pushed or knocked to the ground, the victim usually strikes his head.
- The brain and its coverings are vulnerable to that degree of trauma as would rarely prove fatal, if applied to other parts of the body.

The underlying approach of this chapter is to deal with the most common problems of forensic concern rather than to discuss the subject from the clinical aspect. The diagnosis and treatment of head and spinal injuries are considered in the modern textbooks of neurology and neurosurgery.

Head Injuries

'Head injury', as defined by the National Advisory Neurological Diseases and Stroke Council, "is a morbid state, resulting from gross or subtle structural changes in the scalp, skull, and/or the contents of the skull, produced by mechanical forces". To be complete, however, it should take into account that the impact, responsible for the injury, need not be applied directly to the head.

A couple of important dicta should always be remembered in relation to craniocerebral injuries, which would prevent any unnecessary theorising among the doctors as well as lawyers. These are as follows:

- Any type of craniocerebral injury can be caused by any kind of blow on any sort of head.
- No form of craniocerebral injury is too trivial to be ignored or so serious as to be despaired of.

SCALP INJURIES

Scalp is often, though not invariably, damaged in the trauma that causes injury to the underlying skull and/or brain. In order to appreciate the injuries efficiently from the medicolegal angle, anatomy of the various layers of scalp is being furnished as follows:

Forensic Aspects of Anatomy of the Scalp

The scalp is the portion of the soft tissues of the head extending from the eyebrows anteriorly to the superior nuchal line posteriorly and laterally from one temporal line to the other. Its primary function is to protect and insulate the skull. The scalp consists of five layers of tissues arranged in the following order (Fig. 18.1):

- The skin
- Dense connective tissue
- Galea aponeurotica
- Loose connective tissue
- Periosteum (pericranium)

The skin is normally hair-bearing, a feature that enhances protection and insulation. The dense connective tissue layer can further be subdivided into fatty layer and a deeper membranous layer that contains the major feeding vessels of the scalp. Due to the density of the subcutaneous tissue, inflammatory

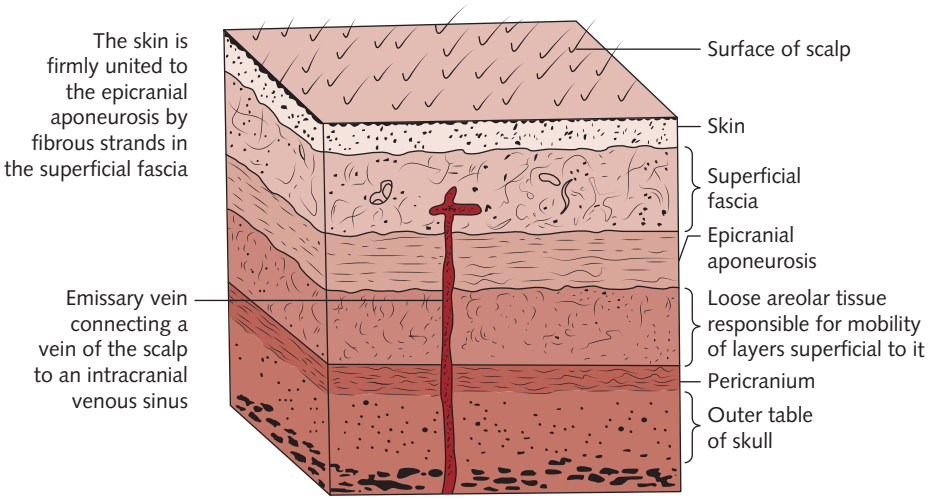


Fig. 18.1 Sketch to show the layers to the scalp.

swelling is slight. Contraction and retraction of the arteries is impeded by this tissue, and haemorrhage from the scalp wounds is often copious. The galea, a freely movable aponeurosis of dense fibrous tissue, is structurally designed to absorb the force of external trauma. It is pierced by numerous emissary veins that connect the veins of the scalp with the intracranial venous circulation, providing an easy pathway for the propagation of infection from the scalp to the intracranial structures. The layer of loose connective tissue between the galea and the periosteum has been aptly termed as dangerous layer of the scalp. The loose composition of the connective tissue permits collection of blood or pus in conjunction with the local haemorrhage or infection. It is through this layer that avulsion occurs and surgical exposures are made. The thickness of the scalp in adults is variable, ranging from a few millimetres to about a centimetre, depending upon the location of the head, age and sex of the individual. In infants, the thickness may be less, but the scalp is highly elastic. Scalp thickness increases with age so that by puberty it approaches the thickness of the adult scalp. From the traumatological point of view, it forms the first barrier to the impact and serves to widen and lower the peaks of transient impacts. The intact scalp over the skull increases resistance to skull fracture by nearly ten times, as has been observed in experimental models. Similarly, presence of mat of hair over the impact site also affords an added protection.

Scalp Abrasions

Abrasions are less common than on other sites because of the presence of thick hair, which also tend to prevent or blur the patterned effect of blunt force injuries. Abrasions, although minor injuries in themselves, may carry medicolegal importance out of keeping with their lack of severity and may be the only representation of some severe deep-seated lesion. **The following case amply substantiates this:**

Two young boys entered into altercation with a middle-aged person on account of a wrongly parked car. Heated exchanges were soon followed by blows causing the middle-aged man to fall on the *pacca* pavement, striking the side of his head. He immediately became unconscious and was transported to hospital, where he was declared dead after sometime. Injuries, present on the person of the deceased, were:

- $0.75 \times 0.5\,cm^2$ abrasion on left temporal region at the junction of the upper part of pterion.
- $0.5 \times 0.5\,cm^2$ abrasion over the front of left knee.
- Subdural haemorrhage over the left temporal region.

The deceased, a Sikh gentleman, was wearing turban at the time of assault. The presence of turban along with thick long hair of the scalp probably prevented severe surface injuries. The case, however, sends a wave of caution, viz., any external injury of the head, even if per se insignificant, may constitute important medicolegal evidence and may be the only clue towards some graver damage underneath.

Scalp Bruises

Bruising of the scalp may occur anywhere. It is usually difficult to be detected because of the presence of thick hair. The only appreciable evidence may be the swelling, as the spilled blood is incapable of extending downwards owing to the presence of bone underneath. After death, difficulty in detecting a bruise may further be enhanced as swelling gets diffused. Commonly, deeper bruising in relation to fibrous galea beneath the skin becomes visible on dissection of the scalp. The bleeding may often be followed by marked oedema, and layers of the scalp may be greatly swollen and thickened by a jelly-like infiltration of tissue fluid. Blood may get collected beneath the pericranium, as is often found in infants receiving head injuries with fractures of the skull. In relation to contusions of the scalp, it has been observed that they are **better felt than seen**. It is always advisable to palpate the entire scalp and shave the suspected area for better appreciation of the bruise.

Bleeding under the scalp may be mobile, particularly under gravity. Thus, a bruise or haemorrhage under the anterior scalp may slide downwards to appear in the orbit, simulating a black eye from direct trauma. **Black eye** (bruising of the eyelids) should be differentiated from blood seeping passively into the orbit. A black eye may be caused by:

- Direct trauma such as punch upon the eye.
- Gravitation of blood over the supraorbital bridge from an injury on the frontal area.
- Entrance of blood into the orbit from behind or above, due to a crack in the walls of the orbit, usually a fracture of the roof of the anterior fossa of the skull (such fracture is often produced from a contrecoup injury caused by a fall on to the back of the head, leading to the secondary fracture of the quite thin bone of the orbital roof).

Scalp Lacerations

Scalp lacerations may be found in association with bruising and abrasions and double or triple lesions may frequently be present.

Lacerations of the scalp are **classically confused with incised wounds** due to splitting of the tissues as the scalp is being sandwiched between the hard underlying skull and the external blunt impact. Distinction between the blunt splits and knife slashes may be difficult but usually possible by careful examination of the margins of the wound and, if need be, examination under the magnifying glass. Presence of foreign bodies like a piece of glass, a piece of stone or fragment/trace of some other material will lend an additional help in determining the kind of weapon involved. A laceration in the scalp is usually characterised by the following:

- Bruising of the margins, although the zone may be narrow
- Head hair crossing the wound are not cut
- Fascial strands, hair bulbs, nerves and vessels, running in the depth of the wound, are irregularly torn.

Many factors influence the formation and appearance of lacerations upon the scalp, such as the contour of the object delivering the force (whether blunt object/instrument/weapon or fist or shod foot or any part of the vehicle), the type of the tissue, position of the body and the velocity of the impact. For instance, a blow on the scalp is far more likely to cause laceration than a blow of similar violence on the abdomen or buttocks, where bruising is more likely to result.

Scalp lacerations **may bleed profusely**. In lacerated wounds of the scalp, the temporal arteries may spurt as freely and forcefully as when may cut cleanly. These arteries being firmly bound are unable to contract and may, therefore, spurt and continue to bleed for a relatively longer period. In a quarrel with her husband, a woman sustained several injuries on her face and head. One of these was a lacerated wound on the right temple. Blood stains were found on the ceiling at a distance of four feet from her bed. They were caused by the spurting of the divided right temporal artery. A young man had been struck on the right temple causing a lacerated wound. Blood spurted to a distance of three feet and a quarter from the place where he was standing at the time of the assault (Peterson, Haines and Webster, *Legal Medicine and Toxicology*, 2nd ed., Vol. I, 294).

Lacerations of the scalp **may follow the pattern of the inflicting object**, though a random splitting is more common leading to stellate, linear, Y-shaped, V-shaped or crescent-shaped appearances. Severe impacts from shaped objects like hammer or some other heavy tool with specific striking area may reproduce the profile of the weapon totally or partly. A blow with an 'angle iron' may provide a resembling shape to the wound imparted by the angle of the metal, just as the etched lines of a file will leave a replicated imprint in the skin where it strikes. Under some situations, where the victim has been kicked or 'stomped', replica of the pattern of a heel may be produced on the scalp. It is obvious that proper documentation of these injuries, including photography, may be of immense help to the law enforcement agencies in linking an assailant with the crime, by comparing patterns of shoes, belts and/or other confiscated weapons to the impressions/marks on the victim.

When the injuries are due to fall(s), the pattern(s) may be highly variable. There may be no laceration of the scalp or there may be simple linear tear or jagged wound, etc. However, in some cases, the falling victim may strike a projecting object such as the edge of a table or a stone/brick lying on the ground/floor. These **'interfering objects'** may produce lacerations or even patterned injuries, which might lead to misinterpretation. Under such circumstances, the witness account and an examination of the scene may provide the background information for proper analysis. Dirt/sand/pieces of stone/brick, etc. may be carried into the wound and might be detected with the aid of ultraviolet light in the gross state or by scanning electron microscopy/polarising microscopy in the tissue specimen. Such findings may carry particular significance in lacerations following a street brawl because a question may arise here—whether the laceration occurred due to a blow or a fall. However, one must keep in mind that an agent/weapon may bear grit or dust and thus soil the wound or else the victim may fall after receiving a blow. Furthermore, site of laceration may also be a material factor at such occasions.

Laceration(s) of the vertex of the skull are mostly the result of fall from a height or striking the area against some projection; for example, when the victim suddenly stands from a stooping or kneeling posture and strikes his head against the corner of a mantle piece or a door of an open cupboard. In other circumstances, the wounds of the vertex are almost certainly inflicted by an assailant.

Incised Wounds of the Scalp

These wounds may be produced by cutting instruments such as a *gandasa*, a spade, a *khurpi*, an axe, a sword, a hatchet, a shovel or a chopper. The wound margins and the tissues running in

the depth of the wound will be helpful in determining the nature of the weapon, as stressed earlier.

The edges of the wound produced by heavy cutting weapons may not be as smooth as those of wounds caused by light cutting weapons like razor or knife, etc., and often show bruising of the margins. If the wound is inflicted obliquely, there will be bevelling of one edge of the wound, which may be helpful in indicating the direction of application of the force. While, if the sharp edge is struck almost horizontally, it produces a wound with a flap.

Wounds of the scalp usually heal rapidly, though in occasional cases fatal results may ensue from the supervention of infection or suppuration may set in and spread into the brain through the emissary veins or through the necrosis of the bone resulting from infection or through a neglected fissured fracture. Thus, cases have been reported where scalp wounds had apparently healed, and yet, death ensued from septic meningitis or brain abscess, after a few days or weeks.

SKULL INJURIES

Forensic Aspects of Anatomy

In discussing the different patterns of skull fractures, Burns arrived at the conclusion that if all skulls were equally thick and equally elastic, the lines of fracture could be calculated on mathematical formulas. In reality, the skull is not a homogenous body, but is composed of panels of bone that differ in thickness and elasticity from individual to individual, and in the same individual in the different portions of the skull. The thickness of the calvaria ranges in adult from 3 to 6 mm. It is thin in the squamous portion of the temporal bone and much thicker in the midfrontal, midoccipital, parietosphenoid, and parietopetrous buttresses. The skull is somewhat thinner in females than in males, and the outer table is always thicker than the brittle inner table. Bone density also varies. Areas of decreased density are frequently seen in the frontoparietal region, in the neighbourhood of the coronal suture, above the roof of the orbit, and in a small segment above the internal occipital protuberance. In contrast, an area of increased density is usually present between the squamous portion of temporal bone and the parietal bone. This explains how skull fractures, although subject to some extent to the laws of mechanics, are so varied and unpredictable.

In foetus, skull consists of fibrous membrane that becomes ossified through a process of cellular differentiation (intramembranous ossification). Ossification starts in individualised centres that make their appearance around the 7th week. In early infancy, the bones of the skull are thin and pliable, and the differentiation between inner and outer tables can hardly be seen. A distinct inner table does not become apparent until the age of 2 years. Patency of the fontanelles adds further protection from trauma. The anatomical configuration and its relatively smaller size in proportion to the skull capacity permit the infant brain to withstand greater trauma than would be possible later in life. As Jackson says, "in an infant, a blow that would perhaps fracture an adult skull often produces only a dent, like that seen in damaged ping-pong ball".

With closure of the fontanelles and union of the sutures, the skull becomes a rigid cavity that gradually enlarges from a capacity of about 350 ml at birth to 1400 or 1500 ml at maturity. With advancing age, partial closure of the sutures takes place, and in the later decades of life, it is not uncommon to find complete bridging of at least some of the sutures. The considerable variations in the sequence with which obliteration of the sutures takes place further prevent prediction of the effects of trauma.

In contrast to the vault, the base of the skull presents many jagged areas. In the anterior fossa, lesser wings of the sphenoid, the cribriform plate of the ethmoid bone and the crista galli represent threats to the integrity of the brain when it is pushed forward in accelerated motion. In the middle fossa, equal threats are provided by the clinoid processes and in the posterior fossa by the foramen magnum.

Skull Fractures

More than one forensic meaning is assigned to the term fracture. As usually used, it implies a break or disruption of bone. Surgical classification of types of fractures has little forensic import. 'Simple fracture' and 'open or compound fracture' are the usual surgical terms. The former refers to a fracture of the bone with intact skin overlying it, and the latter refers to the fact that the fracture site has an open pathway to the atmosphere or that the ends of the fractured bone have penetrated the overlying skin.

It has been reported that in one of four fatal head injuries, skull escapes fracture. The practical implication is that radiological evidence of absence of skull fracture is no indication as to absence of any injury to the brain. The presence of skull fracture is, however, an indication of the severity of force applied to the head.

Mechanism of Skull Fracture

The subject has been extensively studied by Gurdjian, Webster, Lissner and Rowbotham. These and other authors observed as follows:

- When skull receives a focal impact, there is momentary distortion of the shape of the cranium. Infant skulls, which are more pliable and have flexible junctions at suture lines, may distort much more than the more rigid skulls of adults. The area under the point of impact bends inwards and as the contents of the skull are virtually incompressible, there must consequently be a compensatory bulging of other areas, the well-known 'struck hoop' concept. Both these intruded and extruded areas can be the site of fracturing, if the distortion of the bone exceeds the limits of its elasticity.
- In more common circumstances of a wider impact from blunt injury, deformation of skull is less localised but, where the force is sufficient, fractures can still occur from

the same mechanism of exceeding the elastic limits. The fractures may be remote from the area of impact or may accompany the focal depressed fracturing as described.

- When the focal impact is severe, the depressed fracture may follow the actual shape of the offending object, such as a hammer head. The shape may follow only that part of the object that drives into the skull; for example, the circular head of the hammer may strike at an acute angle, so only a part of the circumference of the weapon may operate and produce a corresponding punch in the bone.
- The presence of hair and scalp markedly cushions the effects of a blow, so that a far heavier impact is required to cause the same damage, compared to a bare skull. The pattern and nature of the skull fractures are, however, the same. Here, it may also be worth mentioning that skull fractures may sometimes be caused without any contusion or any other wound on the scalp, though there may be extravasation of blood on its undersurface, as the force of violent impact may be cushioned by multiple layers of a *pugree* or abundant growth of hair on the head.

Types of Skull Fractures

Basilar Fractures Basilar fractures are relatively frequent and often radiologically occult. The relative frequency of such fractures may be attributed to irregular shape and presence of several foramina, making the base of the skull relatively weak. At autopsy, dura needs be stripped thoroughly from the basal calvarium so as to verify or exclude such fractures. **Anterior fossa fractures** are usually due to direct impact. A heavy blow on the chin sustained in boxing may transmit the impact through maxilla to the base of the skull and may result in contrecoup fracture of the cribriform plate of the ethmoid (see under 'Contrecoup Fractures' also). Blood, in such cases, may spread along the tissue planes around the eyes, resulting in peri-orbital ecchymoses that resembles black eyes/spectacle haemorrhage/raccoon eyes (raccoon is an American nocturnal mammal having a distinct peri-orbital colouration). However, **the former** arises from head injury with internal bleeding, whereas **the latter** results from bruising of the orbital and peri-orbital tissues from direct impact injury. Such fractures usually manifest by escape of blood and cerebrospinal fluid (CSF) from the nose (CSF rhinorrhoea). **Middle fossa fractures** usually result from direct impact behind the ear or crush injuries of the head and are followed by escape of blood and CSF from the ear (CSF otorrhoea). Occasionally, it may cause an arteriovenous communication between carotid artery and cavernous sinus. Mastoid haemorrhage from a fracture of middle cranial fossa may be confused with retroauricular scalp bruise, called as Battle's sign (William Henry Battle, a surgeon at St. Thomas Hospital, London, 1855–1936). **Posterior fossa fractures** commonly result from direct impact on the back of the head, for example, striking the back of the head on the ground. It may be followed by escape of blood and CSF into the tissues of the back or neck. Fractures around the foramen magnum, especially the ring fracture, have been described ahead. Sometimes, a fracture extends transversely across the middle region of the base of the skull, along the region of the petrous ridges. The two components/fragments may be able to be brought together and displaced, as if on a hinge. This is referred to as a **hinge fracture**. The common mechanism for its production is severe hyperextension injury of the neck. Such fractures are commonly associated with injuries of the brain stem, especially pontomedullary tears.

Linear Fractures Also called 'fissured fractures', these are linear cracks without any displacement of the fragments and may involve whole thickness of the bone or one or the other table only. They are notoriously difficult to be detected and may not be demonstrable by X-rays. The line of fissured fracture is like that of a hair's breadth and usually follows a devious course along the line of dissipation of the force.

Linear or fissured fractures are likely to be caused by a forcible contact with a broad resisting surface like the ground, blows with an agent having a relatively broad striking surface. When the blow is struck on the side and the head is free to move, the fracture usually starts at the point of impact and runs parallel to the direction of the force. If the head is supported when struck, the fracture may start at a counter pressure; for example, in bilateral compression, the fracture often starts at the vertex or at the base. In case of a blow over the head and subsequent fall resulting in linear fractures, fracture lines produced by the fall are usually arrested by those produced by the blow. Similar may be the situation if two blows are struck one after the other.

In children and young adults, a linear fracture may pass into a suture line and cause **'diastasis'** or opening of the weaker seam between the bones. In infants, particularly in the child abuse syndrome, a linear fracture of a parietal bone may reach the sagittal suture and continue across it into the opposite plate. The continuation may be direct or may be 'stepped', i.e. the two fractures are not in line.

Depressed Fractures Depressed fractures usually result from focal impact of a moving object on the cranial vault. The area struck is driven along the same line of force into the subjacent structures; the depth varying according to the velocity with which the impact is delivered. Thus, an object moving at a high velocity, such as high-powered projectile, will not only perforate the skull but may also cause fragments of the bone to be driven into the substance of the brain. In contrast, any blunt object moving at a lower velocity, such as a hammer or a brick, may create only a simple area of depression that absorbs most of the energy.

Rarely, only the inner table may get fractured and the outer remain intact, and vice versa may also be true. A violent blow with full striking area in operation, such as with a hammer, may detach almost the same diameter of the bone, which is driven inwards, thus often producing a pattern consistent with the offending object. This is why these fractures are also called **'fracture signature' or 'signature fracture'**. A less violent blow

or an oblique blow may produce a localised fracture with only partial depression of the bone. A glancing or tangential blow or a grazing bullet may produce gutter cum depressed fracture, with or without comminuted or fissured fractures.

Impacts with axe or chopper, etc. may leave characteristic lesions in the bone, whether skull or elsewhere. The shape of the fracture produced by such weapons may, to some degree, reveal the direction from which the blow was struck. This is particularly true when a chopping instrument is applied. The undermined edge of the fracture defect is the direction in which the lateral force vector is exerted, and the slanted edge is the side from which the force was transmitted.

Comminuted Fractures Here, the bone gets broken into multiple pieces and they usually occur as a complication of fissured or depressed fractures. The fragmentation of the depressed part of the bone occurs, which are often driven into the subjacent structures. They may be produced in vehicular accidents, or by repeated blows, more or less over the same area, by weapons having relatively small striking surface.

When there is no displacement of the comminuted fragments, the area looks like **spider's web or mosaic**, with fissured fractures radiating for varying distances along the line of dissipation of the forces. But when the violence applied is enormous, the comminuted fragments may get disturbed and, in fact, some of them may be recovered from the surface or substance of the brain.

Pond or Indented Fractures These may be seen in infants where the skull is elastic and usually is produced by forcible compression of the skull by obstetric forceps or impact against some protruding flat object. Fissured fractures usually occur around the periphery of the dent. The fracture is in the form of indentation or simple in-buckling of skull.

Gutter Fracture It is the name used to indicate a furrow in the outer table of the skull, ordinarily the result of a glancing blow by a missile from a rifled firearm. These are frequently accompanied with comminuted depressed fractures of the inner table of the skull.

Ring Fracture This is a type of fissured fracture that encircles the base of the skull around the foramen magnum, usually running 3–5 cm outside the foramen magnum at the back and sides of the skull, passing forward through the middle ears and roof of the nose.

Such types of fractures are usually noticed in the following cases:

- Fall from a height on to feet or buttocks, when the force of fall is transmitted upwards through the spinal column.
- Vault of skull being driven against the spine by falling of heavy load over the vertex or fall from a height on the head or heavy blow over the vertex.
- Violent twisting of the head on the spine, shearing the vault from the base.
- A heavy blow directed underneath the occiput or chin causing the fracture by violently lifting the skull from the spine and thereby breaking it away from its basal attachment.

Separation of Suture (Diastatic Fractures) Diastatic fractures are those in which the fracture line involves separation of one or more cranial sutures. These are most often seen in children and are commonly associated with epidural haemorrhage. They occur as a result of large/broad impact to the head with the blows, falls, industrial/vehicular accidents or under circumstances where the victim, usually a child, is swung by legs against a wall or other immovable object.

Expressed Fractures These are rather uncommon but may occur as massive fragmentation/shattering of skull where the pieces may come to lie outside the normal curvature of the cranium in the pericranial tissues, in the orbits, or physically outside the head. Such fractures can occur due to massive trauma often involving contact/close-range firearm injuries or injuries due to blasts.

Contrecoup Fractures These are mostly seen in orbital portions of the frontal bones as simple linear fractures or sometimes in more complex form as stellate fractures. Bilateral orbital contrecoup fractures are uncommon but may rarely exist as separate fractures. These fractures presumably arise from the pressure differentials between the intracranial orbital surface and the intraorbital space as in occipital falls or heavy blows at the back of the head. The involvement of frontal region may be explained because of development of **'negative pressure'** within this region resulting from differential movements of brain versus skull following occipital impact that leads to implosion of the relatively thin and weak orbital roof. It is unlikely that sufficient forces can be built up in other areas of the skull so as to permit implosion fractures, but presence of some pathological condition or some unusual situation may permit contrecoup fractures to occur elsewhere.

While evaluating the presence of skull fracture at the autopsy, care should be taken against indiscrete use of chisel and hammer. It is preferable to stripe away dura, especially to appreciate linear fissured fractures at the base of skull. Tapping of the skull to elicit a 'cracked pot' sound is a time-honoured and still beneficial method for appreciating the skull fractures.

MENINGEAL HAEMORRHAGES

The extreme fragile nature of the contents of skull invites their closure in the strong bony box of the cranium. Damage may occur either to the neural tissue or to the vasculature, which surrounds and penetrates the neural tissue.

Forensic Aspects of Anatomy of the Coverings of the Brain

The brain is invested in three separate layers of tissue. The outermost layer, **dura mater**, is formed of two layers of tough

collagenous tissue, the external layer of this dura being firmly in apposition with the inner surface of skull and the internal layer merges with the arachnoid. Between the skull and dura, there is a potential space, the so-called epidural or extradural space, which carries considerable forensic importance. The dura forms the falx cerebri and the tentorium cerebelli, and the cranial venous sinuses run within this dura. Polypoid invaginations of the dura penetrate the inner walls of the venous sinuses to form the 'arachnoid granulations'.

The arachnoid is a thin vascular meshwork, which is closely applied to the inner surface of the dura. The name has been derived from the Latin term for the spider because of the spider-web appearance of the tissue. The arachnoid closely follows the contour of the brain but does not dip like the pia mater. Separating the arachnoid layer from the dura is a space termed as the subdural space. Further, arachnoid is separated from the underneath pia mater by a space known as subarachnoid space. This space is filled with cerebrospinal fluid, and the width of the space varies from few millimetres in the young to a centimetre or so in the old where there has been development of cerebral atrophy. (CSF is produced by the choroid plexus of the lateral, third and fourth ventricles. The fluid leaves the ventricles through a small opening in the roof of fourth ventricle, called the foramen of Magendie and the lateral foramina of Luschka and circulates through the subarachnoid space towards the pacchionian granulations, from where it joins the venous blood in the dural sinuses.)

The pia is not a true membrane but is a surface feltwork of glial fibres, which are inseparable from the underlying brain. The layer has little forensic importance.

Any force that succeeds in deforming the skull or changing the position of the brain in relation to the skull may produce damage to the meninges, the cerebral or meningeal vessels and nerves and may contuse and/or lacerate the brain substance or sometimes may only induce a neuronal injury of microscopic dimensions. In fact, many disorders of the central nervous system caused by mechanical trauma are due to injury to the accessory elements, i.e. meninges and blood vessels, and the changes in the nervous tissue are of secondary nature.

Bleeding or haemorrhage may occur in any of the three spaces discussed earlier under the 'Forensic Aspects of Anatomy of the Coverings of Brain'. If the bleeding is small and thin-layered, it is called 'haemorrhage' and if it is in the form of space-occupying lesion because of its large mass, it is termed 'haematoma'. According to the relationship of these haemorrhages to the meningeal coverings and the brain itself, they can be studied under the following subheadings:

Extradural (Epidural) Haemorrhage

Bleeding between the inner surface of the skull and the dura mater is the least common of the three types of brain membrane haemorrhages. Generally, the haemorrhage is associated with linear or fissured fracture of skull that crosses the grooves of the meningeal vessels on the inner surface of the skull. About 15% haemorrhages may occur in intact skulls (Mc Kissock). Only in persons with rather elastic skulls, especially in children, a skull deformation may separate dura and cause extradural bleeding without a skull fracture being present. It may occur in association with the subdural haemorrhage. Usually, it is unilateral but bilateral epidural haemorrhages have also been reported. There were only three bilateral haemorrhages in the 175 cases reviewed by Mc Kissock et al. (1960).

Cause and Source

Rupture of the middle meningeal artery or its branch or the accompanying veins or both is the most common cause, and this explains why the region most often affected is the temporoparietal area. Less commonly, the posterior meningeal artery near the foramen magnum or the anterior meningeal artery near the cribriform plate may get involved and consequently the site of the haemorrhage may be parieto-occipital or fronto-temporal. However, it has been claimed that almost all ruptures take place at a site where the artery is roofed over in a bony tunnel so that it is unable to escape damage from a fracture but as stressed in the beginning, responses can be varied. These haemorrhages are rare during the first 2 years of life due to greater adherence of dura to the skull and the absence of bony canal for the artery.

Other sources of bleeding in this space are the emissary veins and the dural sinuses, mostly the sagittal and lateral. Haemorrhage from diploic venous channels and lakes may also occur but rarely becomes large enough to be significant.

As bleeding commences, it strips off the dura from the undersurface of the skull with progressive accumulation of blood. There is often a free interval of varying duration probably related to a delay in the onset of bleeding due to spasm of the injured artery. This latent interval **(lucid interval)** may not occur if the concussion is prolonged or there is associated brain damage. About half an hour may be sufficient to form a significant arterial haematoma but in Rowbotham series, the range varied from 2 hours to 7 days, but most were apparent after 4 hours.

Subdural Haemorrhage

Subdural haematomas tend to occur most commonly in fifth and sixth decades as compared with epidural haematomas that peak in the second and third decades. Further, subdural haematomas have a less clear association with impact injuries than do the epidural ones. In fact, there need to be no impact upon the head, as it can sometimes occur in infants solely from vigorous shaking. Subdural haemorrhage is probably the most common lesion in fatal child abuse, being that described by Caffey in the classic early descriptions of the 'battered baby'. Acute, subacute and chronic varieties are recognised, but only acute and chronic deserve description because a clear distinction exists between their clinical features and medicolegal importance.

Acute Subdural Haematoma

It is an acute accumulation of blood in the subdural space, being almost always traumatic in origin. Subdural haemorrhage, unlike extradural, is essentially venous in origin and the **various causes may be following:**

- **Rupture of the bridging or communicating veins:** Bridging or communicating veins traverses the subdural space to drain into the parasagittal sinuses, but those present on the inferior surface of the brain drain in the sinuses at the base of the skull following injury. Rupture may occur in case of rotational movement of the brain in relation to the skull, in acceleration or deceleration injuries, without any injuries of the scalp or fracture of the skull. The locations where these communicating or bridging veins are most frequently encountered include the lateral frontal region, the apex of the temporal lobe and the subtentorial region. Lack of muscle fibres and thinness of fibrous walls and elastic lamina predispose these categories of veins to rupture as the brain slides within the skull. Furthermore, it has been reported that parasagittal bridging veins have viscoelastic properties that govern the vessel rupture and depend upon the rate at which the vessels are strained and the direction of strain. Yamashita and Friede have shown that bridging veins appear to be ultrastructurally stronger circumferentially than longitudinally and, therefore, are more resistant to displacements than elongating strains.

  The lesion is often solitary, being associated with the closed head injury where the only other sign may be the bruising of the scalp or even nothing at all—as when an infant is violently shaken.
- **Tears in the dural venous sinuses,** following a blow.
- **Laceration of the dura and tear of middle meningeal artery,** with bleeding occurring into subdural but not in epidural space.
- **Fresh tear occurring in an old adhesion between the dura and the brain** with consequent bleeding.

As the name implies, this lesion is an acute accumulation of blood at the interface between the dura and arachnoid membranes. It is mostly unilateral. Not infrequently, it is associated with injury to the underlying brain substance. Blood tends to accumulate in the base of the skull, especially in the middle fossa. Its distribution will be determined by the position of the head, blood collecting by gravitation in the then dependent part of the skull. In the acute form, blood usually is red, partly fluid and partly clotted. If sufficient interval elapses between injury and death, a fibrous membrane usually spreads over the inner surface of the clot, enclosing it. This layer is usually detectable at about 10 days.

On most occasions, bleeding is slight but fatal compression of the brain by a large subdural haemorrhage can occur within a few hours. It has been suggested that about 100–150 ml is usually the minimum associated with fatalities. Fatality is frequently associated with some concomitant brain injury. If there is no primary brain damage, the mortality from the subdural haemorrhage is usually related to the victim's age, neurological status and delay from the time of trauma to the surgical evacuation of the haematoma.

Chronic Subdural Haematoma (Pachymeningitis Interna Haemorrhagica)

These haematomas blur with the subacute subdural haematomas of older age, but may form a distinct phase when a cellular organising membrane gets formed over the undersurface of the haematoma. Such haematomas are more often encountered in the old persons and in chronic alcohol abusers. The factor responsible may be the increasing subarachnoid space that occurs with diminution of brain size in old age. This increased space with corresponding decrease in the size of the brain allows greater movement of the brain within the cranial vault, even with incidental acceleration/deceleration. Another factor playing a part is the pseudo-elongation of the cortical veins leaving the cortical surface to enter the venous sinuses which, therefore, are likely to be under strain and thus more susceptible to tearing.

An amount of subdural blood insufficient to cause a mass effect may accumulate following minor trauma. This is especially prone to occur in victims with cerebral atrophy due to reasons described above. Although small amounts of subdural blood are usually spontaneously reabsorbed, the haematoma may occasionally become encapsulated by a membrane of fibrous tissue and friable capillaries emanating from the dura mater. Small recurrent haemorrhages from the thin-walled vessels within the membrane cause collection of liquefied blood to enlarge. Another explanation for this enlargement may be that as the membrane envelops the haematoma, it becomes semipermeable to water. The contents of haematoma become significantly liquefied by about 2–3 weeks, and is said to contain high levels of proteins and are, therefore, hypertonic to surrounding tissues. This hypertonic fluid compartment, encased in a semipermeable membrane, enlarges as the water moves into it, to dilute the liquefied clot still further. This chronic subdural haematoma may come to clinical attention months or years after the initial insult when it presents as an intracranial mass and may create features of brain compression ultimately leading to death.

Organisation of Subdural Haemorrhage

The subdural space has no mesothelial lining, and its walls have a limited absorptive capacity, due to which reparative reaction to the presence of blood in it is unique. Further, a subdural haematoma being located beneath the dura, transmits its compressive forces fairly equally onto the gyri and sulci, resulting in an 'undulating' appearance of the compressed surface of the brain, whereas the epidural (extradural) haematoma being located outside the dura, pushes on the thick and fibrous dura, transmitting the compressive forces evenly over a large flat surface area, resulting in an appearance described as 'ruler-straight' surface of the compressed brain. Grossly, acute subdural blood

appears as a maroon coloured film of blood or gelatinous clotted mass that can readily slide off the leptomeninges on surface of the brain. As the subdural blood autolyses and becomes organised, following changes, reportedly, may be demonstrable **microscopically** (these changes need be interpreted cautiously and not rigidly, as there can occur variation in the evolution of changes from individual to individual. At autopsy, detailed description and photographs may invite documentation):

- Within a couple of days or so, macrophages migrate to the area and engulf red blood cells and therefore, haemosiderin is identifiable through iron stains.
- Macrophages and haemosiderin gradually become more prominent as the organisational process progresses.
- Within a week or so, endothelial cells form capillaries and the granulation tissue begin to thicken considerably. Early fibroblastic membrane, the so-called neomembrane (composed of fibroblasts, macrophages, and collagen) is formed. This membrane originates from the dura at the edge of the haematoma, spreads over the inner (i.e., nondural) surface of the clot, interposing itself between the clot and arachnoidal surface.
- After 1–2 weeks, granulation tissue gets more organised with abundant young fibroblasts, macrophages, and blood vessels.
- Eventually, the autolysing blood gets resorbed and a well-developed membrane of fibrous tissue shows its appearance, a development usually requiring an interval of 3–4 weeks. (The centre of the haematoma is likely to show predominantly autolysing blood and therefore, one must obtain sample from the edge of the lesion as the organisational changes here are most prominent and predictable.)

Medicolegal Considerations

As with other injuries, the mechanical cause is the change in the velocity of head, either acceleration or deceleration, almost always with a rotational component. Where a blunt impact is given to the head, subdural bleed need not be situated directly under the area of impact or on the same side of the head. Secondly, it is quite mobile and therefore a lesion originating high on the parietal area may drain down under gravity and cover varying portion of the hemisphere and may even go into the posterior fossa through the tentorial opening.

As in the extradural haemorrhage, there may be lucid interval (latent interval) before clinical signs and symptoms appear. Associated brain damage may, however, cause uninterrupted coma from the time of injury. When there is lucid interval, it may be longer than the average 4 hours of faster arterial bleeding of the epidural haemorrhage. In fact, there is no upper limit to this interval as the acute subdural haemorrhage may merge into chronic condition, which may recur after weeks or even months. In rare cases, they may develop as fast as an extradural haematoma and become fatal by the same mechanism of brain displacement within hours.

Chronic subdural haematomas provide a fertile field in forensic pathology and for legal profession because of special character of this lesion. It frequently occurs without known trauma or other historical cause, often evolves silently, mimics a number of other conditions and is easily missed clinically. Therefore, linkage of haemorrhage with the temporal event and the appropriateness and timeliness of therapy or the lack thereof may become the focus of attention for medical negligence suits, insurance claims and also in criminal cases.

Sometimes, when a collection of recent blood is discovered inside an obviously old subdural haematoma, controversy may arise—whether the recent blood deposition is due to recent trauma. However, it may be kept in mind that it is a part of natural history of such lesions that they bleed of their own accord. In such cases, it is important to determine if there are any other signs of recent traumatic lesions in the brain.

Explanation for sudden decompensation and death in the individuals carrying subdural haematoma may be sought in the rather delicate equilibria existing in the intracranial space amongst the cerebral volume, cerebral blood flow, CSF volume and intracranial pressure. When haematoma has achieved its maximum size—which can be accommodated by egress of CSF, by adjustment of CSF production, transport and absorption as well as by compensatory shift of brain structures—any additional mass effect because of new haemorrhage may be disastrous leading to evolution of coma and death within hours.

Subarachnoid Haemorrhage

It is the most common intracranial lesion observed following blunt trauma to the head and occurs almost invariably with cerebral contusions and lacerations, but shows mixed aetiology (Table 18.1). Following are the usual causes, traumatic as well as nontraumatic:

- Nontraumatic subarachnoid haemorrhage:
  - Rupture of an aneurysm of an artery supplying the brain
  - Rupture of an intracerebral haemorrhage of nontraumatic origin (apoplectic haemorrhage or stroke) into the subarachnoid space.
- Traumatic subarachnoid haemorrhage:
  - Direct trauma to the brain with focal areas of subarachnoid haemorrhage
  - Trauma to the side of the face and neck with fracture of a cervical vertebra with tearing of the enclosed portion of a vertebral artery
  - Tearing of one of the thin-walled arteries at the base of the brain due to sudden hyperextension of the head upon the neck.

Acute Nontraumatic (Spontaneous) Subarachnoid Haemorrhage

Spontaneous subarachnoid haemorrhage is almost always due to rupture of a berry aneurysm, though at occasions the origin of the haemorrhage may be difficult to detect if the rupture and consequent haemorrhage has destroyed the greater part of the

Table 18.1 Salient Features of Epidural, Subdural, and Subarachnoid Haemorrhage

Features	Epidural (extradural)	Subdural	Subarachnoid
Location	Between skull and dura	Between dura and arachnoid	Between arachnoid and pia
Cause	Head injury	Mostly due to injury (massive leakage through meninges can also occur)	*Natural:* aneurysm, high blood pressure, angioma *Traumatic:* cerebral contusions, damage to internal carotid, vertebral or basilar artery
Confusing entity	Can be confused with heat artefact	Seldom confused with other bleeding	Can be artefact from rough opening the skull
Aetiology	Mostly middle meningeal artery or its branches are ruptured	Mostly due to rupture of bridging (communicating) veins that traverse the subdural space to drain into the parasagittal sinuses	Due to natural vessel leakage from vessels on brain surface, or vessels from within brain, or from injury
External manifestation	Often blood under the scalp	Often no external manifestation	No external manifestation unless other injuries are present
Gravity	Can be space occupying	Often space occupying	May be space occupying if source is arterial
Distribution	Usually on one side but can be both	Unilateral or bilateral	Focal, semi-localised, diffuse, or bilateral
Brain surface	Being located outside the dura, it pushes on the thick and fibrous dura transmitting the compressive forces almost evenly over a large flat surface area resulting in an appearance, the so-called 'ruler straight' appearance of the compressed surface of the brain	Being located beneath the dura, it transmits its compressive forces fairly equally onto the gyri and sulci resulting in an 'undulating' appearance of the compressed surface of the brain	Brain surface usually not distorted

aneurysm (berry aneurysm—a saccular aneurysm of the cerebral artery usually at the bifurcation of the vessels in the circle of Willis. Its narrow neck of origin and larger dome resemble those of a 'berry', hence the nomenclature. Thomas Willis, an English anatomist and physician, 1621–1675). The aetiology of saccular aneurysms is uncertain. However, some genetic factors are considered to be important in their pathogenesis. Cigarette smoking and hypertension are expected predisposing factors for their development. Although they are sometimes referred to as congenital, aneurysms are not present at birth but develop overtime owing to the underlying defect in the media of the vessel wall. They may occur singly or multiply and may rupture spontaneously or upon head trauma. Even the emotional upset that accompanies trauma (in fact, the blow may never be struck, but only threatened) can trigger cardiovascular changes such as sudden increase in blood pressure, precipitating rupture of the aneurysm. It has also been forwarded that berry aneurysms seem to rupture more often in intoxicated persons. However, the fact that many assault situations occur in an alcoholic environment suggests that the association may be parallel rather than causative. Polson and Gee (quoting Knight) described a case wherein two British sailors got involved in a drunken fight, when one was kicked on the head. He went into coma and died several days later. Autopsy revealed a ruptured berry aneurysm on the circle of Willis. The defence counsel maintained that in the deceased drunken sailor, rupture of aneurysm was far more likely to have occurred from the raised blood pressure (including an increased pulse pressure between the systole and diastole) than from the actual blow. However, the view was accepted neither by the trial court nor by the subsequent Appellate Court.

The legal problem exists as to the relationship of the trauma to the fatal bleed. The time interval is naturally extremely important. The acid test is—would death have occurred when it did, if the assault had not taken place? The law says that an assailant must "take his victim as he finds him" and that if a sick man is assaulted and dies (while the same assault upon a fit man would not have killed him), that is the misfortune of the assailant as well as for the victim. Occasionally, when little or nothing appears to complicate the injury at the time, and even more, when a long symptom-free interval ensues before frank rupture and bleeding, doubt as to connection between injury and disease should rank high. Blood under arterial pressure is forced into the subarachnoid space, and the victim is stricken with a sudden, excruciating headache and rapidly looses consciousness. Rapid death from bleeding around the base of the brain can be attributed to some brain stem affectation, causing immediate cardiorespiratory arrest. However, at occasions, death may be delayed for minutes, hours or days. Microscopic examination of the aneurysmal tissue may be rewarding in this context. Presence of degraded haemoglobin in its wall and in the surrounding tissues suggests previous leakage, helping to establish the relationship of leakage to the alleged traumatic event.

Degenerative or inflammatory changes in the wall of the lesion will be demonstrable depending upon the duration of survival. Angiographic study before removal of the brain will be helpful in locating the site of bleed. Common sites of involvement in order of frequency are shown in Figure 18.2.

Acute Traumatic Subarachnoid Haemorrhage

Bleeding from subarachnoid space is caused by the same mechanism as that in the subdural space, i.e. shear stresses and rotational movements of the brain leading to tearing of bridging (communicating) veins that leave the cortex and cross the arachnoid space to open into the dural venous sinuses. But where laceration, contusion or infarction of the cortex is present, the bleeding will come from the cortical veins and small arteries, directly into the subarachnoid space. It may also arise from the intracerebral bleeding breaking through the cortex into this space.

The site of appearance of traumatic subarachnoid haemorrhage is influenced by the nature and extent of injury. Where it is produced as a result of blunt force impact with or without meningeal bleeding or cortical contusion/laceration, etc., it occurs either where the bridging veins within the subarachnoid space are most numerous, or where rotational forces are most likely to cause tears. Therefore, the usual sites of appearance of this haemorrhage will be parietal and temporal lobes, the undersurface of the frontal lobes and the cerebellum. But when the subarachnoid haemorrhage is secondary to the laceration/contusion of the brain, then its localisation and extent depends upon the primary injury.

Acute subarachnoid haemorrhage may at occasions be due to **traumatic avulsion of an otherwise normal intracranial vertebral artery**. Contostavlos, Mant and others described a circumstance wherein a blow to the high neck (such as with a fist), critically localised immediately below the mastoid process and behind the mandible, could fracture the transverse process of the atlas resulting in damage to the wall of the vertebral artery within the foramen transversarium. This could lead to haemorrhage dissecting along with the wall of the artery and eventually forcing its way into the posterior fossa. Careful dissection of the high posterior neck and exposure of the vertebral artery in its extracranial course over the arch of the atlas is warranted in such cases, since the external local evidence of the blow/cutaneous mark may be inconspicuous. Sudden death of four ice hockey players with massive basilar subarachnoid haemorrhage was attributed to presumed injury of the vertebral artery due to blow by a puck driven at high velocity to the high neck. In the same report, another player collapsed and died when

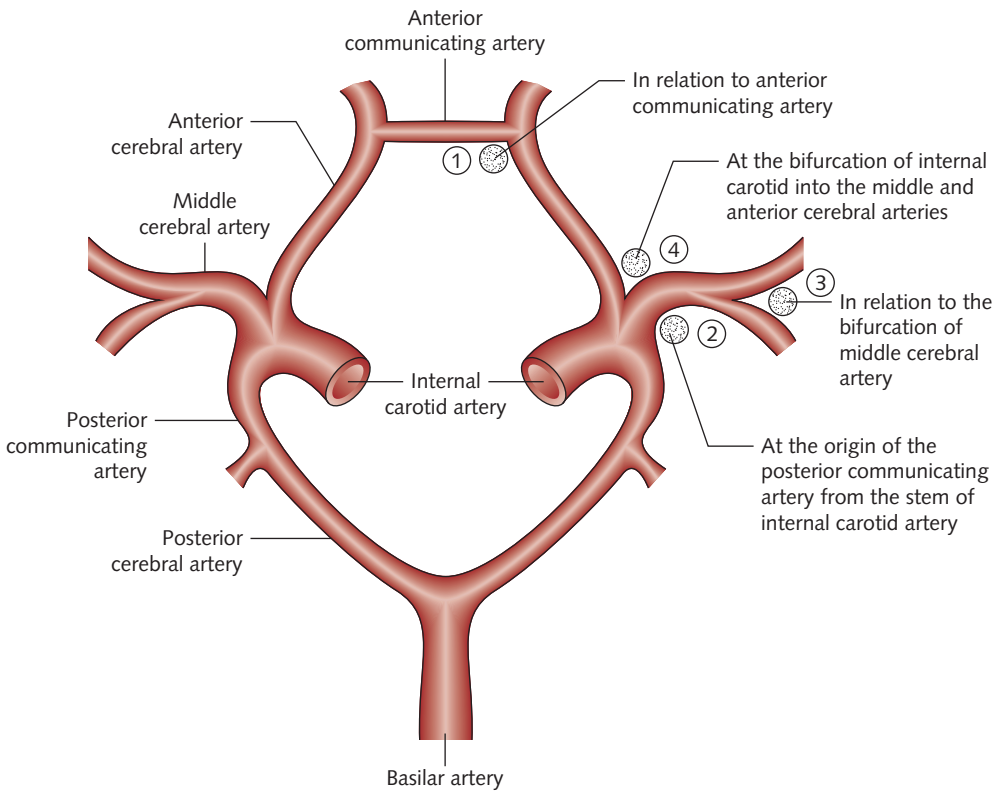


Fig. 18.2 Diagrammatic representation of principal sites of berry aneurysms in the circle of Willis. The serial numbers indicate the frequency of involvement (in more than 85% of cases of subarachnoid haemorrhage, the cause is massive and sudden bleeding from a berry aneurysm on or near the circle of Willis. A leaking aneurysm may affect behaviour leading to conflict, an accident, or a fall with subsequent rupture of the aneurysm).

struck with a fist in an altercation (Maron BJ, Ploiac LC, Ashare AB, Hall WA. Sudden death due to neck blows among amateur hockey players. *JAMA* 2003:599–601).

CEREBRAL INJURIES

The neuropathology of brain damage is a complex subject but a forensic expert has to be conversant with the general principles of causation in order to offer some interpretation of the injuries. There may be a wide range of results from a given insult to the head and as already stressed in the beginning of this chapter, unnecessary theorising about the relationship of extent of trauma to the lesion produced must be discouraged. Well-known aphorism of Munro and the other dictum cited earlier speak highly of this caution to be exercised by all concerned.

Mechanism of Cerebral Injury

Damage to the brain may occur in any one or more of the following ways:

- By direct intrusion of any foreign object such as a penetrating weapon, bullet or some other projectile or fragments of skull in a compound comminuted fracture of the skull.
- By disruption of brain in closed head injuries. Here the mechanism of injury is complex and variable. Brain is almost incompressible, and purely axial impact may give rise to little or no damage. But the impact is almost always accompanied by some rotatory component also, which is now considered to be primarily instrumental in causing brain damage. It is the change in velocity, acceleration or deceleration, with a rotational component, that leads to damage. It follows that no actual blow or fall needs to be suffered by the head to cause brain damage. A typical example in this context is the occurrence of subdural haemorrhage by mere shaking of the head in cases of child abuse syndrome.

In either acceleration or deceleration, the initial sudden change in velocity is applied to the scalp and skull, and the skull then transmits the change to the brain through the anatomical suspensory system within the cranium, consisting of falx cerebri and tentorium cerebelli, which divide the cranial cavity into three compartments, viz., cerebral hemispheres, cerebellum and the brain stem. When violent relative movements take place between the brain and the dura, the cerebral tissue may get dragged against the sharp edges as well as flat surfaces of these membranes. Further, the interior architecture of the cranial cavity, as has already been discussed earlier, adds fuel to the fire, and is believed to be responsible for the common localisation of cerebral damage at the tips and undersurface of the frontal and temporal lobes.

According to Gurdijan and Holbourn, damage to the cerebral tissue may be caused by any one or more of the following processes:

- Compression of the various units of brain by their being forced together.
- Pulling apart of the units through tension.
- **Sliding or 'shear strains',** which move adjacent strata of the tissues laterally as may be seen when a pack of playing cards being displaced, each card sliding upon its neighbour. **Holbourn defines 'shear strain'** as, "a strain produced to cause adjoining parts of the body to slide relative to each other in a direction parallel to their places of contact" (Fig. 18.3).

Coup and Contrecoup Damage to the Brain

This aspect of brain damage is of considerable practical importance, and the neuropathology of its production may be summarised as under:

- When an impact is imparted to a mobile head, the site of maximum cortical damage is most likely to be underneath or at least on the same side as the impact. This is so called, 'coup lesion' (Fig. 18.4A).
- When a moving head is suddenly decelerated as in case of a fall, though there might be a coup lesion at the site of the impact, there is usually cortical damage on the opposite side of the brain—'contrecoup lesion' (Fig. 18.4B).

Taking into account the forensic aspect of anatomy of the skull (particularly the interior configuration), forensic aspect of anatomy of its meninges dividing the cranium into three compartments and the mechanism of production of the cerebral injuries (all have been discussed in detail earlier), various points of practical implications emanating from the prior discussion in relation to coup and contrecoup damage of the brain may be as follows:

- There may occur only contrecoup damage without any coup lesion.

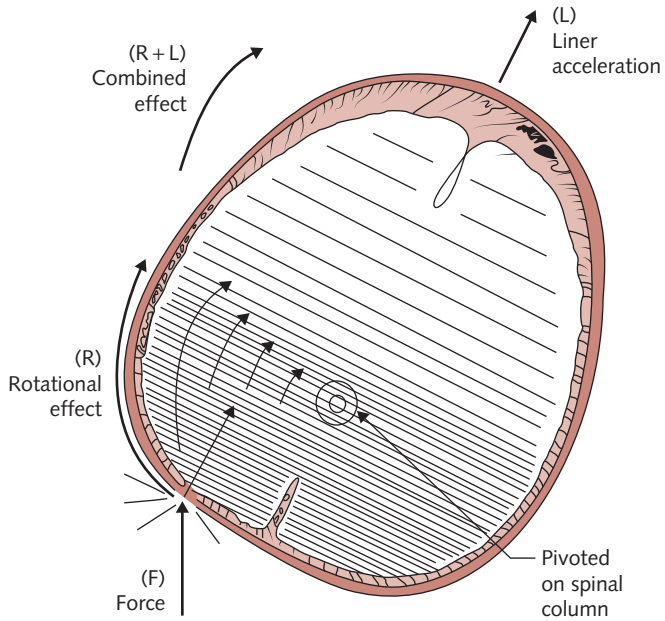


Fig. 18.3 Diagram showing resolution of force (F) into linear and rotational strains responsible for making the adjacent laminar elements to slide over each other with progressive relative displacement of the structures.

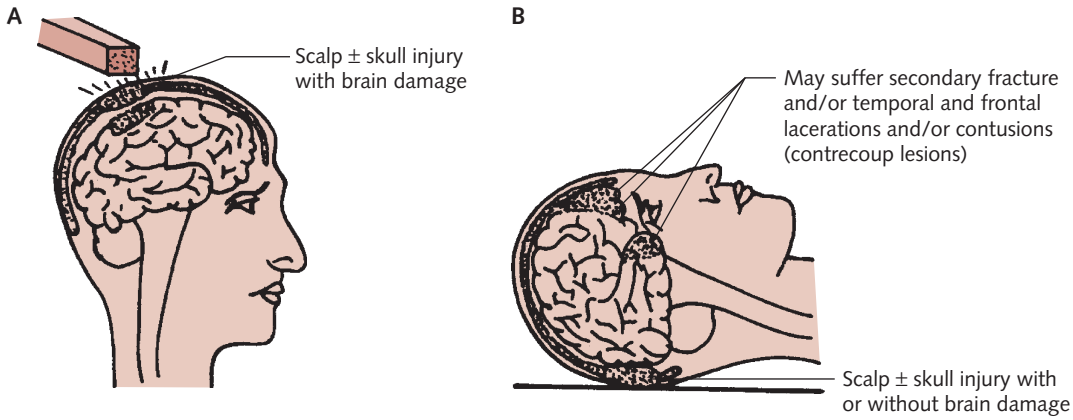


Fig. 18.4 Coup and contrecoup damage to the head: **(A)** coup lesion in 'fixed head'; **(B)** coup and contrecoup lesions in a 'moving head' resulting from sudden deceleration/arrest of movement. The area suddenly striking against something (ground, wall or some other material) shows the presence of coup lesions, whereas the one lying at the opposite side to this area shows the development of contrecoup lesions due to transmission of force through the anatomical suspensory system and dragging of the cerebral tissue through the interior architecture of the cranial cavity.

- Severe coup and/or contrecoup lesions may be present with or without fracture of skull.
- The common sites of cerebral damage, as explained earlier, are the tips and under-surfaces of the frontal and temporal lobes.
- It is virtually unknown for a fall on the frontal region to produce occipital contrecoup, probably due to the relatively smooth internal surface of the posterior cranial fossa of the skull.
- In a temporal impact, the contrecoup lesion may not appear on the contralateral hemisphere but on the opposite side of ipsilateral hemisphere from the impact against the falx cerebri.
- The extent of contrecoup damage may be disproportionately related to coup damage.
- A fall on the occiput may transmit a sufficiently severe force so as to fracture thin bone in the anterior fossa.

Case: Medicolegal Importance of Contrecoup Injuries

On 19th October, 1996, the victim had a scuffle with some miscreants and allegedly received *lathi* blows on his head. He was then admitted to a hospital, where he had to undergo surgery apart from other conservative management but eventually death ensued after about 3 weeks. The intriguing aspects of the injuries were:

- A vertically placed healed wound, 6 cm in length, involving left frontal and parietal area. Anterior extremity was seated 6 cm above the lateral angle of left eye and posterior extremity at a point 6 cm posterior to this. Impressions of the stitches were appreciable running across this scar. On dissection, no bony or cerebral injury was detected.
- On the opposite side of the above mentioned scar, i.e. on the right frontoparietal area, a curved (C-shaped) healed wound with impression of the stitches was discernible. The anterior extremity was placed 4.5 cm above the lateral angle of right eyebrow, marching upwards towards midline in a curved fashion and then running some distance along the midline, proceeding posteriorly over the parietal region and then extending downwards and laterally, ending against the right parietal eminence. On dissection, a piece of bone ($8 \times 7.5\ cm^2$ involving right frontoparietal sites, lying loose in its place) and underneath a subdural haemorrhage measuring $6.5 \times 5.0\ cm^2$ were revealed. Obviously, this C-shaped scar with underlying loose piece of bone was of surgical origin in an attempt to evacuate the haematoma. This was the first clarification sought by the defence counsel. Next, he pleaded his point that the injury to the brain on the right side was due to contrecoup effect originating from the coup impact on the left side, i.e. blow with a blunt force (say with a *lathi* blow) on the left side could be responsible for causing injury to the brain and its meninges on the opposite side.

Here, the injury on the left side was simple as no bony or cerebral injury was demonstrable, but the right side showed the presence of cerebral injury that had been turned complex by the surgical intervention. Surgeons should clearly lay down the initial status of the area inviting surgery (both external as well as internal) vis-á-vis the details of the intervention. The contention of the defence counsel, probably, was to suggest to the honourable court that his client (assailant) never intended to kill the victim but merely to harm him, and unfortunately the death occurred due to indirect effects (contrecoup effects) rather than the injury itself.

Cerebral Concussion (Commotio Cerebri)

Historically, the term 'concussion' was used to describe a 'reversible traumatic paralysis of nervous function'. The term was

introduced by Pare (a French military surgeon, 1510–1590) and has been derived from the Latin 'concutere' meaning 'to shake'. It is popularly known as **stunning**. Trotter (1914) described it as, "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 the actual moment of the accident." The condition has also been referred to imply an immediate but transient loss of consciousness associated with short period of amnesia. The mechanism of loss of consciousness in concussion is believed to be a transient electrophysiologic dysfunction of the reticular activating system in the upper midbrain caused by rotation of the cerebral hemispheres on the relatively fixed brain stem. (Angular or rotational acceleration of the head must be present to produce the clinical entity known as concussion—Genarelli TA, Spielman GM, Langfitt TW et al.). Gross and light microscopic changes in the brain are absent. However, biochemical and ultrastructural changes, such as mitochondrial-ATP depletion and local disruption of blood–brain barrier, have been reported. Courville (1953) has discussed the condition in depth. Changes in nucleus and cytoplasm of neurons, the composition of the cerebrospinal fluid and in the electroencephalograph have been reported sometimes (see also 'Diffuse Axonal Injury' ahead).

The condition may be produced by direct violence on the head or by indirect violence as a result of a violent fall upon the feet or nates from a height or by an unexpected fall on the ground as may be seen in traffic or industrial accidents. In severe cases, brief convulsions may occur, or autonomic signs such as facial pallor, faintness, bradycardia with hypotension, etc. may be seen. The extent of retrograde amnesia usually correlates with the severity of injury. It carries medicolegal importance and may be associated with automatism wherein the individual may not remember any event relating to criminal or violent behaviour. As reported, memory is regained in an orderly fashion from the most distant to recent times, with islands of amnesia occasionally persisting in extreme cases. It seems to be a protective mechanism, caused by loss of sensory input before the latter is transferred to permanent memory storage in the brain. Though it is usually of minutes' duration, it can extend up to several days before the head injury. Other symptoms may include throbbing headache, vertigo, giddiness, or transient blackout, mental irritability, etc. DJ Reddy reports that gross intracranial damage could exist with an intact skull, remaining clinically symptom free and still prove fatal (JIAFS, January 1964, 17).

Damage to the structures may take place depending upon the severity of inertial loading of the head. However, in this type of injury, most of the strain is insufficient to cause structural damage. It seems appropriate to recognise mild concussion as the first step on the scale of the continuous spectrum of brain injury and therefore, concussion may be considered as a mild form of diffuse axonal injury. It has been advocated that the effects of classic concussion may actually involve the same disruptive axonal phenomenon in proportion to the degree of inertial force traumatising the brain. Evidences are surfacing that the severity and duration of functional impairment may be governed by repeated concussions and that the effects of minor head trauma may be cumulative. This explains the condition of 'punch-drunk syndrome/traumatic encephalopathy or dementia pugilistica' seen in professional boxers (see "Head Injuries in Boxers" also). This may also be a problem in other contact sports that engender blows to the head (in American football, cerebral concussions account for 9 out of 10 head injuries, and 1 in 5 university football athletes each season).

Cerebral concussion may be followed by **post-concussion syndrome**, which refers to a constellation of symptoms independent of objective findings on neurological examination. Usually, there is a complex of symptoms persisting months after the head injury and shows various combinations of headache, irritability, anxiety, lassitude, vertigo, blurred vision, easy fatigability and insomnia, etc. Based largely on experimental models, some believe that subtle axonal shearing lesions or some biochemical alterations may account for the cognitive symptoms even when the brain imaging shows normal findings. In moderate and severe trauma, neuropsychiatric changes like difficulty in concentration, memory, and other cognitive deficits may be present. As reported, in mild head injury, these symptoms last for an average of 2 weeks; whereas in moderate head injury, they have higher incidence and longer duration.

Diffuse Axonal Injury

Diffuse axonal injury (DAI) was first described under the heading of 'diffuse degeneration of white matter'. Since then, a variety of terms have been used to point out the nature of the entity, viz., by mechanism—'shearing injury'; by location of the underlying damage; and by combination of mechanism and location of the principal changes—'diffuse white matter shearing injury'. The entity was originally described in a series of patients in whom there was diffuse brain injury without an associated intracranial mass lesion. Adams et al. (1989) introduced the grading, i.e. Grade I—presence of axonal swellings and axonal bulbs throughout the white matter; Grade II—presence of a focal lesion in the corpus callosum in addition to widely distributed axonal injury; Grade III—represents worst injuries characterised by diffuse axonal damage in the presence of focal lesions in both corpus callosum and brain stem. On the other hand, **diffuse vascular injury (DVI)** has been identified as widespread, multiple peri-arterial, perivenular, or pericapillary haemorrhages in the cerebral white matter, cerebellar white matter, cerebral cortex, basal ganglia, thalamus, and brain stem. Both DAI and DVI are produced by acceleration of the head, but axon injury occurs at lower acceleration levels than those required to cause vascular rupture (experimental studies have shown that there is a direct response of the cerebral microvasculature to the lateral head acceleration). Therefore, it has been suggested that DAI and DVI depend upon the same mechanism, with the degree of axonal and vascular damage being determined by the intensity of the head acceleration.

The formerly held view that axons were ruptured/damaged at the moment of injury (primary axotomy/immediate axonal disruption) no longer seems to be appropriate. Now it is considered that other processes also take place leading to delayed axotomy wherein the affected axons undergo lobulation in about 6–12 hours and secondary axotomy occurring after 24–72 hours, which may be influenced by the species, nature and intensity of injury.

Immunohistochemistry has added much knowledge in explaining the axonal damage. By using antibodies against beta-amyloid precursor protein (βAPP), axonal damage has been found in a small series of patients with mild head injury, but death occurred from unrelated causes (Blumbergs et al., 1994). Blumbergs and co-workers derived a 'sector scoring method' through which they could recognise variable amounts of axonal injury and other abnormalities in patients with any of a wide range of Glasgow Coma Scores. As reported, the aging of the axonal injury can be approached as under:

- Identification of dystrophic axons through H & E stained sections usually requires a post-injury survival time of at least 18–24 hours. Further, in case of a few days' survival, the injured axons become progressively widened and assume a varicose appearance. Eventually, they will appear as 'bulbs' or 'spheres' demonstrable with H & E staining techniques.
- Immunohistochemistry reveals axonal injury sooner. βAPP immunochemistry is a useful marker of axonal injury in formalin-fixed paraffin-embedded human brain. It labels injured axons and can reveal axonal injury after 2–3 hours of survival [βAPP is normally present in nerve cell bodies and in axons, but not detectable because of its small quantity. However, under acute injury to the axon (injury may be due to a variety of reasons, namely any infection causing destruction of brain tissue, toxins including carbon monoxide and ischaemia/infarction, etc.), βAPP acts as an acute phase reactant and accumulates in the axons, thereby distending them and allowing their visualisation].
- Evaluation of DAI at autopsy needs critical histological examination of brain tissue. For this purpose, brain needs fixation in 10% formalin prior to processing fragments for paraffin embedding. Preparation of blocks from arterial boundary zones, the parasagittal white matter, the internal capsule, the corpus callosum, the hippocampi, the cerebellum and various levels of brain stem has been advocated. Such sectioning is advocated for differentiating axonal injury arising out of ischaemic complications due to raised intracranial pressure.

Cerebral Contusions

Application of linear or more commonly laminar stresses to the head may disrupt the soft tissue of the brain, especially the cortical region associated with damage to the blood vessels. If the integrity of the cortex is maintained but there occurs extravasation of blood into its substance of the affected area, the region gets bruised and swollen and constitutes 'contusion'. The area of contusion may vary from tiny punctate haemorrhagic spots in the grey matter to large areas involving white matter including cerebral convolutions spreading over sulci.

In usual type of cortical contusion seen in a closed head injury, the cortex appears blue or red or brown due to extravasation of blood into its substance. If the victim survives for sometime, there may be added discolouration from the associated cortical infarction. The lesion is often wedge-shaped, having base on the surface and tapering away into the deeper layers.

Lindenberg and Freytag introduced new names for contusions in the brain that do not fit into coup or contrecoup. Contusions found in deeper structures of the brain along the line of impact are called **intermediary coup contusions**. Contusions caused by skull fracture are called **fracture contusions**. Contusions in the cortex and white matter of the frontal and central convolutions near the upper margins of the hemispheres show no relationship to the area and direction of impact. They are called **gliding contusions** and are caused by stretching and shearing forces occurring in the region of arachnoid granulations, during to and fro gliding of the brain within the skull in moderately severe impact. Contusions in the cerebellar tonsils and the medulla oblongata produced by momentary shifting of the brain towards the foramen magnum are called **herniation contusions**.

Cerebral Lacerations

A greater degree of disruption, producing macroscopic tearing of the substance of the brain, results in 'laceration'. Therefore, it may be considered as an extension in severity of contusion in which the mechanical separation of the tissues can be seen. In cerebral lacerations and most of the contusions, the pia and often the arachnoid matter are disrupted, so that the blood from damaged cortical vessels leads into the subarachnoid or even into the subdural space. Lacerations and contusions are most often encountered in those areas of the brain where the cortex is likely to come into contact with the irregularities in the internal profile of the skull. Therefore, tips and undersurfaces of temporal and frontal lobes are the common sufferers.

Intracerebral Haemorrhage

Intracerebral haemorrhage, either infiltrating the brain tissue or forming actual haematoma, is common in severe head injuries. They may occur at the time of impact or soon afterwards **(primary)** or may occur during the succeeding period due to changes in the intracranial pressure **(secondary)**. The latter are seen more often as the victims of head injuries now survive longer due to availability of modern life-saving facilities, so that there is time for the secondary lesions to creep in. These haemorrhages may rupture through the cortex into the meningeal spaces, which may be termed as 'burst lobe'.

Differentiation, whether the haemorrhage has been caused by head injury or a 'sudden stroke' due to natural cerebral haemorrhage resulting in fall and consequent head injury, is extremely difficult; particularly in elderly subjects with hypertension and cerebral atherosclerosis. Presence of left ventricular hypertrophy, history of hypertension, site and extent of haemorrhage may provide useful parameters for such differentiation (Table 18.2). Furthermore, consistency/inconsistency of the haemorrhage with the degree of head injury is another guide in this regard. Various differentiating points, as gathered from the literature, may include the following:

- In traumatic intracerebral haemorrhage, the interval between the injury and onset of 'stroke' is usually a week or less, rarely longer than 2–3 weeks.
- Present information indicates that the injury to the head must be sustained with the head in motion, for traumatic intracerebral haemorrhage results from the coup-contrecoup mechanism.
- The location of typical post-traumatic effusions into the brain is in the central white matter of the frontal or, more often, the temporo-occipital regions. Spontaneous haemorrhages due to hypertension are more commonly found in basal ganglia, thalamus, pons and cerebellum, which are uncommon sites for post-traumatic damage.
- A history of arterial hypertension in a florid, overweight individual prior to the onset of 'stroke', evidence of degenerative arterial disease (either clinically or postmortem), and particularly the discovery of degenerative changes in the arteries at the margin of the haemorrhage would favour the conclusion of a spontaneous rather than a traumatic aetiology.
- Secondary post-traumatic haematomas are more common in young healthy individuals, while apoplexy incident to hypertension is more common in adults past middle age. However, age alone is not a criterion in either one or the other, for relatively young adults may have arterial hypertension, and older individuals are not immune from traumatic intracerebral haemorrhage.

HEAD INJURIES IN BOXERS

A wide range of injuries may be produced in boxing contests but head is frequently involved. Boxers are at risk of both the acute and chronic damage to the brain. By far the most common injury is the subdural haemorrhage, as is obvious from the mechanism discussed earlier in this chapter.

Punch-drunk syndrome (punch drunkenness/traumatic encephalopathy; also known by names like 'slug happy', 'slug nutty' or 'goofy', etc. amongst the boxers) refers to chronic changes in the brain of boxers, which usually manifest after many episodes of minor head injuries. The lesions may include subdural, subarachnoid and intracerebral haemorrhages, diffuse axonal injury, focal ischaemic lesions, cortical atrophy, slight hydrocephalus, thinning/tearing of corpus callosum, scars or patches of gliosis and brain contusions. The chief symptom of its onset is the deterioration in speed and coordination, seen more readily in properly trained boxers than in crude fighters. This may be followed by slurred speech, slow thought process, expressionless face, stiff limbs, defective memory, and occasional outbursts of violence.

A few of the victims may demonstrate pontine haemorrhage, the so-called 'boxer's haemorrhage'. Brain stem haemorrhage may occur because at the extreme of fight, musculature usually

Table 18.2 Differences between Traumatic Intracerebral Haemorrhage and Spontaneous Cerebral Haemorrhage

Features	Traumatic intracerebral haemorrhage	Spontaneous cerebral haemorrhage (apoplexy)
Cause	Head injury	Hypertension, arteriosclerosis, rupture of aneurysm, etc.
Age	Usually victims are young subjects with history of head injury. Fracture of skull associated with brain lesions will favour the occurrence of traumatic aetiology	Victims are usually middle aged or elderly with history of hypertension, arteriosclerosis, etc. (no history of trauma, fracture of skull or brain injury unless the patient sustains it on falling down unconscious)
Onset	Interval between the injury and onset of symptoms due to haemorrhage is usually a few hours or even a week, rarely longer than 2–3 weeks	There is no such time interval ('stroke' occurs all of a sudden)
Mechanism and manifestations	Usually results from 'coup-contrecoup' mechanism, sustained when the head is in motion Haemorrhage in brain substance along with contusion/laceration suggests violence May be noticed with post-concussional features Variability in development of coma (coma from beginning, or concussion → consciousness → coma)	Sudden rise of blood pressure due to great excitement from any cause, e.g. alcohol, scuffle, assault, etc. may precipitate the episode (especially in victims with hypertension, atherosclerosis, cerebral aneurysm, cerebral tumour/angiomata, etc.) Not so Patient usually remains deeply unconscious
Location	Typically noticed in the central white matter of the frontal or tempro-occipital regions	Noticed usually in the ganglionic regions

gets relaxed and muscle tone is decreased; therefore, the motion of the head is more pronounced. Consequently, acute flexion or extension can readily occur, and thus the brain stem can be pinched over the tentorium.

CEREBRAL SWELLING/OEDEMA

Following trauma, swelling or oedema occurs either in a focal pattern around an intracerebral haematoma or diffusely throughout the cerebrum or cerebellum. The pathological process probably involves disturbance of vasomotor tone causing vasodilatation and disturbance/loss of autoregulation with an increase in both intra- and extracellular fluid.

RAISED INTRACRANIAL PRESSURE: PATHOPHYSIOLOGY AND SEQUELAE

The adult skull may be regarded as a rigid unyielding box containing brain, CSF and blood. An increase in the volume of any one of the components will result in an increase in intracranial pressure (ICP), unless there is a proportionate decrease in the volume of one or the other components (Monro-Kellie doctrine). This is the so-called 'autoregulation process', which comprises of maintaining a constant cerebral blood flow wherein the brain adjusts the intracranial vascular resistance by altering the vessel diameter and tone. However, the limit of compensatory volumetric changes can be exceeded by a too rapid or too great a change in the volume. After initial compensatory/adaptation mechanism occurring through shifting of CSF and displacing blood from venous structures, a critical point is reached when even small changes in volume cause exponential increases in ICP.

In a normal adult, ICP is usually in the range of 0–10 mmHg. Pressure over 20 mmHg is considered abnormal and as reported, rise of ICP above 40 mmHg is manifested by neurological dysfunction and impairment of electrical activity of the brain. If not corrected, the increasing ICP is likely to cause death by deformation of tissue plus shifting of the structures, development of herniae, and secondary damage to the brain stem. Development of these herniae leads to obstruction of CSF flow and development of pressure gradients between the various intracranial compartments. Blood vessels crossing the sites of such herniations may become pinched, leading to vascular complications. Vascular damage to the midbrain and pons is thought to be due to downward traction on the central perforating branches of the basilar artery. In general, the more slowly a focal mass expands, the more likely it produces distortion of the brain without resulting in an early rise in ICP. On the other hand, if the lesion/mass expands rapidly, death usually follows soon from high ICP, and the effects like distortion and herniation of the brain hardly have time to take place.

Manifestations of increased ICP will depend upon the extent of compression and the availability of space for displacement of structures in the various compartments (fossae) of the cranial cavity, i.e. **in the middle fossa**; structures lying in relation with the sharp edge of the tentorial hiatus are the usual sufferers. Increased pressure in this area leads to the following:

- Herniation of the uncus of the medial temporal lobe that leads to compression on the brain stem. (Further rise in ICP may lead to even lateral displacement of the brain stem causing contralateral corticospinal tract to impinge against the opposite tentorial edge. This may become responsible for a localising pseudo-ipsilateral hemiparesis, the so-called 'Kernohan notch' phenomenon.)
- Compression of the ipsilateral corticospinal tract in the crus cerebri causing contralateral hemiparesis.
- Compression of the ipsilateral third nerve and oculomotor nucleus in the midbrain causing pupillary dilatation and failure of reaction to light.
- Displacement of cingulate gyrus under the free edge of the falx producing a subfalcine hernia.

In the **posterior fossa**, increased pressure will result in herniation of cerebellar tonsils into the foramen magnum and compression of the medulla. This can lead to rapid respiratory failure. Progressively increasing pressure may lead to further downward displacement of tonsils (coning) leading to sheering of the vasculature supplying the brain stem, causing haemorrhages known as **Duret haemorrhages**. Rarely, a posterior fossa mass may displace cerebellar tissue upwards through the tentorial opening to produce a 'reversed tentorial hernia'.

Evidence of cerebral oedema may be noted in the form of flattening of gyri, filling of sulci, evidence of grooving of one or both unci (sometimes, unci may be discoloured as a result of incipient infarction), or in the severe cases, hippocampal herniation through the tentorial opening, etc. For examination of brain at autopsy, it is better to fix it where neurological issues are involved, either traumatic or from disease process (there may not be any need for fixation if no cerebral lesions are expected or apparent on external examination of the brain wherein 'wet cutting' usually serves the purpose). Fixation of brain provides firmness to the tissue, which allows thinner and more accurate sections to be made, as well as better histological preservation. For fixation, brain is suspended in a specially designed tank made of fibreglass containing 10% buffered formalin (buffer is a substance/chemical/device used for lessening the effect of a blow/collision/impact, etc.). The quantity of the solution should be sufficient to allow the brain to float clear of the bottom of the receptacle. There are lugs moulded into the sides to hold the suspensory strings, which support the brain by means of a paperclip hooked under the basilar artery. An alternative method of suspension is to leave the falx intact and use it to suspend the brain down in formalin.

Spinal Injuries

The spine and head should be considered as part of the same system in relation to trauma. Spicer and Strich have shown that

haemorrhage into the spinal root ganglia may be associated with head injury. Electroencephalographic changes have been shown to occur in about half of the victims of cervical spine injuries. From the functional point of view, the upper two cervical vertebrae provide most of the rotational movements and the lower five, flexion and extension.

CONCUSSION OF SPINE

This condition can occur without any evidence of external injury to the spinal column, from a forcible blow on the back or a fall from height or a bullet injury but is commonly seen in railway accidents and motor car collisions, hence also known as **railway spine**. Signs and symptoms may appear immediately or delayed for hours or days. There may be paralysis of upper and lower limb or lower limb alone with the involvement of bladder and rectum. The individual may present with headache, giddiness, restlessness, neurasthenia, loss of sexual power and weakness in the limbs. The paralysis is of temporary nature and recovery may occur within about 48 hours.

The condition may be attributed to the mechanism similar to that seen in the brain in closed head injuries and may be due to some momentary collision of the cord against the wall of the canal or a transient deformity in the profile of the canal due to violent acceleration/deceleration or rotational strains.

Injuries to the spine/spinal cord may be studied under the following subheads.

INJURIES TO THE UPPER CERVICAL SPINE

The first cervical vertebra (atlas) supports the occiput and is held in place by a number of ligaments. The transverse ligament of the atlas encloses and restricts the motion of the odontoid process of the second cervical vertebra (the axis). Disruption of this ligament may occur in rotational injuries of the upper cervical spine resulting in atlanto-axial subluxation with or without odontoid fracture, which may damage the pons or medullary pyramids. Vertical impacts to the head with a straightened neck may lead to compression fracture **(Jefferson's fracture)** of the anterior and posterior arches of the atlas with the lateral displacement of the lateral masses onto the axis. Another common fracture, the so-called **'hangman's fracture'** consists of fracture of the pedicles of axis resulting in anterior dislocation of C2 on C3 with or without odontoid process fracture. This injury is typically met in judicial hangings and vehicular accidents in which the neck is forcibly hyperextended and rotated.

MIDDLE AND LOWER CERVICAL INJURIES (HYPEREXTENSION AND HYPERFLEXION INJURIES)

Injuries to the cervical spine and cord between spinal segments C4 and C8 occur with greater regularity and constitute the most common type of immediately nonfatal spinal injuries. Cord lesions may occur with or without spinal fractures but injuries to the spinal ligaments may be encountered almost invariably. The motions responsible are hyperflexion, hyperextension, hyper-rotation and/or compression of the spinal column. Hyperflexion injuries may result from blows to the back of the neck, shallow water diving injuries and in vehicular accidents (frontal impact). Hyperextension injuries may again be seen in wrestling matches or fights where a forceful 'hammerlock' is used. Rotational forces may produce subluxation with facet interlocking and/or other forms of dislocation with impingement of the cord. Out of the hyperextension and hyperflexion injuries, hyperextension is more dangerous because weak anterior longitudinal ligament is incapable of maintaining the integrity of the cervical spine during hyperextension whereas during flexion, the strong musculature of the posterior part of neck is capable of protecting the spine. The term **'whiplash injury'** has been assigned to these hyperextension and hyperflexion injuries encountered in vehicular accidents. Middle-aged and elderly with pre-existing spondylosis are particularly vulnerable. Same condition may occur following a violent blow (rabbit punch) over the spinous process of upper cervical vertebrae. Fracture, dislocation or subluxation of middle cervical spine, usually results in more severe injury to the cord than similar injuries sustained to the upper cervical region where there occurs sufficient space about the cord to accommodate encroachment on the spinal canal.

THORACIC AND LUMBAR SPINAL INJURIES

The upper thoracic spine from T1 to T10 enjoys more resistance to injuries than does the cervical spine because of added stability of the thoracic rib cage and costal vertebral ligaments. Fracture or dislocations and rotational injuries require great force and consequently are comparatively uncommon. The lower thoracic and lumbar spine, however, is quite vulnerable to injury because of increased flexibility in this region and lack of lateral stability of the ribs. Fractures and/or dislocations can occur here with or without injury to the spinal cord. Rotational and flexion forces seem to be more important in the production of injuries in this region. In the lower lumbar and lumbosacral region, compression injuries with 'bursting' fracture of the vertebral body(s) are most common but may not necessarily involve the cord.

INJURY TO THE SPINAL CORD

Spinal cord injury may result in clinical state of quadriplegia or paraplegia. Quadriplegia (tetraplegia) is the paralysis of all the four limbs and usually indicates an injury above the level of emergence of the roots serving the brachial plexus (fourth cervical). It is possible that some function may be preserved. Paraplegia is the paralysis of the lower extremities and variable portion of the trunk due to injury to the spinal cord below the emergence of the brachial plexus (first or second thoracic segment). The spinal cord injured person may suffer either complete or partial loss of function below the level of injury. In the latter, in which some motor and/or sensory function is preserved, prognosis is usually better. Some experts use the terms quadriparesis and

paraparesis to describe the incomplete paralysis while reserving quadriplegia and paraplegia for the complete motor paralysis. Ducker and Walleck (1985) indicated that 85% of those who show an immediate complete injury will tend to retain a complete symptomatology at the end of 1 year, whereas those with immediate incomplete signs and symptoms have a greater tendency to show some additional neurological recovery by the end of a year.

Pathology of Spinal Cord Injury

At the very outset, it may be kept in mind that the victim dying of acute spinal cord injury may exhibit little or no change in the spinal cord tissue itself. The usual types of pathological changes seen in impact injury to the cord are usually consistent, regardless of the mechanism of the injury. Even in clinically complete traumatic spinal cord injury with total loss of function below the level of lesion, the cord is functionally but not usually physically transected. Actual physical transection only occurs in extreme cases where massive fracturing and distorting of the spine, penetrating injuries, crush injuries or other devastating injuries have occurred. Spinal cord involvement is usually encountered in association with fracture and/or dislocation of the spinal bone(s). However, it has been recognised that cord may be traumatically injured in the absence of the said injuries to the spinal bones. It has been indicated by Davis et al. (1971) that soft tissue disruption and haemorrhages are frequently encountered at the site of the fracture and/or dislocation or ligamentous tears. Bleeding can occur into the spinal meninges (haematorrhachis) and/or into the substance of cord (haematomyelia) and this may extend along the axis of the cord, upwards as well as downwards. Therefore, it becomes imperative to examine the spinal column by X-rays and to examine the soft tissues, bones and canal carefully. In this regard, it is important to know the relationships between the level of vertebrae and the spinal cord.

Penetrating Injuries of the Spinal Cord

Penetrating injuries of the spine and spinal cord are entitled to separate discussion. These may result from missiles and by some other penetrating instruments/weapons. Regarding penetrating wounds by missiles, it may be borne in mind that they can cause paralysis without grossly obvious damage to the spinal cord. This is probably due to the effect of 'shock wave' and large temporary cavity which accompanies the high velocity missile, even if the missile does not happen to make a 'direct hit' on the cord itself. A major difficulty in evaluating the spinal cord injuries is that the level of cord injury may not correlate with the level of external wound. In addition to shock wave and temporary cavity effects of high velocity missile, other factors responsible for such incompatibility may be as under:

- There may exist some individual variations in the relative position of the cord.
- Mature spinal cord is anatomically shorter than the axial skeleton and the disparity progresses at lower levels of the cord. For example, conus medullaris injuries correspond to a level of about the first lumbar vertebra.
- The position of the cord within the spinal canal usually changes with body posture and movements. Hence, the exact stance of the victim at the moment of injury matters much in the proper evaluation.

Penetration of the cord by a knife or other sharp/blunt pointed instruments may occasionally be encountered. Stab wounds may show the same anatomic and coincidental disparities of relationships of the level of neurological damage to the wound on the vertebral column as do the missile injuries. However, it may seem surprising how the weapon should pass into the cord with complete bony encasement. It is obvious that only very heavy blade can fracture and depress the lamina. However, even a light blade may be able to effect its penetration towards the cord, if it enters between the laminae, as when the victim is bending when struck or the blade may be directed from below upwards to penetrate between the overlapping laminae. In the cervical region, the laminae are narrower, and a horizontal thrust can penetrate. A puncture wound (even by a needle) in the space between the first and third cervical vertebrae may cause almost instantaneous death due to injury to the medullary centres or upper part of spinal cord. The process of such killing is known as **'pithing'** and this type of puncture wound can easily be overlooked. Also noteworthy may be the 'ice pick' wound created by some small narrowly pointed instrument which can penetrate dorsolaterally at the intervertebral foramina. As considerable force is usually required to achieve penetration, it may result in the blade being broken off. After the blade has entered the canal, it may penetrate the cord or push the cord aside. The latter situation may be ascribed to the tough fibrous capsule that accompanies the pia mater of the spinal cord. If pushed aside, the cord may get contused due to its collision against the bony wall, and this may explain unexpected clinical symptoms as compared to the anatomical injury.

Medicolegal Considerations of Spinal Injuries

Forensic issues revolving around the spinal injuries may include aspects like mortality, morbidity, quality of life and survival potential. With modern techniques for maintaining nutritional support, bowel and bladder functions and respiratory support, etc., long-term survival for such victims may be expected. The most critical period for survival is usually the first 3 months after injury. Factors influencing survival include the level of spinal injury, residual degree of respiratory control, degree of sensory and motor disabilities, age and prior status of the victim and degree of associated systemic injuries. In the individuals having injuries below the fourth cervical level, stabilisation of respiration may be a lesser issue than the bowel and bladder function. The personal idiosyncrasies may outweigh the physical injuries and the victim's own response to his injury may play a

significant role in the outcome. Depression and suicide may be the other complications of spinal injuries. Other circumstances inviting forensic considerations may include spinal injury during surgery or administration of spinal anaesthesia, in connection with child abuse, gymnastic or other exercises and in karate training or demonstrations.

Trauma

FACIAL TRAUMA

As a rule, facial wounds heal rapidly owing to their great vascularity. However, they are grievous if they are severe and cause permanent disfiguration or deformity. Such permanent disfiguration may be due to scar or keloid formation, or due to derangement or loss of tissues. Pulping of face can result from vehicular run over injury or blunt impact by a heavy brick/stone or some other object. Complex contours of the face may intercept impact with consequent characteristic damage.

Abrasions and contusions in or around the **mouth and nose** could suggest forceful opening of the mouth to administer something, or forcible closure of mouth and nose as may be encountered in smothering. Superficial lacerations of inner aspects of lips can occur due to forceful apposition of lips against teeth. Injuries to lips can also result from blunt impact such as fisting. A blow on the head sometimes causes bleeding from the nose due to partial detachment of its mucous membrane without any injury to the nose. The bone is usually fractured at its junction with the frontal bone. Blood from fractured site may be inhaled or swallowed. During a fainting attack, a person may strike his nose against the ground or some object and sustain a fracture of nasal bone.

Penetrating wound of the nose caused by thrusting a pointed instrument up the nostril may result in death by injuring the brain through the cribriform plate of the ethmoid bone, though no sign of external injury is evident (concealed puncture wounds). Left nostril or the septum of a woman is liable to be injured by pulling out the nose ring worn by her. Occasionally, the lips or nose may be cut off or bitten off as a revengeful act. As reported by Lee et al., there have been instances where the nasal aperture has been the site of gunshot suicidal fire.

Injuries to the **eyes and ears** are not uncommon. Injury leading to permanent loss of vision of either eye or loss of hearing of either ear constitutes grievous hurt. They may occur from blunt trauma as in the case of a fall or blow, or from penetrating trauma as well. During a quarrel, ears may be bitten off or cut off, and their lobes may be torn by pulling out the earrings either with the intention of causing hurt or committing theft. A severe/hard blow over the external ear may cause rupture of tympanic membrane. Abrasions, contusions and/or lacerations can occur to one or both ears, from accidents or from deliberate actions.

The term **black eye** refers to accumulation of blood around the eyeball and eyelids, which manifests as a darkish discolouration around the eye. This can be resulted directly from blunt trauma over the eye or from indirect force. Gravitational seepage of blood from injury higher up in scalp may lead to ectopic contusion/bruising of eyelids. Percolation of blood into the orbit may be due to a contrecoup injury of head. A simple fall on the face on a flat surface does not usually cause a black eye, because the prominence of the eye brow, cheek-bone and nose prevent damage to the orbit.

Penetrating wounds of the cornea are also relatively common, causes being numerous; therefore, types of wounds encountered may vary considerably. Incisional and punctured wounds are quite common and show greater variability. Sometimes, there may be haemorrhage in the anterior chamber of the eye due to blunt trauma (hyphema). The eyes may be gouged out with the fingers. However, it needs to be kept in mind that birds of prey generally first attack the eyes of a dead body, when exposed in a field or jungle.

Injuries to the **teeth** are encountered in varied circumstances. They may get dislocated or fractured either by a fall or by a blow with a blunt weapon, such as a fist, a shoe, the butt end of a *lathi*, etc. According to Andreasen and Schutzmannsky, most dental injuries occur shortly before school age and are primarily due to falls. Playground injuries are quite common after the child is of school age. Bicycle accidents resulting in fractured teeth and injuries to surrounding areas are also common in school-age group. In teenage group, oral trauma is frequently associated with athletic activities and automobile accidents. Oral injuries sustained during fights are common in older age group. Addicts have more dental disease than normal individuals. It is believed that bruxism frequently is a contributing factor in the relatively large incidence of fractured posterior teeth noted in narcotic addicts.

Injuries caused by mechanical violence, in all probability, leave abrasions, contusions, and/or lacerations on the lips and/or on the gums, etc. The dislocated tooth/teeth may at times get aspirated or be swallowed. Cases of false reports about the loss of a tooth are usually encountered with a view to charging the accused with an offence of grievous hurt. It is, therefore, necessary that the following points should be taken into consideration when reporting on a person who alleges to have his/her tooth knocked out:

- The number of teeth present in each jaw.
- The condition of the neighbouring and other teeth as to whether they are firm, shaky or diseased.
- The condition of the socket of the missing tooth, as to whether there is any stump left if a tooth is fractured, whether there is any bleeding/laceration, etc.
- The condition of the lips and gums as regards the presence of injury.
- If a tooth is sent with the injured person, it should be examined to ascertain if it corresponds to the missing tooth. After examination, the tooth should be sealed in a packet and handed over to the police personnel accompanying the injured person.

- X-ray examination of jaw may reveal fracture of alveolar margin from the site of dental injury. Root of the concerned tooth could also be examined under X-ray.

Majority of **facial bone fractures** result from automobile accidents. Not unusually, however, they result from violent forces exerted on the face by assault either with a fist or with a heavy object. **Mandible**, though the strongest of all the facial bones, gets involved too often. Mandibular fractures can be typically divided into two types, i.e. closed (no break in the skin) or open/compound (in which skin and mucosa are also damaged). Symptoms usually include pain, malocclusion and trismus. Respiratory distress due to displacement of tongue into the throat may result from fractures of symphysis. In both types of fractures, the jaw usually remains wired until clinical evidence of stability rather than X-ray evidence determines healing. Fractures of **zygoma** (cheek bone) usually occur as a result of violent blow to the face from a fist or heavy object. They are most commonly seen in assaults or athletic injuries. Because of the thickness and heaviness of the body of the bone, blows to the zygoma usually lead to fracture at three weak areas about its periphery, i.e. frontozygomatic, zygomaticotemporal and zygomaticomaxillary sutures. Due to such involvement, the zygomatic fracture is often referred to as a 'tripod fracture'. **Maxillary** fractures, on the other hand, more often result from an automobile accident in which the driver or passenger is thrown up against the dashboard or steering wheel, or through the windshield.

CERVICAL TRAUMA

Superficial wounds of the neck may or may not cause serious bleeding, but penetrations, incisions and deep lacerations usually produce copious bleeding due to severance of carotid and/or jugular vessels. A forceful blow over the neck can cause a fracture of the larynx, involving thyroid cartilage or rupture of the trachea to cause death either by spasm or oedema of glottis or by suffocation due to internal bleeding into the larynx or due to surgical emphysema. However, a skillfully delivered karate-type blow may not leave more than a minimal local evidence of damage.

Wounds of the sympathetic and vagus nerves may be fatal, and those of the recurrent laryngeal nerves cause aphonia. In case of a wound of the larynx, speech is usually not possible, if the wound is below the vocal cords. However, a person may be able to speak in whisper if the wound is not gaping. Occasionally, the question whether or not a person with 'cut throat injury' can speak assumes immense importance. This may supplement or negate the contention that whether the victim was/was not able to call for assistance or whether the persons in an adjoining room heard any noise or not. Harvey Littlejohn cites a case (*Forensic Medicine*, 1925, London: J & A, Churchill) wherein a woman, in an attempt to get away with the thyroid gland tumour divided windpipe below the vocal cords. On the arrival of the doctor, she was conscious, and narrated that she had torn the tumour out of her neck as the same was choking her and that she wanted to die. In another case (*Lancet* 1909;1:1501), a boy's throat was cut across and the larynx divided just above the vocal cords. Facial and lingual arteries were also severed. After receiving the injury, he was alleged to have made a statement involving certain persons. The doctor stated that the wound would not have prevented the boy from speaking though the voice would obviously grow fainter during the gradual succumbing of the boy to injuries.

Wounds of the neck are mostly incised and rarely punctured. They are more often homicidal than suicidal and rarely accidental. In a suicidal case, the person usually holds the weapon in his right hand and starts the incision from the left side of the neck drawing it to the right. Tailing of the wound is therefore seen on right side. Carotid arteries are not frequently injured as they slip backwards when the head is extended. Bleeding is usually venous, and loss of consciousness is gradual. However, death may take place quickly from air embolism, due to air being sucked in by negative pressure in the veins. A person attempting suicide generally makes repeated horizontal, parallel, shallow, half-hearted cuts on the neck initially before he gathers enough courage to make the final lethal cut. These preliminary shallow cuts are called as hesitation cuts/exploratory cuts/feeler strokes/tentative cuts. A homicidal cut throat wound is invariably quite deep, and obviously lacks hesitation cuts. However, cases have been reported where superficial cuts resembling hesitation cuts were present along with the main wound. (For differences between suicidal and homicidal cut throat injuries, see the Chapter on 'Injuries by Sharp Force'.)

The chief danger in incised and stab wounds of the neck is from haemorrhage due to an injury to blood vessels. Death is due to haemorrhage, air embolism consequent upon the entry of air into the venous system, or due to asphyxia from filling of air passages with blood. Wounds of the large vessels may not necessarily be rapidly fatal, and an individual so wounded may be capable of physical and volitional acts.

Sometimes, air from wounded respiratory passages enters into the subcutaneous space resulting in subcutaneous emphysema, which may dissect down into the mediastinum and is responsible for subsequent respiratory obstruction. Hyoid bone can get fractured from blunt impacting force, or from blunt constricting force, as in manual strangulation. Scratch abrasions and/or contusions are suggestive of throttling, while a pressure abrasion in the form of a ligature mark is indicative of hanging or strangulation. (For details of mechanisms of fracture of hyoid bone, please see the Chapter on 'Asphyxial Deaths'.)

THORACIC TRAUMA

Chest carries a semi-rigid bony case, enveloping vital organs that are softer, more mobile and deformable. The scope and extent of injuries to the lungs vary with the degree of violence/impact and other attending factors. Injuries may range from simple bruising or laceration to massive damage or collapse, with or without fracture of ribs. Most cases of lacerations of lungs are due to traffic accidents, fall of heavy object on the chest, compression

of the chest (traumatic asphyxia), and uncommonly assault. Generalised trauma to the chest (blast lung) may cause multiple contusions and tears to the lung substance due to linear and rotational strains. Details of blast injury to the lung have been given in the Chapter 'Firearm Injuries'.

Trauma to the chest usually challenges the integrity and viability of the individual. As in other cases, severity of the injury is related to magnitude of the kinetic energy delivered, which can be expressed by the formula $KE = \frac{1}{2}MV^2$. It is apparent that the velocity of the wounding object is the most important factor in determining the extent of the tissue damage. When velocity is doubled, kinetic energy or the destructive force is quadrupled. The energy may be exerted by a moving or accelerating object on a stationary victim, or the damage is of the deceleration type in which a moving victim collides with another moving or stationary object, e.g. a vehicular accident.

A compression of chest may lead to disturbance in cardiac function, and even death may follow with little or no evidence of external injury to the chest wall. Surface injuries may include slashes, lacerations, bruises or abrasions. Blows on the chest may produce concussion of the chest causing shock, and rarely death. Simple contusions of the chest wall may be followed by pleurisy or pneumonia. Blunt injuries on areas lying against bones, such as shoulder and shoulder blades, may sometimes cause linear lacerations that may be confused with slashes. A close and careful inspection will usually suffice to resolve the issue. Nonpenetrating wounds, at occasions, may cause free bleeding from the divided mammary or thoracic arteries.

Traumatic **fractures of the bony rib cage** are usually produced by blunt trauma and rarely, by a missile. The severity of these injuries ranges from simple fracture of a rib to the involvement of several ribs at multiple points producing the so-called **flail chest or stove-in chest**. In direct violence, such as by blows, stabs or pressure with the knee, the broken ends are likely to be driven inwards; whereas in indirect violence, such as by muscular contraction during violent coughing or convulsions, fractured ends are likely to be driven outwards. The ribs more vulnerable to fractures are fourth to eighth ribs, as they are attached at both the ends, and are comparatively more unprotected. Bilateral symmetrical rib fractures in front near the costal cartilages and at the back near the angles may occur in traumatic asphyxia. Such fractures may also occur when a person sits on the chest and compresses it considerably by means of knees or elbows, by trampling under feet or by means of bamboos. They may not always be accompanied by external injuries or ecchymoses of blood in the soft tissues over the ribs. **Nobbing fractures,** commonly found in 'battered baby syndrome', are due to holding of the child with both hands and shaking it violently. Fractures of ribs on both sides close to the spine may occur in this process, imparting a nobbing appearance.

As mentioned earlier, flail/crush/stove-in chest is the result of fracture of several ribs in more than one place or simultaneous fracture of the sternum and several ribs. A portion of the chest wall loses connection with the rest of the rib cage, and moves independently and paradoxically from the intact portion. In addition, the to and fro motion of the chest wall with each respiratory cycle leads to mediastinal instability. Thus, a flail chest involving a large portion of the chest wall can be lethal because of the combined cardiac and pulmonary dysfunction.

Fracture of the **sternum** is rare. It is ordinarily due to direct violence, and usually occurs transversely either between the manubrium and body or a little below. The fragments usually remain in apposition or the upper portion passing backward. It may be fractured by indirect violence as a result of forcible flexion or extension of the body, or a forceful direct impact of the bone against the steering wheel of a vehicle. The arch of aorta being quite near the surface adjoining the sternal border may also get involved. (Obviously, due to such placement, the vessel may also get involved with an instrument/weapon of small dimensions, leading to fatal consequences.) The sternum may rarely be fractured spontaneously by muscular spasm caused during violent coughing. Fracture may also occur following external cardiac massage. Fractures of the ribs (usually of 3rd to 5th), particularly at costochondral junctions on the left side, may also occur, with minimal surface bruising.

In case of penetrating injury of chest by sharp penetrating weapons, pointed ends of fractured ribs or gunshot wounds, there may be little or no external bleeding but profuse and fatal internal haemorrhage. This may be due to valve-like overlap of tissue at the wound. Collected blood may be liquid, clotted or usually a mixture of both. The tissue damage inflicted by a stab wound is largely determined by the size of the weapon and the course it travels, whereas in case of gunshot wounds factors like velocity of the missile, the course of the missile through the tissues and presence or absence of dissipating energy usually determine the tissue damage. As a general rule, low-velocity missiles/bullets tend to confine their destructive effect to the trajectory, whereas high-velocity missiles produce far greater tissue damage, even at distant places due to dissipating forces.

Due to the large and accessible target area, the chest is very frequently the site of a homicidal stabbing. Serious injury or death is common because of seating of vital structures within the thorax. Common target area is the region against the heart on the front of chest. Involvement of back of the chest is infrequent because of protection afforded by muscles and shoulder blades at the back. Sides of the thorax are not so often stabbed due to hindrance afforded by the protecting arms. Although the knife is the most common weapon involved, the type of the weapon may vary depending upon region to region. Sharpened iron rods and even pointed sticks or other pointed instruments may be employed. The weapon almost always makes its way through an intercostal space, though not infrequently a rib or costal cartilage may be 'nicked' or even completely transected. Sometimes, the weapon may be deflected upwards or downwards into adjacent intercostal space after impacting against the rib. Factors influencing the entry of the weapon/instrument into the tissues have been elaborately discussed in the Chapter, 'Injuries by Sharp Force'.

Once within the thorax, the pleura often gets involved; thus, pleural space becomes open to the external environment. Pneumothorax is the usual outcome. (There are **three types of pneumothorax**, i.e. simple, open and tension. It may be caused by penetrating or blunt trauma, or iatrogenically during minor surgical procedures like thoracentesis or during pleural or lung biopsy. In simple or closed pneumothorax, a wound in the chest wall or lung permits air to escape and to collect in the pleural cavity. The wound may become sealed spontaneously or it may necessitate tube thoracotomy with water-seal drainage. Open pneumothorax is usually associated with a large defect in the chest wall that permits air to enter freely from the atmosphere into the pleural cavity. That is why it is often referred to as 'sucking chest wound'. Cardiopulmonary function can severely be affected due to this coupled with instability of the mediastinum. Tension pneumothorax is resulted when air is under extreme pressure within the pleural cavity. The wound acts as a one-way valve allowing air to enter the pleural space without an avenue for its escape. This produces progressively increasing intrapleural pressure leading to collapse of the lung and mediastinal shift).

The heart may be injured from nonpenetrating or penetrating trauma to the chest. Blunt trauma leading to involvement of heart is relatively infrequent. Involvement may be encountered following steering wheel injury in which the heart is compressed between the chest wall and the vertebral column. A violent blow on the chest with a fist or some heavy object can also damage the heart. The myocardial damage from blunt trauma may range from superficial contusion to full thickness rupture. Rarely, ventricular septum, pupillary muscles, chordae tendinae or the valve leaflets may be involved during blunt trauma. At occasions, pericardium may get ruptured, and if the defect is large enough, the heart may herniate and get strangulated. (The **traumatic rupture** of heart needs to be differentiated from spontaneous rupture. In traumatic rupture, the heart is usually ruptured on the right side and towards its base. The ribs and overlying tissues are often damaged. Rarely, the rupture may occur without leaving any external mark of violence damage. **Spontaneous rupture** of heart may occur in circumstances where the organ is already weakened by some disease injury. Elderly are the usual victims, and the rupture in such cases occurs mostly in the lateral, anterior or posterior wall of left ventricle. Sudden exertion and increased blood pressure may be the accompanying factors.

Penetrating wounds of the heart are extremely serious and usually fatal. A rupture or penetrating wound of the atria is more dangerous than a wound of the ventricle because the auricular wall is thin and less contractile and therefore, bleeds profusely. On similar lines, a penetrating injury to the right ventricle is more dangerous than that of the left. It is possible that foreign bodies, such as bullets, or fragments of shells, may remain embedded in the myocardium for months or years without producing symptoms. In such cases, missile may act like a plug, effectively checking any severe haemorrhage.

Rupture of the **diaphragm** is commonly caused by deceleration type of injuries. Also, a blow to the abdomen or chest, a crushing injury, or jackknifing of the body may cause a sudden increase in intra-abdominal pressure and produce disruption of the diaphragm. The most commonly involved site is the central portion of the left side of diaphragm. Rupture may also follow herniation of the intra-abdominal viscera into the thorax. Penetrating trauma, as mentioned earlier, may also involve diaphragm.

Intrathoracic vessels may get injured because of sudden deceleration in an automobile collision, a fall from height or an air crash. Disparity between the speeds of a fixed and a mobile portion of the involved vessels is the usual mechanism of production of injuries, i.e. the fixed portion coming to an abrupt halt whereas mobile segment continues on its path. Thus, shearing force causes disruption of the vessel. Thoracic aorta is the commonest victim of this type of injury. Disruption occurs most often at the aortic isthmus, distal to the origin of the left subclavian artery, where the aorta is fixed by the ligamentum arteriosum. Usually, the vessel wall is circumferentially transected and death occurs from exsanguination.

Foreign bodies may get lodged anywhere in the respiratory tract. They can be aspirated or enter as missiles. With time, the foreign bodies usually get encysted and fixed by fibrous tissue. Aspiration of foreign bodies into respiratory tract occurs mainly in children. Peanuts, marbles, coins, bunttas, buttons are among the frequently aspirated items. Occasionally, it may be seen in an adult. Occlusive foreign bodies in the trachea are likely to cause death by asphyxiation. Partially occlusive foreign bodies in the airway may behave as one-way valve, permitting entry of air, but impeding its exit. Organic foreign bodies in the respiratory passage absorb water/fluid and swell up. Thus, they may get impacted at one location. Nonorganic foreign bodies, on the other hand, do not change size and therefore tend to move unless they are wedged. Foreign bodies within the cardiovascular system are usually bullets or fragments of bullets. These may get lodged in an artery, vein or the heart and may remain fixed or embolise. It is possible that foreign bodies, such as bullets or fragments of shell, may remain embedded in the myocardium for months or years without production of significant symptoms. Missile may act as a plug, effectively checking any severe embarrassment.

ABDOMINAL TRAUMA

In the so-called 'magic box' of the body, structures can be injured by a variety of traumatic insults. At times, no surface lesion may be evidenced in spite of severe or fatal internal haemorrhage. Nature and extent of clothing may contribute to this absence of surface injuries. Since the origin of recorded history, abdominal trauma has had dire implications for survival. Like the Greek warrior of the wall of Troy, the American Marine in Vietnam wore body armour to minimise the effects of abdominal and thoracic insults. In general, the damage following trauma depends upon the consistency, mobility, state of distension of organs, the type of the force, the site of impact and the resistance offered by the abdominal wall under a particular situation.

Solid organs such as liver and spleen rupture more readily than hollow organs like stomach and intestines.

Liver is the quite frequently involved organ in vehicular accidents and in falls. Its large size, fixed location and solid consistency make it an easy target for blunt injury to the upper abdomen and thorax, especially on right side. Nonaccidental rupture of the liver may be caused without a weapon. Harvey cites a case where it was ruptured by a kick, and two others in which the rupture was caused by kneading with the knees and elbows or 'kil kani'. Substance of the liver may be involved while the surface remains intact. Similarly, liver injury may be seen without any external marks of violence. Subcapsular tears produce intrahepatic haematoma, which may eventually rupture into peritoneal cavity, causing death hours or days after the injury. Stab wounds of the liver often provide clues about the nature of the weapon as the organ is fixed and of solid consistency.

Spleen, because of its thin capsule, weak supporting tissue and friable pulp, is easily susceptible to blunt injury to the left hypochondrium and left lower thoracic wall. The injury may vary from minor laceration of capsule to fragmentation. Lacerations with capsular tears will lead to bleeding into the peritoneum. Subcapsular lacerations may result in the accumulation of blood in the parenchyma, which may lead to delayed rupture and intraperitoneal bleeding. According to Clark et al. (1975), delayed rupture may occur at any time after abdominal trauma, but 75% cases of delayed rupture occur within 2 weeks after the trauma. Taylor mentions a case in which rupture of both stomach and spleen occurred from a fall of about 20 feet, and in which no bruises or other external signs of injury were evident.

Stomach, in its distended form is more liable to be involved. It may get bruised or lacerated following blunt trauma. In adults, rupture is usually situated at the pyloric end along the lesser curvature because of reduced elasticity due to a deficient muscular layer and paucity of mucosal folds. In children, however, the greater curvature is most frequently involved in rupture. Delayed rupture may occur at the site of bruising involving the entire thickness of wall. Accidents during anaesthesia have sometimes led to stomach rupture. Spontaneous rupture of the organ is quite rare as the smooth muscle coat is able to accommodate pressure and volume changes to a considerable extent.

Kidney injuries are rare, as they are deeply situated in the abdomen. However, direct trauma to flanks and lumbar region and indirect trauma such as fall can injure the organ. A sudden impact from behind can push the lower ribs forward and can cause contusion and/or laceration of the kidney. In violent impact from front against flanks, the kidney may be pushed against the ribs or transverse processes of vertebrae. Lacerations are common with right kidney as it is relatively more fixed in children, and scanty perinephric fat may be a contributory factor for the increased incidence of renal injuries. Perinephric haematoma without renal injury can occur with blunt trauma.

Pancreatic injuries are rarely an isolated phenomenon. They are usually associated with injury to the other abdominal organs. Pancreas, as a rule, tolerates injury poorly. Local injury such as caused by a penetrating instrument may evolve into a pseudocyst or abscess. Larger injuries such as large scale disruptions may evolve into massive haemorrhagic pancreatitis and death from exsanguination. The insulting nature of pancreatic injuries is attributed to the release of digestive enzymes that digest the pancreatic lobules with devastating consequences.

Because of its placement across the vertebral column, the pancreas is fixed in position and thus, gets involved by compressing abdominal trauma. Lacerations frequently occur across the mid position of the body of the gland at the junction of the head with the tail. However, a kick or punch in the upper abdomen may also injure the organ. External injury to the abdominal wall may not be visible in such cases. Metabolic by-products of the enzymatic breakdown of the substrate of pancreatic tissues may result in far reaching haemodynamic changes like profound vasodilatation, hypotension, etc. The most helpful diagnostic clue to the pancreatic injury is an elevation of the serum amylase level.

Small bowel (intestines) injuries mostly result from automobile accidents and impact against the steering wheel. Injury may occur due to crushing of the bowel against the lumbosacral spine or due to shearing of the bowel and its mesentery at points of fixation. The most common sites are the first portion of the jejunum and the terminal portion of the ileum. Bruising of a distended or kinked loop of intestine is rare. Damage by blunt force may range from bruising lacerations to avulsions or intramural haematomas.

Colon and rectum injuries are rare. However, various circumstances leading to injuries to these sites may be like wounds through the perineum, forcible thrusting of blunt or pointed objects through the anus, accidental swallowing of pins and needles (especially in tailors, carpenters and cobblers, etc.). Rarely, forcible injection/introduction of air/gas/liquid as a practical joke may be encountered. Diagnostic and therapeutic instrumentation such as proctoscopy and enema may be other causes of injury.

Considerable force is required to damage the large bowel; therefore, it is obvious that associated injuries are often present. The bowel may be compressed against the vertebral column or burst by a sudden blow against a distended loop. The site of injury is usually near the junction of mobile and fixed portions of the bowel, such as junction of the sigmoid and descending colon, or at the junction of the caecum with the ascending colon.

Injury to the extraperitoneal rectum is usually incidental to fractures of the pelvis as this portion of the rectum is more or less fixed to the pelvis. Thrusting of a stick or other similar object into the anus is a mode of torture occasionally practiced. In majority of such cases, other injuries also accompany this type of violence. Sometimes, injury may be connected with sodomy.

Bladder injuries may occur due to blunt trauma to the lower part of the abdomen, pelvic fractures, obstetrical trauma and some endoscopic procedures. A full/distended bladder is decidedly more susceptible to injury. When the bladder enlarges, its wall becomes thinner and less able to withstand pelvic fractures and usually ruptures intraperitoneally through the weakened dome.

The empty bladder enjoys the relative protection afforded to it by the pubic arch and gets usually damaged extraperitoneally in association with pelvic fractures.

In intraperitoneal ruptures, the urine leaks into the peritoneal cavity producing chemical peritonitis. Extraperitoneal ruptures, as written earlier, are most commonly associated with pelvic fractures involving pubic rami and symphysis pubis. The mechanism of rupture usually operates either through the stresses placed on the lateral ligaments anchoring the bladder base or through direct injury by the bony fragments. Under such situations, urine enters the space of Retzius and thereafter may dissect along with abdominal wall into the inguinal canal, scrotum and through the obturator foramen into the thigh, or through the sciatic notch into the buttock region leading to tissue necrosis along such paths. Rupture of the bladder can also occur due to accidental trauma such as fall from height or on a projecting object or sometimes by some instrument while procuring abortion. Pre-existing intrinsic bladder disease, growth or diverticula, etc. increases the susceptibility of the bladder to rupture with lesser degrees of trauma.

Limbs/extremities may be the victim of any type of injury. Arms are often involved in knife wound, either as defence wounds of hands or lower forearms or from deeper slashes or stabs sustained in a scuffle. Unless some major blood vessel is involved, such injuries are rarely dangerous. Blunt injuries to the limbs are extremely common; particularly in vehicular accidents, any combination of injuries may be encountered. 'Brush burns' are frequently seen in vehicular accidents where the body is skidded across some rough surface. 'Flaying' of the skin of legs due to rotatory injury from wheels has been described under 'types of lacerations' in the chapter 'Injuries by Blunt Force'.

Injuries to the extremities necessitating amputation or permanent impairing their power constitute grievous hurt. As regards injuries inflicted by others, it may be pointed out that severe injuries to the extremities may be produced without a weapon. Violent twisting of a limb, for instance, may cause dislocation of a joint. Further, though crushing by ropes or cords may produce comparatively slight injuries to the extremities, yet indicate infliction of severe torture.

Trauma to the **external genitalia** is not uncommon. These were encountered with considerable frequency during the Vietnam Conflict, owing to the prevalence of 'booby trap' land mine devices employed in that war. In general, **male external genitalia** may get traumatised by kicks or fisting to the perineum or squeezing the scrotum and penis. Severe contusions may lead to death, or severe compression of the testes may prove fatal from shock.

Penile strangulation may occur due to voluntary or involuntary placement of a constricting apparatus around the penis. Young adults may employ a number of devices for masturbatory activities. The more elderly males may employ such devices to increase potency. Once the penis is incarcerated, eventual development of oedema in the distal portion prevents removal of the device. **Penile skin injuries** may include abrasions, contusions or lacerations. Zipper represents a frequent source of cutaneous injuries. The trouser zipper may entrap penile skin (usually in the region of the foreskin). Circumcision injuries may also be seen. Loss of penile skin may occur in either the child or an adult because of overzealous traction on the prepuce prior to excision of the foreskin. The presence of cremasteric reflex almost always preserves the testes. Testicular and scrotal injuries usually occur in young adults. Scrotal lacerations may result from gunshot or other piercing instruments. Blunt trauma resulting in testicular contusion, laceration or dislocation may occur in sports activities, falls or saddle injury from bicycles or motorcycles, etc. Seizing by the testicles is a common method of assault in India. Chevers mentions a case in which a man dragged another in this way with such violence "that the whole preputial integument was torn away". Incised wounds may be attended with severe haemorrhage. An individual may mutilate himself by cutting off a portion of the penis. In India, removal of the male organs was formerly being practised in order to produce eunuchs for immoral purposes. Rarely, incised wounds may be inflicted from a sexual motive/revenge, or during self-defence to thwart the designs of the assailant. Harvey cited a case wherein a woman at Kachar inflicted a deep and severe wound on the penis of her father-in-law, who wished to take liberties with her.

Undoubtedly, majority of traumatic lesions of the vulva and vagina are originated from sexual activities. It may appear surprising that injuries may well result from intercourse between **consenting parties**. In unprepared and unaroused tense partner, damage is much more likely to occur than in one who has reached the excitement phase of human sexual response. Many predisposing factors have been forwarded as contributing to vulva-vaginal injuries from coitus. These may include prepuberty or virginity, recent vaginal surgery, pregnancy, alcoholic/drug intoxication, genital health status of vulva and vagina, clumsiness, vaginismus, undue active involvement of the female, exceptional coital positions, postmenopausal stage of the female, multiple consorts, male brutality during the coitus, etc. The extent and location of coital injuries vary. Minimal hymenal laceration may result in only minimal blood loss in one virgin, whereas another may experience an excessive tear accompanied by profuse haemorrhage. Similarly, vault injuries may show wide range of severity. However, majority of vaginal coital lesions involve the fornices, dominantly the posterior fornix, more often on the right side. This may be due to the larger size of the right fornix leading to greater incidence of lacerations on this side.

Female genitalia can also become the target for an **assault**. Fisting/kneeling/kicking against the area have been reported. Thrusting a stick or some other pointed object/instrument into the vagina is not uncommon. At occasions, attention also needs to be drawn to the surprising situations where victims of rape or other sexual assaults manifest trauma to other parts of the body in the absence of demonstrable damage to the genitals. At one time, several cases of murder by wounding female genitals occurred in Scotland. In one of these, death occurred in 10 minutes; in another, a wound of the labium (three-quarters

of an inch long and three inches deep) proved rapidly fatal from loss of blood. Taylor mentions a case wherein a woman, about 36 years of age, was kicked by her husband in lower abdomen while she was in a stooping posture. She died in about an hour from loss of blood. A wound measuring about an inch in length and half an inch in depth was observed at the edge of the vulva, extending from the pubes along the ramus. The left crus clitoridis was crushed throughout its length, leading to fatal haemorrhage.

Accidental trauma to the vulva is frequent. Children appear to be especially prone to such injuries, which generally originate from falling astride gates, bars, chairs or pointed/projecting objects. Although the vascularity of vulva and perineum is less in children, extensive bleeding can occur. Ezell et al. report that the most common agents responsible for such injuries are the open dresser drawer and the tricycles. In adult females, direct trauma to vulva is likely to be complicated by the development of a distinct haematoma because of presence of large venous plexus and loose areolar tissue. However, injury with a sharp or pointed object is more apt to produce external bleeding. The absence of any evidence of external trauma, however, does not preclude injury to the pelvic organs. Hakanson describes a case of a 5-year-old child who presented with haematuria, but with no evidence of external injury. The child had reportedly received injury while sitting on a pointed phonograph spindle; the spindle entering the anal canal, perforating recto-vaginal septum, and proceeding through the vagina entered the bladder.

Genital self-mutilation is rare in either sex. In the past, many women used to suffer from inadvertent injuries during attempt towards abortion. Deliberate mutilation of genitals may occasionally be seen in patients carrying hostile-dependent relationship with others. The purpose may be to attract attention. French and Nelson described a case wherein a woman had injured herself creating superficial lacerations of genital tract. It was alleged that the trauma was directed to express hostility towards her husband, who was always showing only sexual interest in her, to the exclusion of her other attributes. Goldfield and Glick described the case of a 19-year-old patient wherein the diagnosis of self-mutilation was approached by finding the gentian violet dye under the patient's fingernails (she had painted her genitalia with gentian violet that led to vaginal bleeding).

Finding of **foreign bodies** in the vagina can have different interpretations. Out of mere curiosity, younger children sometimes insert candy, toys and pencils into the vagina. In an adult female, obviously, it is not always a chance happening and usually bears some relationship to individual's sexual behaviour. All manners of objects have been removed from the vagina. Hawkins and Bourne quote the famous case of Bland Sutton, which involved the removal of a small bust of Napoleon from the vagina, presumably introduced as a supreme act of hero worship. In the past, use of a variety of household devices by the unskilled individuals was responsible for accidental retention of foreign bodies in the genitals.

Chemical agents have been reported to be responsible for injury to the female genitalia. Various douche solutions can act as irritants and in the ongoing era of female hygiene spray deodorants, such injuries are not uncommon. Erythema and inflammation of external genitalia are common due to use of such agents. The use of potassium permanganate as douching agent dates back to the turn of century. In the late 1940s and in the 1950s, numerous reports of injuries by this agent were available in the developed countries.

Intrauterine contraceptive devices can also lead to injuries to the genitalia. From ancient times, people have been using all sorts of **barrier contraceptives** to prevent pregnancy. Women used vaginal pessaries made of crocodile and elephant dung, pomegranate seeds, bee's wax and numerous other plant and animal materials. Similarly, men used to wear condoms made of intestines of animals. These ancient methods have been replaced by vaginal diaphragms, caps and condoms, now mostly made up of rubber and latex materials, and by spermicides. [Invention of condom is attributed to a physician named Dr. Condum, who recommended it to Charles II (1660–1685) to prevent illegal off-springing. More probably, the term 'condom' that appeared in print for the first time in 1717 was derived from the Latin word 'condus', which means a receptacle.]

19 CHAPTER

Transportation Injuries

After going through this chapter, the reader will be able to describe: Mechanism of vehicular injury | Pattern of injuries to the driver, front-seat occupants and rear-seat occupants of a motor car | Pattern of injuries to the pedestrians, motor cyclists and pedal cyclists, etc. | Aircraft accidents, railway accidents and vehicular conflagration | Medicolegal aspects of transportation injuries

Problems related to transportation injuries and eventually to the death of the victim of the accident may call upon the entire spectrum of forensic expertise. The injuries may occur in any form of transportation, viz., roads, railways, vessels and aviation. Numerically, road traffic accidents account for the great majority worldwide.

A retrospective study carried out by Dr. Vishal Garg and Dr. SK Verma at AIMSR, Bathinda (Punjab) during (1st April 2007 to 31st March 2009) revealed that out of 784 cases studied, 59.4% comprised of road traffic accidents, 12.1% of poisoning, 9.4% of fall from height, and 8.3% contributed towards suicidal attempts plus assaults/homicidal cases. Male preponderance was quite evident, and age group commonly affected was 21–30 years. Rural victims surpassed urban ones. The study concluded that road traffic accidents and poisoning cases continue to be a growing menace, incurring heavy loss of manpower and human resources in the form of death and disability along with a corresponding drain of potential economic growth.

Mechanisms of Vehicular Injury

The dynamics involved in any injury by mechanical force have been thoroughly studied by De Haven. Although it is not within the scope of this presentation to discuss the physics that plays a role in the road crashes, a few basic concepts may help to clarify the nature of the lesions associated with trauma and their underlying mechanisms:

- The extent of an injury sustained is directly proportional to the degree of acceleration or deceleration to which the occupant of the vehicle is subjected. A constant speed, however rapid, has no affect whatsoever as is evident from the space travel or the rotation of the earth. It is the change of rate that is traumatic, i.e. the acceleration or the deceleration. The 'G' formula is used to calculate the mean force involved in a 'real life' accident. Impact of deceleration forces may be calculated from the formula:

$$G = \frac{Kv^2}{d}$$

  Where G is expressed as gravitational force, v is the initial impact of speed, d is the stopping distance and K is the constant (0.034) with speed in miles per hour and distance in feet. With kilometres per hour and metres, K is 0.0039.
- During acceleration or deceleration, the tissue damage produced will depend upon force applied per unit area. De Haven's study of survivors of free falls up to 150 ft has shown that the body may tolerate and expand a force of 200 times the force of gravity for brief intervals, during which the force acts in transverse relation to the long axis of the body. When the forces are not evenly distributed over the entire body (as in traffic accidents) extensive injury may result from the forces concentrated on a few square inches of the body.
- In the common 'frontal impact' there never instant arrest of the vehicle, even when it runs into a huge immovable structure, the vehicle itself deforms from the front so that there is always some deceleration distance and time. This emphasises provision for the crumpling of the front and rear of the car, leaving the central rigid cell that comprises the passenger compartment. The aim is to extend the stopping distance and time, so that the G value acting on the occupants is reduced.

The type of vehicle (other than the motorcycle) makes little difference to the mechanism of injury but most statistical surveys divide them into cars and light vans under 1.5 tonnes on the one hand and heavier vehicles such as trucks and buses on the other. Heavy vehicles naturally suffer less than cars and vans because of their greater mass and strength and also their

height above the ground. **Attending to the motor cars, the injuries may vary according to the position of the occupant**.

INJURIES TO THE DRIVER

When the most common frontal impact occurs, the unrestrained driver first slides forwards so that his legs strike the facia/parcel shelf area and his abdomen or lower chest contacts the lower edge of steering wheel. The body then flexes across the steering wheel and begins to rise, the heavy head goes forwards and there is flexion of the cervical and thoracic spines. The upward and forward component causes the head to strike the windscreen, the upper windscreen rim or the side pillar. The windscreen is often broken by the head and the whole body may be ejected through the broken glass, to land on the bonnet or sometimes on the roadway ahead or on the side (Fig. 19.1).

Depending upon the above events, the injuries encountered in the drivers may be:

- Impact against the facia may produce abrasions, lacerations and fractures of the legs around knee or around the upper shin level.
- Pressure of feet upon the floor, especially when it is intruded by any structural component, can cause fracture anywhere from foot to femur. Hip joint may be dislocated posteriorly and even the fractures of pelvis are not uncommon.
- Impact of the abdomen and chest against the steering wheel may cause severe internal injuries. Trauma associated with an impact on the chest by the steering wheel or column is often severe, yet external evidence of injury may be minimal, or absent, particularly if the victim is wearing a number of clothings as in the winter season. There may be bruising of the skin surface, but this may not be evident even in the presence of severe internal injuries, again the winter season is notorious for such possibilities. Lacerations of the skin are uncommon unless the steering wheel snaps producing such injuries. Other steering wheel lesions may be bruising of the lungs, fractures of ribs and/or sternum, cardiac contusions and haemothorax or pneumothorax or both.

In the abdomen, **liver rupture** is frequent involving any part. Subcapsular tears can occur with the formation of subcapsular haematoma, which can rupture later on. Spleen may show tears in some cases, often around the hilum and rarely, it may be avulsed from the pedicle. The mesentery and omentum may show bruising in some cases and rarely there may be laceration and fenestration sufficient enough to cause a lethal haemorrhage.

In the chest, **lungs may get injured**, either from intrusion of the fractured ribs or from the otherwise blunt impact. There may be air bullae under pleura or collection of blood and a pneumothorax or haemothorax may result. The interior of the lung may be involved even in the presence of intact visceral pleura from the transmitted force or extreme variations in intrathoracic pressure accompanying the impact.

Heart may get damaged even in the absence of external marks or thoracic cage fractures. Bruising of the epicardium and underlying myocardium is not uncommon. Avulsion of heart may be seen in high speed impacts. Less severe damage may lacerate the ventricles or atria and cause gross haemorrhage. Coronary artery thrombosis has been described following contusion over a coronary artery. Penetrating injuries from sternum, ribs or external objects may lacerate the heart directly.

A more common thoracic injury, associated with the deceleration, is the **laceration of aorta**. The mechanics involved may be through the severe whiplash effect on the thoracic spine. Another probability is the 'pendulum' effect of the heart within the relatively pliable thoracic contents. When the thorax is violently decelerated during an accident, the heavy cardiac mass attempts to keep moving ahead and may literally snatch itself from its basal mountings, the most rigid part of which is the aorta. Separation usually takes place at the point where the aorta is attached to the spine, at the extremity of the arch. Sometimes there may be additional transverse intimal tears adjacent to the main tear, the so-called 'ladder tears' as they may resemble the rungs of a ladder.

Head, neck and face involvement in the drivers is relatively frequent as a result of projection against, and/or ejection through the windscreen. The face often suffers multiple lacerations from contact with the shattered windscreen glass. These lacerations are usually bizarre shaped or 'sparrow-foot' patterned. Injury to the head may be caused by the impact of head against the windscreen rim or corner pillar or after ejection. The injuries may include scalp contusions, lacerations, fractures of skull, intracranial haemorrhage/haemorrhages or damage to the brain.

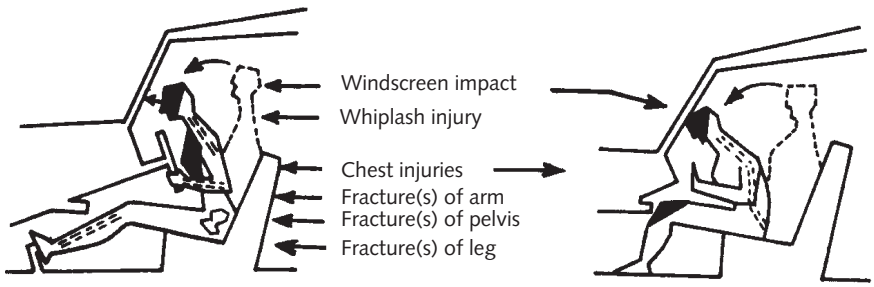


Fig. 19.1 Usual injuries to the driver and front seat passenger.

Injuries to the neck, the so-called **'whiplash injuries'**, have been stressed recurrently. There is often a double component in that the hyperflexion of deceleration is followed by a rebound hyperextension when the head meets an obstruction in the front. Rear impacts also cause the double whiplash effect. The injuries may result in fractures and/or dislocations, especially at the level of 5th and 6th cervical vertebrae. Rigid head restraint can reduce injuries from hyperextension. Other injuries may be atlanto-occipital dislocation with or without laceration of tendons, ligaments, separation of cartilaginous lining of the articular surfaces and intra-articular haemorrhage.

Less common injuries are the injuries to the upper limbs that may occur from the transmitted force through gripping the steering wheel or from impact against the windscreen, pillars, intrusive roof, bonnet or ground when held up in a reflex protective position.

INJURIES TO THE FRONT SEAT OCCUPANTS

The position of front seat passengers in the car is even more dangerous. Though there is no steering wheel to impact against the chest or abdomen, its absence also denies the little protection offered to the driver in reducing the collision with the windscreen possibly by giving him something to brace against. Another factor may be the fact that the driver usually pays attention constantly to the road and so is better placed in appreciating the impending crash, compared with the occupants who may be unaware of the approaching danger and fail to 'brace up' any nearby structure. Any range of injuries may be seen in the occupants and no specificity can be assigned.

INJURIES TO THE REAR SEAT OCCUPANTS

During the forceful deceleration impact, the unrestrained rear seat occupants are projected forwards and strike the back of the front seats. They may be thrown over the seats, striking and contributing further injuries to the front seat passengers and may even be ejected through the windscreen, which is smashed by them or by the people in front. In roll-over accidents, they may get churned up inside the vehicle, when multiple injuries may occur by hitting against the various structures.

EJECTION CRASH INJURIES

Ejection from the vehicles results in severe and multiple injuries to the driver as well as the occupants. Ejection was found to be second only to the steering wheel as a major cause of injury in the large series of cases studied by the Cornell group. Ejection occurs mostly in the roll-over accidents. According to the Cornell group, the risk of fatal injury as related to ejection or non ejection is five to one.

Injuries to the Pedestrians

These are probably the most common fatalities worldwide. Most pedestrians are struck by motor cars and the type of vehicle makes a difference in the production of injuries which, unlike the injuries to the automobile occupants, are an acceleration process and not a deceleration one. Injuries may be grouped as (Fig. 19.2).

PRIMARY IMPACT INJURIES

These include the injuries that are sustained when any part or parts of the victim first strike the vehicle. Such injuries carry importance in the sense that they may bear design of the part of the vehicle causing the injury in the form of imprint abrasion and/or patterned bruise, etc., thereby helping in reconstructing the events. The part of the body involved will depend upon the position of the person in relation to the vehicle when struck, i.e. whether crossing the road or walking with or against the traffic, etc. The position of the injuries will be further modified by the fact whether both feet were on ground or one was raised, the nature of surface of the road and the footwear of the victim.

SECONDARY IMPACT INJURIES

These injuries are the result of impact between the body part(s) and the vehicle for the second time as when the victim after striking against the vehicle is further scooped up/or otherwise hurled up on the vehicle resulting in injuries to the other parts of the body by the same vehicle.

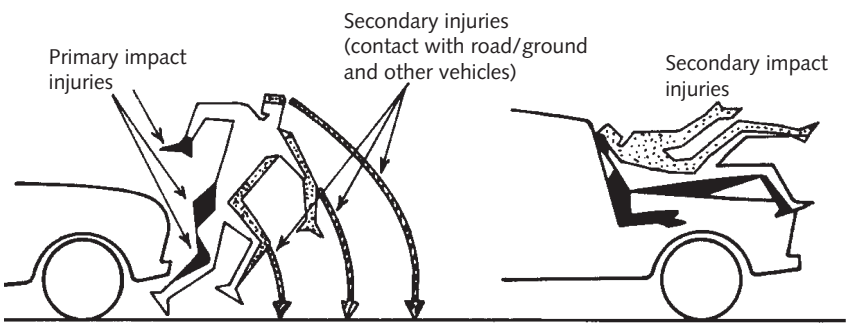


Fig. 19.2 Usual pattern of pedestrian injuries.

SECONDARY INJURIES

These are sustained by the victim after being knocked down by the vehicle and striking the ground with the subsequent risk of being harmed by some different vehicle, thus receiving the injuries by striking against the ground or some object on the ground as well as those sustained through some other vehicle.

CRUSH INJURIES

These may be seen when the person has been run-over, the severity depending upon the weight of the vehicle and its clearance from the ground. Cases have been reported in which 'jumping' of the wheels has occurred, thus reducing the extent of crush injuries to the minimum and involving only one side of the body.

As there is considerable variation in the automobile models, there is little point in mentioning specific names and, therefore, mechanics of usual injuries met in a common type of vehicle (say, a car) may be described. **In a typical case**, the first impact tends to knock the legs and rotate them to the oncoming vehicle resulting in the so-called **'bumper injuries'** on the legs (fractures of tibia and fibula, often compound and comminuted). If the leg is weight bearing at the time of impact, the tibial fracture tends to be oblique whereas if not stressed, as may be during walking, the fracture line is usually transverse. In such a case, the leg with higher placed injury usually represents the one in contact with the ground supporting the body weight at the moment of the impact. Sometimes, the level of injuries may be low as compared to the height of the bumper, suggesting application of the brakes at the moment of the impact thereby causing dipping down of the bonnet.

Depending upon the profile of the front of the vehicle, the hit pedestrian is either thrown forward or scooped up onto the bonnet top. **If thrown forward**, secondary injuries will be suffered as a result of striking the ground. A further hazard may be the danger of being run over by the vehicle, if the victim is projected directly in front of the vehicle. Sometimes he/she may be dragged by the underbelly of the vehicle and seriously soiled and injured. If thrown to the side, the victim may be run-over by other vehicle overtaking the vehicle in question. **If scooped up**, the victim may land on to the bonnet or against the windscreen or corner supporting pillar. The flat bonnet usually does relatively little harm, though some abrasions, minor lacerations or friction burns may be the result. Striking against the windscreen or the side pillar is the most frequent cause of severe head injury. In case of high speed collision, victim may be thrown up on the roof and sometimes somersaulting so that the head strikes to the roof and finally to the ground, with the subsequent risk of being harmed by some other vehicle.

In general, the severity of the injuries will depend upon the magnitude of impact. But it is virtually impossible to estimate the speed from the nature of injuries. These can be fatal even at slow speed and yet, occasionally, high speed impacts may follow only insignificant injuries. In Ashton's series, half the deaths ensued at speeds less than 30 miles per hour.

Soft tissue injuries may include abrasions, bruises lacerations and crushing injuries. Typical 'brush-burning' may be found when the pedestrian is dragged or scrapped against the rough surface. These are usually superficial abrasions without significant haemorrhage into the skin and subcutaneous tissue. Due to the friction-type force responsible for brush-burning, protuberant parts of the body are predominantly involved. A characteristic lesion is **'flaying injury'** encountered in run-over cases. The rotator effect of the wheel against a fixed limb or head may strip-off almost the entire tissue down to the bone. When the wheel passes over the abdomen or pelvis, multiple parallel striae or shallow lacerations may result owing to the ripping tension in the skin (Fig. 19.3A and B). Great internal injuries may be caused with little surface injuries. **Some 'patterned injuries'** may sometimes be observed involving soft tissue, which may help in identifying the vehicle in a 'hit and run' accident. The most common is the 'tyre marks'



Fig. 19.3 Injury marks in an accident victim: **(A)** extensive lacerations and fracture of pelvis, **(B)** graze.

outlined in intradermal bruising, usually caused by the skin being forced into the grooves of the tyre tread and the edge of the raised rubber tracing out the pattern.

Injuries to the Motorcyclists

The motorcyclists are much more prone to receiving serious injuries, and this vulnerability may be due to the inherent instability of the two-wheeled vehicle. Generally, the behaviour of the younger age group involved in rash driving and greater acceleration capacity of the vehicle are the other contributing factors.

The two extremities are the commonly affected, though any part of the body may be involved. As the rider almost invariably falls to the ground, head injuries are common and often grave, leading to 80% of deaths according to Bothwell. A typical fatal injury is the fracture usually from secondary impact with the ground. Temporoparietal fractures are common often with contrecoup brain injury. In violent accidents, a basal skull fracture is usually seen. This exhibits as a transverse crack across the floor of the skull, crossing behind the greater wing of sphenoid bones, through the pituitary fossa to the opposite side. This may also be associated with fissure fractures passing upwards to the temporal bones. At autopsy, the base of the skull may be appreciated to have divided into two halves, each moving independently of each other like a hinge, the so-called **'motorcyclist's fracture'**. Neck suffers quite often and Mant found cervical spine fractures in over a quarter of his series.

'Ring fracture' around the foramen magnum may be encountered in some cases, caused by an impact on the crown of the head. Ring fracture of the base of the skull and atlanto-occipital avulsion due to anteroflexion on the motorcycle riders have been reported by H Maeda, T Higuchi and K Mognchi. In their case, the driver of the motorcycle sustained a 'ring fracture' and the pillion rider an atlanto-occipital avulsion, dural tear, etc. Mode of action of the accelerating forces to the heads of the victims along with their physiques may explain the mechanism that caused different injuries.

The role of **'safety helmets'** in the prevention of head injuries cannot be overlooked. The severity of the impact may defeat the protective role of the helmet. However, these helmets act by providing a rigid barrier against the impact and allowing the protected head to skid across the road surface, thus prolonging the stopping distance and time to reduce the G force of deceleration. Vulnerability to fatal injuries is much more with helmetless motorcyclists than the helmeted ones. Rarely, at high speed impacts, helmet may be penetrated or the head and brain may be damaged by the transmission of blunt force. **Crash bars** are another safety measures provided in some cases, to protect the legs. Unless extremely strong, these crash bars may sometimes themselves trap the legs if they happen to bend on impact.

A peculiar phenomenon, called the **'under-running'** or **'tailgating'**, may rarely be seen in the motorcyclists, where a rider drives into the back of a truck or some other heavy vehicle. This may occur due to sudden and unexpected stoppage of the heavy vehicle. In such cases, motorcyclist's head and shoulders are smashed against the tail-board. Decapitation may result in extreme cases. Falling from the vehicle, particularly at high speed, may result in the injuries to the extremities as well as the chest and abdomen.

Injuries to the Pedal Cyclists

A pedal cycle carries the same instability but has far less speed. Again, head injuries are common due to the fact that height above the ground is significant, and the rider meets a passive fall this may be complicated by any forward motion or projection from impact by the motorcycle. Secondary damage to the shoulders, legs, anus may also occur. A peculiar injury, i.e. the entrapment of the leg between the wheel spokes, has been described by Strauch.

Aircraft Accidents

Medicolegal expert and forensic pathologist have a large role to play in aircraft accidents. Their role is particularly valuable in evaluating the injuries and also in identification of the dead. The latter exercise requires combined efforts of aircraft investigators, forensic odontologists and pathologists. Needless to say that it is a wide field and anyone interested in details may consult particular books and writings dealing with the mechanics of air crashes, types of injuries sustained and the mass casualty aspects, etc.

Sustenance of injuries in aircraft crashes vary widely, from total disintegration of the body to relatively insignificant ones. Where crash occurs at relatively high altitude, fragmented bodies may be distributed over a wide area, especially if the aircraft suddenly depressurises and there is massive ejection. Some of the circumstances influencing injuries and/or deaths may include the following:

- Failure of aircraft at high altitude is usually associated with total casualties. However, there may well be some survivors where a landing or taking-off accidents occur. (Mechanical failure of the pressurisation system of the aircraft results in **rapid** or **explosive** decompression. Such decompression may also result from external or internal perforation of the plane's hull. Loss of pressure in less than a second is referred to as explosive decompression, and if longer, it is referred to as rapid decompression. The classic examples of air crashes involving explosive decompression were in the 1954 Comet disasters in the sea of Italy. In these disasters, a defective part in a portion of the fuselage had provided a point of weakness.)

- Fire is obviously a major hazard; therefore, death may at occasions be due to burning, smoke inhalation or carbon monoxide poisoning. A raised carboxy haemoglobin may indicate:
  - burning as a cause of death or survival in fire;
  - sublethal incapacitation of the pilot as the cause of an accident;
  - an abnormality of the engine leading to accident; and
  - postmortem artefact. Dominguez (1962) concluded that fragmentation due to explosion and postmortem incineration do not raise tissue carboxy haemoglobin.
- Alcoholic intoxication of the pilot may be the other source. In International Regulations, commercial pilots must not drink within 8 hours of flying. Toxicological analysis, therefore, should always be a part of the exercise.
- A look for some natural disease in the pilot needs appreciation. In one survey in UK, 8.5% of the aviators were found to be suffering from coronary artery disease and another 15% from moderately severe coronary stenosis. The circumstances of aircraft travel are such that a fatal accident is likely outcome from even a minor acute disability. As pathognomonic changes of cardiac ischaemia take sometime to appear, actual coronary heart disease can only be inferred from the presence of the precursor condition. Mason et al. (1963) reviewed nine aircraft accidents attributed to coronary disease in pilot, based on dual evidence of pathology and history. They opined that four of these accident were almost certainly to have been so caused, four were very likely, and one was assessed as likely, and it was considered impossible to be dogmatic. **Occasionally**, accident may be due to some functional disease which cannot be documented at autopsy. Idiopathic epilepsy may be an obvious example. Hence, the autopsy surgeon may extend his inquiries into the medical and personal history of an accident fatality.
- Availability/non availability of medical facilities, ambulances and trained medical staff remains an important factor.

The high altitude at which the aircrafts operate, presents a special problem. The cabins are pressurised to prevent hypoxia. Death resulting from altitude problems can be due to hypoxia, hypothermia or dysbarism. However, such deaths being physiological in nature are usually not obvious and may be masked by superimposition of trauma. Presence of fat or bone marrow emboli in the lungs or in other tissues is usually a convincing finding for a functioning circulation for at least a brief period following trauma. Where hypoxia is suspected, lactic acid estimation is of utmost significance (brain lactic acid levels exceeding 200 mg% are indicative of hypoxia). Predisposing factors influencing the outcome of injuries could include age, obesity, exercise, ascent rate, attained altitude, nitrogen pressure before ascent or descent, previous injury and of course the temperature. The autopsy surgeon may lend valuable assistance to the investigation of the circumstances leading to death if he can distinguish between injuries resulting from impact and those arising from a catastrophe at high altitude. Philp and coworkers suggest that gaseous emboli and traumatic shock are operating jointly in the genesis of fatal post-descent shock. They report that presence of gas in the circulation leads to the formation of microthrombi and blood sludging at the blood–gas interface.

TYPES OF INJURIES

As stressed earlier, injuries vary very widely, i.e. from total disintegration of body to relatively insignificant ones. However, some commonly occurring injuries may be as under:

- Leg injuries are extremely common, the passengers being crushed against the seats in front.
- Fractures of spine are also common, especially the fractures of thoracic spine. Up to 78% of such injuries have been reported in some disasters. These are mainly hyperflexion injuries due to massive deceleration when the aircraft strikes the ground. Cervical spine injuries associated with facial injuries may be encountered due to hyperextension when the face is flung against the back of the seat in front of the victim.
- Intrathoracic injuries due to squeezing of the chest by pressure against the sternum may also occur.

The issue of identification of the victims is of critical importance. Victims may run into hundreds and therefore, causing a major organizational problem for the authorities. As stressed in the beginning, efforts of many experts are required for such purposes. Naturally, age, sex, race, stature as well as personal details like surgical scar, other scars, tattoos, surgical prosthesis like artificial limbs and congenital deformities carry importance. (The task may be eased by the availability of an accurate passenger manifest. The subject had been discussed by Stevens and Tarlton (1963), who estimated the relative W values of visual recognition, possessions, clothing, pathology, dentistry, X-rays, and the like as aids to identification.)

Dental aspects carry unique significance. Teeth are one of the few parts that resist conflagration. Dentures, metal fillings, special dental work, extractions and other dental attributes all constitute important evidence leading to identification, if pre-existing dental records can be made available. Provisions for accommodating bodies, adequate facilities for postmortem examination with photography and radiography are necessary under such situations.

Railway Accidents

India carries one of the largest railway networks in the world and accidents from rail operations may not be unexpected. Children playing in the vicinity of the rail track or pedestrians using the track as a convenient route for walking may get accidentally involved. Persons leaning too far from the windows may strike

their head upon passing railway fixtures, bridge abutments, tunnel sides or electric poles, etc. Suicides have also been reported where a determined suicide will deliberately lie across the line or even place his/her head for achieving self-destruction. A peculiarly puzzling situation may be there when a person is pushed or thrown from the speeding train, putting the doctor in a dilemma to categorically opine about the manner of death.

Railway accidents may be broadly classified into the following groups:

- Accidents where the casualties are actually to the people on board the train.
- Accidents where the people other than on board are involved.
- Accidents where the people on railway-premises are involved. Such people usually include staff and can be directly or indirectly connected to numerous occupations relating to railway affairs. (Workers working in close proximity to the high voltage overhead cables are usually at risk. Head may touch the live conductor and the current directly involving the brain, uncommon in other types of electrocution.)
- Collision between a train and another vehicle at a road-railway crossing (commonly called a 'level crossing') is another source of injuries and even deaths. Such crossings where a public road crosses a railway track are usually poorly manned with either no barrier at all or with only a flimsy lifting pole. (As per news item, '*The Tribune*' dated 3rd February, 2004, a ghastly mishap occurred when an express train ploughed through a crowd of people at the level crossing killing five persons. The gateman after managing the gate for some particular train, had to immediately down the barrier for another train that was already on the way for which, probably he had no information. This led to the trapping of persons who were crossing the track and thereby resulting in fatalities.)
- High winds, heavy rain, heavy fog and other vagaries of the weather also occasionally lead to train accidents. Rain and floods may loosen the foundation of the track and lead to accidents. Landslides may present an additional hazard.
- Hooliganism and vandalism affecting the rail track or moving trains has also been observed in the recent years. Deliberate laying of the objects on the rail tracks or throwing of objects at passing trains may form a part of the malicious strategy.

In general, any type of trauma can be seen in such accidents. However, some kinds of injuries may be more commonly seen. Extremely severe destruction of the body may occur with separation of the limbs and extrusion of organs. It may sometimes be possible to estimate distance between two sets of wheel injuries to show that the person might have stretched his body across the entire width of the standard gauge track. Certain features like wheel marks upon the body, dirt and grease contamination, and manner of severance of tissues deserve special observation. Possibility of a murdered person being placed across the tracks needs to be kept in mind. Forensic laboratory evidence can sometimes reveal a non accidental cause. The usual search of alcohol and other drugs must be made, as suicides often resort to multiple means to ensure self-destruction.

Vehicular Conflagration

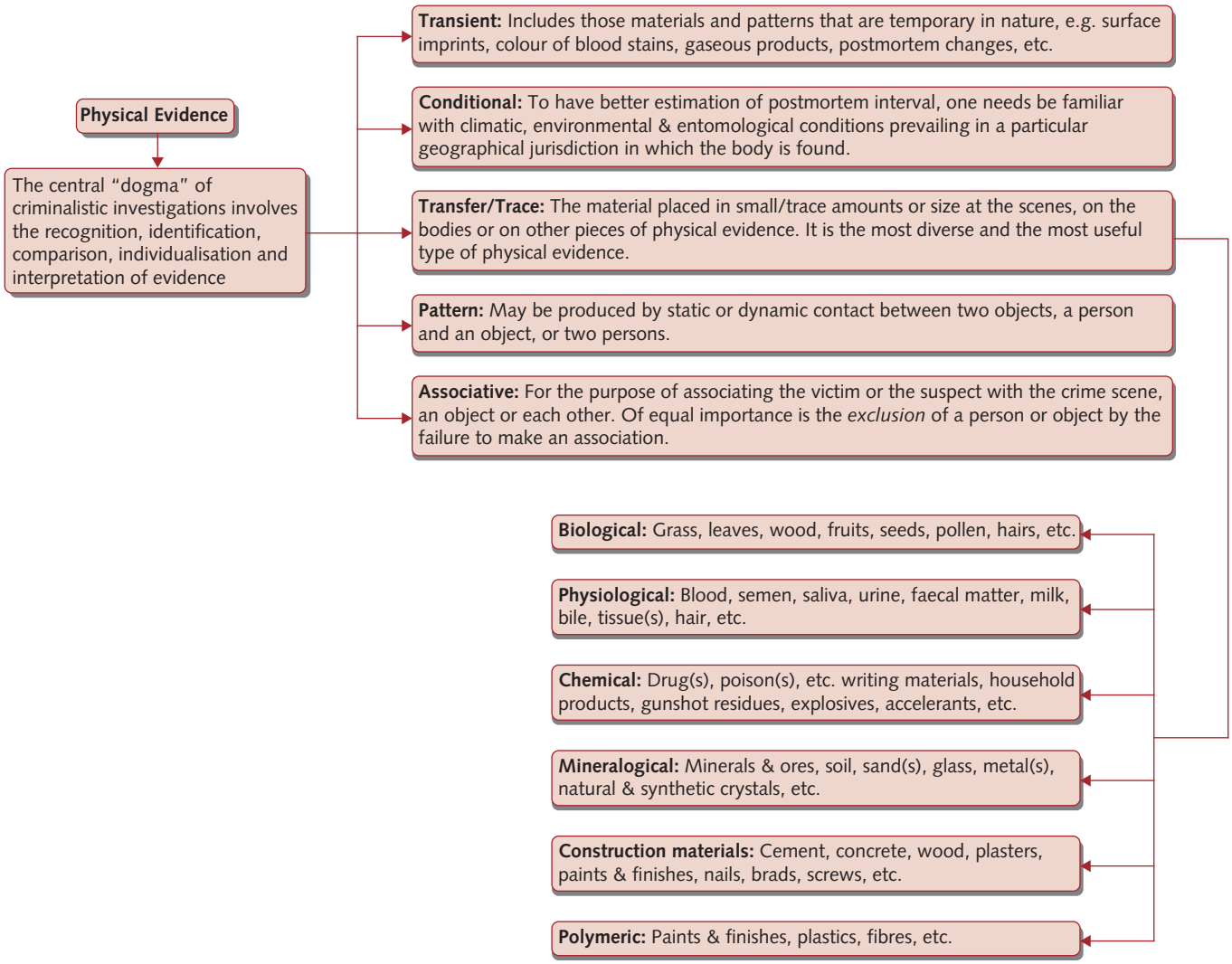
In crashes involving fire, victim's body frequently undergoes advanced burning until it is extracted. Tasks of the medicolegal experts under such circumstances usually include (*i*) identification of the decedent, (*ii*) evaluation of blunt and other trauma and (*iii*) evaluation for smoke inhalation or other indicators showing that the person was alive and/or conscious during the fire (circumstances may be there when the victim dies immediately from severe blunt force injuries, only to have the vehicle subsequently ignite and become engulfed in flames). It is difficult, if not impossible, to grossly distinguish antemortem from postmortem burns, especially in charred bodies. Pre-autopsy X-rays must be obtained in order to assess for the unexpected foreign objects like bullet or some part of the blade of some weapon, etc. X-rays will also be helpful in the identification of the decedent if some unique orthopaedic hardware or some surgically implanted devices are demonstrable. As thermal injury is notorious in modifying or destroying pre-existing injuries, one needs to exercise caution in their evaluation (heat is known to shrink tissues as the water is released and the proteins get coagulated). Ultimately, the evaluation of the fire's contribution towards death rests in the documentation of severity of injuries balanced against evidence of smoke inhalation, which is assessed by the presence and quantity of carbonaceous material in the airways and carboxy haemoglobin concentration in the blood. One should also keep in mind that other toxic gases may be produced as a by product of burning vehicular components.

Medicolegal Aspects of Transport Injuries

The goal of any criminalistic examination is to provide scientific and factual data that can link a suspect to a case or exonerate the suspect. Edmond Locard—an early 20th century criminalist—postulated, "when objects, persons or surfaces come in contact with each other, there is a mutual exchange of materials. This transfer may result in identifiable trace materials that can be used to link the objects, persons or surfaces to each other". Such trace/transfer evidence is amongst the most diverse and the most useful types of physical evidence available in the field of criminology, transportation accidents ranking high. Though the fact that death has been the result of multiple injuries is often obvious, yet the extent of litigation cannot be gauged at the time of autopsy. A driver may perpetrate a homicide against pedestrian or occupants of another vehicle by using the vehicle as a weapon or may cause the death of a passenger in his vehicle in the context of

a suicide-homicide. Psychological autopsy and postmortem toxicology may assist in the diagnosis of "traffic suicide"/"autocide".

In general, a mix of factors may be operating in a given scenario, viz. (*i*) factors attributable to the scene of accident (nature of the surface, material lying there and the nature of material, etc.); (*ii*) factors attributable to the vehicle (condition and design, speed, supervening factors like running over by another vehicle or conflagration etc.); (*iii*) factors attributable to the environmental condition (extreme hot weather, heavy rains, too cold and foggy weather, etc.); (*iv*) factors attributable to the victim (location and seating of the victim in the vehicle, ejection/nonejection of the victim, etc.); and (*v*) eventual role played by some disease in the driver including toxicological evaluation. It may be worth mentioning that absence of signs of ill health, even the absence of physical signs of disease, by no means exclude the possibility of its presence and, indeed, may have been there for sometime and may become revealed by some accidental happening. Peptic ulcer, hypertension, coronary artery disease, diabetes, neoplasm, etc. may be a few examples wherein the disease may have progressed for some period in the past without giving rise to symptoms or attracting attentions of either the victim or of those with whom he has been in contact. Therefore, a detailed documentation of autopsy findings including scaled photography, collection and dispatch of specimens/samples including trace materials to the forensic science laboratory (Flowchart 19.1), and critical evaluation of ultimate data/findings (through multidisciplinary approach) in the light of circumstances will go a long way in reconstruction of events, especially in 'hit and run'/'hit and skip' accidents.



Flowchart 19.1 Categorisation of physical evidence. Based on Locard's exchange principle, 'trace/transfer' evidence is amongst the most diverse and the most useful types of physical evidence in investigations.

20 CHAPTER

Medicolegal Examination of the Living

After going through this chapter, the reader will be able to describe: Clinical forensic medicine | Medicolegal examination of the victim of assault and extending opinion | Classification of sexual offences | Meaning and scope of the offence of 'rape' with particular emphasis on implications of consent/nonconsent | Medicolegal examination of the victim and of the alleged accused of rape and extending opinion in either case | Medicolegal examination in 'unnatural sexual offences' and extending opinion | Medicolegal aspects of 'semen' | Acid phosphatase test and its medicolegal importance | Medicolegal importance of pregnancy | Presumptive, probable and positive signs of pregnancy | Differential diagnosis of pregnancy | Surrogate motherhood | Child abuse

Forensic medicine may be considered as the medical science that applies the principles and practice of medicine to the elucidation of various queries in judicial proceedings. It means that there must be as many specialities as there are in the medical practice. It would, therefore, not be apt to view the subject of forensic medicine from the autopsy table alone. There are numerous occasions when a doctor is called upon to examine a living person, for medicolegal purposes, sometimes for the benefit of the examinee but sometimes to his disadvantage, such as examination of an accused person. Whatever may be the case, the question of consent should never be forgotten and only in exceptional circumstances it may be dispensed with, as enumerated in the Chapter, 'Consent to and Refusal of Treatment'.

This aspect of examination of a living person for medicolegal purposes may be termed as **Clinical Forensic Medicine**, as there exist a number of circumstances for his medicolegal examination. The victims of an assault, sexual offence, accidents, drunkenness, etc., all require examination and a report upon their conditions so that the legal proceedings may be initiated. In accidents, examination is invited for the injuries suffered and the opinion on prognosis, so that the matters of insurance and compensation may be evaluated. Insurance companies in many cases require a medical examination in order to assess the insurance risk of an applicant. Examination of the suspected malingerers and examination for issuing certain certificates like age certificate, certificate for disability, certificate for entry into service, for driving purposes, for taking part into national and international games, for certain admissions and certificate for illness, all add to the numerous cases for which the medical examination is required for legal purposes.

At the very outset, it needs be stressed that the record must be complete and the report must be prepared after acute observation because record is the 'measure' by which it is judged at a later date, may be after years in occasional cases.

Medicolegal Examination in Assault Cases

CONSENT

Examination of a patient or a victim of assault should not be made without his permission or of parent/guardian. However, a person accused of criminal offence may be medically examined without his consent on the request of the police (Section 53, CrPC, 1973). Details may be seen in the Chapter entitled 'Consent to and Refusal of Treatment'. In the medicolegal reports, as prevalent in Punjab, Haryana, Himachal Pradesh and Union Territory of Chandigarh, there is specific space on the left side of the report, where the doctor usually records the consent in the manner described in relevant annexures (i.e., Annexures 2–5).

HISTORY

It may include **general history** in the form of any past or present illness, any medication, any history of operations and the usual questions relating to matters of occupation, hobbies, height, weight, family history, etc. It also includes the **specific history** relating to the particular situation for which the examination is being undertaken. Some kind of story is normally supplied by the agency requesting the examination, but it is always preferable to take it from the concerned individual or his/her close relatives, and the doctor should amplify it as much as possible

by putting them questions. It must include exact nature, place and the associated factors of the incident. The question of admissibility of evidence is a matter for legal authorities, but the doctor is entitled to write down in his report anything that he thinks relevant.

GENERAL PHYSICAL EXAMINATION

General physical examination should be complete from 'top to toe' including observations of height, weight, general built and appearance, skin of the entire body surface showing any trace evidence or any superficial injury, healing injury or old scar, deformity/congenital defect, etc. Size, site and orientation of the injuries must be described with reference to the well-known surface landmarks. It is preferable to have photographs or at least sketches, which may be of great value at a later date when the case is being dealt with in the court. It is not advisable for any doctor to pounce upon the specific part involved in the incident without carrying out complete general examination.

EXAMINATION OF SPECIFIC AREA

Examination of the specific area involved in the incident should follow the general physical examination. It may range from mere palpation of any fracture/deformity, measurement and detailed description of the injuries including their exact location and orientation, taking all relevant specimens and advising necessary investigations.

REFERENCE TO A SPECIALIST

No doctor can be an expert in every field and the modern tendency is to achieve specialisation in the various branches of medicine. Thus, a doctor should preferably refer the matter to a surgeon or orthopaedician or neurologist, etc., depending upon the merits of each case to have a comprehensive view of the case. Many medicolegal issues, especially concerning compensation for accidents or insurance matters, depend heavily upon the future outcome of any disability and it is always advisable to have the advice of a specialist in such cases.

OPINION

It is to be given at the end of the examination and must be based upon its findings. It may sometimes be withheld till the reports from the specialist (in cases where something has been referred to some specialist) or reports of the X-rays or laboratory investigations are at hand. The opinion consists of three constituents:

- Nature of injuries
- Probable duration of injuries
- Kind of weapon used in inflicting injuries

Nature of Injuries

Nature of injuries needs to be classified as **simple, grievous** or **dangerous**. Some books have mentioned that a doctor need not classify the injuries in the report and his opinion on them is only to guide the investigating officer, but the author is of a different view. The injuries should better be classified and mentioned under the proper column of the medicolegal report, after taking all findings into consideration. Agreed that the ultimate outcome rests with the court, but the court in turn is to be assisted by the evidence of the doctor. Not classifying or declaring the injuries can raise many undesirable queries or assume unpleasant situation in certain cases, and the doctor may invite unnecessary pressure/counter pressure. Therefore, it would be in the fitness of things to declare the nature of injuries after consulting the entire record and if need be, after consulting some senior colleague or the literature available on the subject.

Approximate Duration of Injuries

The approximate duration of injuries should be mentioned after observing the age-related changes in the injuries. The age of the injury is important, because its appearance may or may not correspond to the time when it is alleged to have been inflicted and furthermore, all the injuries found on a person may not have been produced on the same day. The words 'approximate' and 'duration' are significant, as there is no scientific method available that can yield precise results. Indeed, the degree of reluctance of the examiner to pinpoint the time interval may be a measure of his/her competence.

Kind of Weapon

The kind of weapon in many cases does not pose any problem. Examination of the wounds on the body and defects on the clothing sufficiently speak of the kind of weapon, i.e. whether blunt or sharp or blunt-pointed/sharp-pointed or firearm or dry/moist heat and the like. In some cases, wounds produced by broken pieces of glass/earthen wares or by teeth, etc. or wounds produced on body prominences may present some difficulty but examination by a hand lens and the experience of examiner will help to resolve the issue.

DISPATCH OF SPECIMENS/ARTICLES

Manner of collection of specimens and their proper dispatch is also vital, which ensures the 'pedigree' of any specimens taken, and maintains the chain of events for that particular specimen.

EXAMINATION OF EXHIBITS

Weapon

If a weapon alleged to have been used in producing injuries is brought by the police, its length, breadth, shape etc. need to be documented. Particulars of the handle and blade (wherever necessary) should be noted down in details. It should be examined for marks of bloodstains or fragments of hair, fibre, pieces of clothes, etc. adhering to it and be returned to the police in a sealed parcel/packet duly labelled with the particulars of the case under due receipt mentioning date and time.

Foreign Bodies

When any foreign body such as some splinter of broken glass, a piece of some stick/rod, broken point/portion of some instrument/weapon, bullet (whether deformed or broken), pellet (whether deformed or broken) or wadding of a firearm or remnant of some clothing found lodged in a wound or in its surrounding tissues, it should be carefully documented, preserved and sent to the forensic science laboratory (FSL).

Clothing

Clothes need to be examined for any blood/other stain, cuts, rents, tears, soiling, or burning etc. coinciding with the wound(s)/damage(s) on the underlying parts of the body. However, these might not coincide with the wound(s)/damage(s) if the garment worn at the time of assault was very loose and was disarranged during the struggle. Care needs to be exercised in distinguishing fake firearm burns or holes preferably by having an opinion from the FSL. Clothes then be properly marked, signed and handed over to the police in a sealed cover with particulars of the case under receipt mentioning date and time (if clothes are wet, the same need be air dried before sealing).

Medicolegal Examination in Sexual Offences

Sexual offences may be considered as acts of sexual intercourse and/or sexual interference with a person or animal against the provisions of law. These may be classified into three groups:

Natural Sexual Offences

- Rape
- Incest
- Adultery

Unnatural Sexual Offences

- Sodomy
- Buccal coitus (oral coitus)
- Lesbianism/tribadism
- Bestiality

Other Sex-linked Offences

- Indecent assault
- Some unlawful perversive acts, the so-called sexual perversions
- Offences under the Immoral Traffic Act, e.g. kidnapping of a woman, unlawful prostitution, etc.

RAPE

From the medicolegal point of view, a doctor is expected to examine both the alleged victim and the alleged assailant. The routine of examination should not vary from the other cases, but it is preferable to follow some schedule rather than proceeding haphazardly, in which case some important aspects may be skipped.

The word **'rape'** is derived from Latin term 'rapio', which means **'to seize'**. Thus, rape literally implies forcible seizure. In other words, rape is violation with violence of the private person of a woman, or it may be considered 'as the ravishment of a woman without her consent, by force, fear or fraud'. Here, it would be in the fitness of things to write detailed provisions regarding 'rape' as given under the IPC. It would enable the doctors/students to appreciate the jugglery of the legal language vis-á-vis the medical findings.

Section 375 (Rape)

A man is said to commit 'rape' who, except in the case hereinafter excepted, has sexual intercourse with a woman under circumstances falling under any of the following descriptions:

Firstly	Against her will.
Secondly	Without her consent.
Thirdly	With her consent, when her consent has been obtained by putting her or any person in whom she is interested in fear of death or of hurt.
Fourthly	With her consent, when the man knows that he is not her husband and that her consent is given because she believes herself to be lawfully married to that man.
Fifthly	With her consent, when, at the time of giving such consent, by reason of unsoundness of mind or intoxication or the administration by him personally or through another of any stupefying or unwholesome substance, she is unable to understand the nature and consequences of that to which she gives consent.
Sixthly	With or without her consent, when she is under 16 years of age.

Explanation: Penetration is sufficient to constitute the sexual intercourse necessary to the offence of rape.

Exception: No Court shall take cognisance of an offence under Section 376 of Indian Penal Code, where such offence consists of sexual intercourse by a man with his own wife, the wife being under 18 years of age, if more than 1 year has elapsed from the date of the commission of the offence [CrPC (Amendment) Act, 2008 (w.e.f. 31.12.2009)].

Section 376 (Punishment for Rape)

(1) Whoever, except in the cases provided for by Subsection (2), commits rape shall be punished with imprisonment of either description for a term that shall not be less than 7 years but that may be for a term that may extend to 10 years and shall also be liable to fine unless the woman raped is his own wife and is not under 12 years of age; in which case, he shall be punished with imprisonment of

either description for a term that may extend to 2 years or with fine or with both.

The court may, for adequate and special reasons to be mentioned in the judgement, impose a sentence of imprisonment for a term of less than 7 years.

(2) Whoever
  (a) being a police officer commits rape
    - within the limits of the police station in which he is appointed; or
    - in the premises of any station house whether or not situated in the police station to which he is appointed; or
    - on a woman in his custody or in the custody of a police officer subordinate to him; or
  (b) being a public servant takes advantage of his official position and commits rape on a woman in his custody or in the custody of a public servant subordinate to him; or
  (c) being on the management or on the staff of a jail, remand home or other place of custody established by or under any law for the time being in force or of a woman's or children's institution takes advantage of his official position and commits rape on any inmate of such jail, remand home, place or institution; or
  (d) being on the management or on the staff of a hospital, takes advantage of his official position and commits rape on a woman in that hospital; or
  (e) commits rape on a woman knowing her to be pregnant; or
  (f) commits rape on a woman under 12 years of age; or
  (g) commits gang rape

shall be punished with rigorous imprisonment for a term that shall not be less than 10 years but that may be for life and shall also be liable to fine.

The court may, for adequate and special reasons to be mentioned in the judgement, impose a sentence of imprisonment of either description for a term of less than ten years.

Explanation 1: Where a woman is raped by one or more, in a group of persons acting in furtherance of their common intention, each of the persons shall be deemed to have committed gang rape within the meaning of this Subsection.

Explanation 2: 'Women's or children's institution' means an institution, whether called an orphanage or a home for neglected women or children or a widows' home or by any other name, which is established and maintained for the reception and care of women or children.

Explanation 3: 'Hospital' means the precincts of the hospital and includes the precincts of any institution for the reception and treatment of persons during convalescence or of persons requiring medical attention or rehabilitation.

Section 376 (A): Intercourse by a man with his wife during separation—whoever has sexual intercourse with his own wife, who is living separately from him under a decree of separation or under any custom or usage, without her consent, shall be punished with imprisonment of either description for a term that may extend to 2 years and shall also be liable to fine.

Section 376 (B): Intercourse by a public servant with a woman in his custody—whoever, being a public servant, takes advantage of his official position and induces or seduces any woman who is in his custody as such public servant or in the custody of a public servant subordinate to him to have sexual intercourse with him, such sexual intercourse not amounting to the offence of rape shall be punished with imprisonment of either description for a term that may extend to 5 years and shall also be liable to fine.

Section 376 (C): Intercourse by a Superintendent of a jail, remand home, etc.—whoever, being a Superintendent or Manager of a jail, remand home or other place of custody established by or under any institution takes advantage of his official position and induces or seduces any female inmate of such jail, remand home, place or institution to have sexual intercourse with him, such sexual intercourse not amounting to the offence of rape shall be punished with imprisonment of either description for a term that may extend to 5 years and shall also be liable to fine.

Explanation 1: 'Superintendent' in relation to a jail, remand home or other place of custody or a women's or children's institution includes a person holding any other office in such jail, remand home, place or institution by virtue of which he can exercise any authority or control over its inmates.

Explanation 2: The expression 'Women's or Children's Institution' shall have the same meaning as in Explanation 2 to Subsection (2) of Section 376.

Section 376 (D): Intercourse by any member of the management or staff of a hospital with any woman in that hospital—whoever, being on the management of a hospital or being on the staff of a hospital takes advantage of his position and has sexual intercourse with any woman in that hospital, such sexual intercourse not amounting to the offence of rape shall be punished with imprisonment of either description for a term that may extend to 5 years and shall also be liable to fine.

Mechanism of Erection and Orgasm

The homologous structures of the male and female reproductive systems respond to sexual stimulation in a similar fashion (Figs. 20.1 and 20.2). The erectile tissues of a female, like those of a male, become engorged with blood and swollen during sexual arousal. During sexual excitement, the hypothalamus of the brain sends parasympathetic nerve impulses through the sacral segments of the spinal cord, which cause dilatation of arteries serving the clitoris and vestibular bulbs. The increased blood flow causes the erectile tissues to swell. In addition, the erectile tissues in the areola of the breasts become engorged.

Simultaneous with the erection of the clitoris and vestibular bulbs, the vagina expands and elongates to accommodate

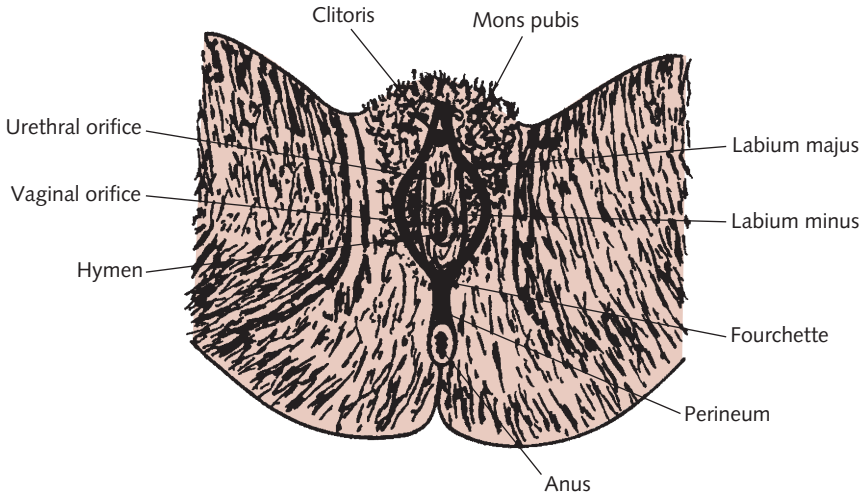


Fig. 20.1 Diagram showing external female genitalia referred collectively as vulva.

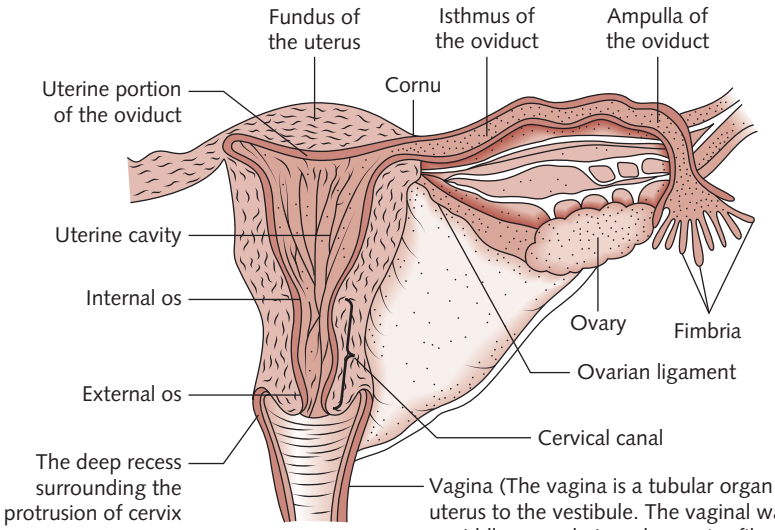


Fig. 20.2 Diagram showing components of female reproductive system.

the erect penis of the male, and parasympathetic impulses cause the vestibular glands to secrete mucus near the vaginal orifice. The vestibular secretion moistens and lubricates the tissues of the vestibule, thus facilitating the penetration of the erect penis into the vagina during coitus. Mucus continues to be secreted during coitus so that the male genitalia do not become irritated as it could if the vagina became dry.

The position of the sensitive clitoris usually allows it to be stimulated during coitus. If stimulation of the clitoris is of sufficient intensity and duration, a woman will experience a culmination of pleasurable psychological and physiological release called orgasm. Associated with orgasm is a rhythmic contraction of the muscles of the perineum and the muscular walls of the uterus and uterine tubes. These reflexive muscular actions are thought to aid the movement of sperm through the female reproductive tract toward the upper end of uterine tube, where an ovum might be located.

General Considerations

In the English law, the rule that a boy under the age of 14 is incapable of performing sexual intercourse was abolished by the Sexual Offenses Act, 1994; and 'doli incapax' (incapable of committing a crime or tort) presumption is also abolished by

the Crime and Disorder Act, 1998. **In India,** a boy of any age will be equally liable for committing the offence of rape like a man of any age unless it is proved that the boy was incapable of committing the offence medically (under the IPC, the word 'man' denotes a male human being of any age; and the word 'woman' denotes a female human being of any age). In awarding punishment, courts are guided by Sections 82 and 83 of IPC. Secondly, in India, only man can be held guilty of committing rape on a woman while in some developed countries like UK and USA, the majority of rape laws are gender blind, allowing inclusion of males too. In India, a woman may be charged to have committed 'indecent assault' on a man.

The crux of the offence of rape is the sexual intercourse against the will and without the consent of a woman. The use of two phrases 'against her will' and 'without her consent' denotes different concepts. Every act done against the 'will' of a person is done without his 'consent', but an act done without the consent of a person is not necessarily against his 'will'. A woman may be 'willing' for sexual intercourse but may not give consent for fear of detection or social stigma. Sexual intercourse with an unconscious woman cannot be said to be against her will but will be without her consent. The woman must have voluntarily participated in the sexual act after exercising her intelligence and clearly differentiating as to the resistance and assent. Whether the alleged consent by the victim was **a mere submission or a 'willing consent'** depends upon the circumstances of each case. However, when the victim is below 16 years of age, sexual intercourse, in any case, amounts to rape and the question of consent or nonconsent does not arise. Some authors describe this as **statutory rape**.

Changes in the Law

- **Recording of statement of the victim** shall be conducted at the residence of the victim or at the place of her choice in the presence of her parents or guardian or near relatives or social worker of the locality and as far as practicable, it should be recorded by a woman police officer. Further, the statement may also be recorded by an audio-video electronic means.
- **When the trial relates to** an offence under Sections 376 and 376-A to 376-D of the IPC, the trial shall be conducted as far as practicable by a court presided over by a woman.
- **Attaching report of medical examination of the woman** has been made mandatory while presenting the report/challan to the magistrate empowered to take cognisance of the offence relating to Section 376 and 376-A to 376-D of the IPC.
- **Trial in camera:** Section 327 of the CrPC has been amended making the provisions for trial of rape cases or an offence under Sections 376-A to 376-D of the IPC in camera and prohibition of publication of trial proceedings in such cases without the prior approval of the Court or subject to maintaining confidentiality of name and address of the parties.
- **Presumption as to absence of consent in certain prosecutions for rape:** The Evidence Act was amended by inserting Section 114-A, which lays down that in a prosecution for rape under Clause (a), (b), (c), (d), (e), or (g) of the Subsection (2) of Section 376 of the IPC, where sexual intercourse by the accused is proved and the question is that whether it was without the consent of the woman alleged to have been raped and she states in her evidence before the Court that she did not consent, the Court shall presume that she did not consent; thus shifting the burden of proof of innocence on the accused.
- **Character assassination of prosecutrix prohibited:** Through amendment of 2003 (Act 4 of 2003), a provision to Section 146 of Indian Evidence Act was inserted reading as, "in a prosecution for rape or attempt to commit rape, it shall not be permissible to put questions in the cross-examination of the prosecutrix as to her general immoral character".
- **Intercourse by public servant with woman in his custody:** Section 376-B to 376-D of the IPC were introduced to comprise a group of Sections creating a new species of rape, the so called **custodial rape** wherein the offence is committed by those persons who happen to occupy supervisory positions indulging in having sexual intercourse with a woman in his custody (or in the custody of a public servant subordinate to him) by inducing or seducing the woman after taking advantage of his official position.

Pre-requisites for the Examination of Victim

Some pre-requisites must be met before marching to the actual examination. The Supreme Court disapproved the refusal of some government hospital doctors (particularly in rural areas, where hospitals are few and far between) to conduct any medical examination of a rape victim unless the case of rape is referred to them by the police. Such a refusal to conduct the medical examination necessarily results in a delay in the ultimate examination of the victim by which time the evidence of rape may have been washed away by the complainant herself or be otherwise lost:

- A requisition for the examination of the victim should come from an authorised person (if the victim reports directly, she needs to be examined after obtaining due consent and the police information to be sent immediately afterwards).
- An authorised person should identify the victim about whom there should be a mention in the request. Two identification marks should be noted in addition.
- Consent should be obtained if the victim is of 12 or above 12 years of age but if she is below 12 years or is of unsound mind or is intoxicated, consent should be obtained from her parents or legal guardians. Further, the consent must be 'informed consent' as she must be told that any evidence obtained may be used in the court and may go for or against her.
- Presence of adult female attendant/nurse during the entire examination. Sometimes, the victim may request to be

examined by a female doctor. This is understandable as the victim having undergone horrible experiences may behave inimically being intimately examined by a man. However, due to insufficient number of female doctors, particularly in peripheral areas, the victim can be examined by any registered medical practitioner preferably employed in some government hospital (Section 164A of CrPC; see under the Chapter, 'Consent to and Refusal of Treatment').

History Taking of Victim

It should include general as well as specific history.

General History

- Any history of past operations.
- Details of any medication or alcoholic intake during the past 24 hours.
- Enquiry as to the past sexual experience, particularly acts of any consenting sexual intercourse because finding of evidence of recent sexual activities, especially semen, might have been due to this legitimate sexual activity during the previous few days and may pose a problem in interpreting findings in the present case.
- Menstrual and obstetrical history with special reference to the last menstrual period, the type of menstrual protection normally used, use of any hormonal or contraceptive medication, any surgical involvement in the past delivery (episiotomies, forceps delivery, etc., may alter the normal genital anatomy and may have some relevance to the pattern of genital injury).

Specific History

- Place, date and time of alleged act.
- Date and the time of instituting the complaint, and an explanation of any delay in the complaint.
- Clothing: whether same or changed. If same, careful observation must be made. And if changed, by whom and whether washed or not?
- History of ejaculation.
- Did the victim struggle, scream or injure the assailant in any way.
- Has the victim taken bath or washed any part of her body since the alleged act.

A general observation must be maintained as to the patient's/victim's demeanour as it may play an important role when all the findings are weighed together. It may also help in evaluating the mental status of the victim.

Examination of Victim

It may also be subdivided into **general and specific**. The victim should be made to stand upon a white sheet of paper so that anything that falls from the clothing or body surface can be collected and preserved for further investigations. Any area of soiling or damage should be noted. If garments are wet, it is better to hang them in a safe place for drying because packing wet garments may affect subsequent laboratory investigations. They should be packed in a clean paper bag without undue folding so that the soiled areas remain safe.

General Examination

It should include the patient's height, weight, general built, routine examination of all the systems. Examination of teeth and secondary sex characters is of particular importance in assisting the determination of approximate age and general character of the victim.

Skin Skin needs to be examined carefully from the top of the head to the soles of the feet. Any soiled area should be cleaned with cotton swabs, moistened with sterilised water. They should be air dried before being packed in sterilised containers for laboratory investigations. The use of ultraviolet lamp will reveal the areas of fluorescence on the skin that may represent areas of seminal soiling, and all such areas also need swabbing.

During the examination of the skin, search is required to be made for any loose hair or any other foreign substance on the skin surface. If found, should be collected and preserved for further investigations.

Fingernails demand close examination. Presence of ragged or broken nails, any chipping of nail polish/varnish, etc. should be observed. General shape and configuration of the nails should also be noted. Blood or even skin tags may be found under the nails, which need to be scrapped out carefully and sent to the FSL for blood grouping or even DNA profiling, which may match a suspect later in the investigations.

The whole body surface must be scrutinised for injuries including any old injury (Fig. 20.3A). Specific attention should be paid to the recent injuries. The following injuries, in isolation or in varying combinations, may be encountered:

Abrasions, although minor injuries in themselves, may carry importance. These injuries may be the result of fingernail marks or frictional movements against some hard rough surface or of scratches by thorns, grass or other foliage. Abrasions on the flanks may be the result of forcible and rough pulling down of underclothing of the victim by the assailant.

Bruises are often considered an important corroborative sign of use of force, and their exact size, shape and position should be recorded. The fact that they change their colour with the passage of time and also that they may not appear on the skin surface for up to a few hours after the injury should be kept in mind. Of particular importance are the small, roughly circular or oval fingertip type bruises consistent with grasping injury and may sometimes be associated with an opposing bruise on the opposite side of the limb or neck, caused by contralateral digital pressure. Bruising on the inner surface of

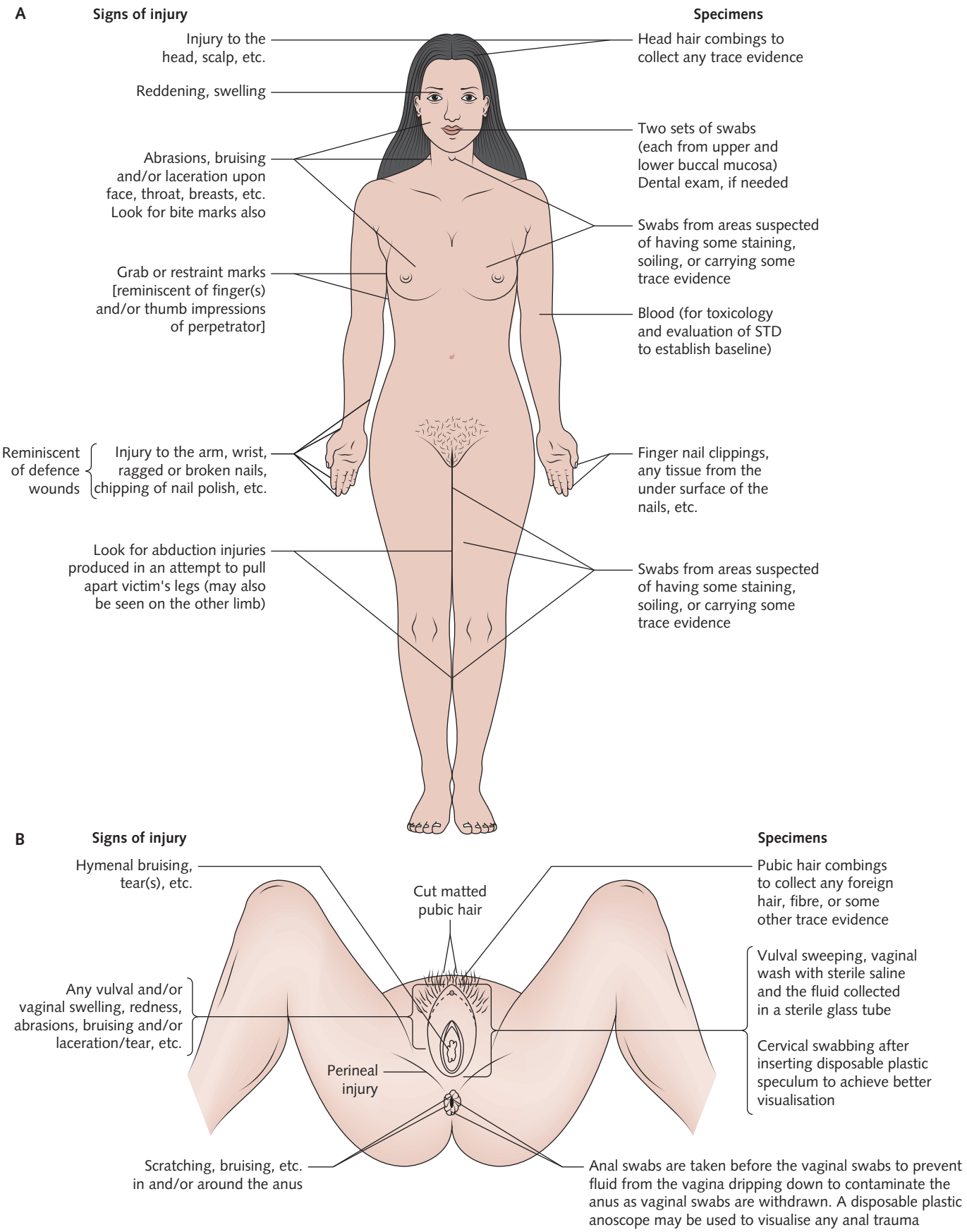


Fig. 20.3 Examination and collection of specimens in a victim of rape: **(A)** general physical examination and **(B)** local examination.

the thighs or knees, if seen, may be suggestive of victim's legs being forced apart by the pressure exerted by the assailant. Ultraviolet lamp has a place in the examination of bruising too. Before the colour changes are apparent, areas of extravasated blood beneath the skin can be appreciated if the area is illuminated by ultraviolet light.

Lacerations and incised wounds are less common but when present, should be extended due attention.

Bite Marks Bite marks may either by produced during excessive demonstration of love/enjoyment/passion where they usually present as an area of reddish-purple discolouration showing tiny haemorrhagic dots with haphazard distribution produced by suction coupled with pressure or the marks may be result of robust infliction where they are the result of strong apposition of teeth leaving an impact abrasion and occasionally laceration. In the former case, they may imply 'willing participation' in the act whereas in the latter case, it may imply infliction against resistance. Opinion of a forensic odontologist may be sought.

In the context of bite marks, examination of the lips of the victim also demands attention. It is inner surface, which is usually involved with blows to the face or by the pressure of hand/hands across the mouth to prevent screaming or by violent attempts at kissing the victim.

Under general examination, **eyes also deserve observation**. The pupils and the reflex activity give an indication about intoxication or concussion following a blow or any other blunt force impact to the head. Redness and swelling of the eyelids and general suffusion of the conjunctivae may be consistent with history of prior crying. Petechial haemorrhages on the conjunctivae, eyelids and the skin of the face may be due to pressure on the throat during the act of the assault.

Specific Examination

Examination of the genital area should be carried out in good light with the patient in a comfortable position so as to allow full exposure of the genital area (Fig. 20.3B). The usual schedule to be followed may be as under:

Pubic Hair The pubic hair should be inspected; any matted hair, if present, should be cut away as close to the skin surface as possible and retained for laboratory examination. Entire pubic area should next be combed and any evidence available collected. For comparison purposes, some pubic hair should be pulled out so that the root characters are available for comparison purposes. Hair often may be transferred between the parties by the contact necessary during the act.

The species of hair and the part of human body from where the hair had come can usually be determined by microscopic examination. The advent of Neutron Activation Analysis (NAA) has led to many claims of knowing individuality of the hair sample(s) but as yet it is not regarded as a method of positive identification.

Tops of Thighs, Vulva, and the Perineum These should next be examined and any area of injury or soiling be noted. Swabs from the introitus, perineum, and the anal margins should be taken before any digital contact has been made for examining the area. A brief account of injuries ordinarily expected at various age groups needs some description.

At the outset, it needs be remembered that there is great variation in the appearance of the external female genitalia, with age, sexual maturation, body size/shape, and other attending factors depending upon the circumstances of each case. As a general rule, the younger the individual, the more prominent the tissue of the unoestrogenised genitalia and further, the more likelihood of these becoming contused, abraded, or lacerated. Injuries (or lack thereof) need be carefully recorded and documented photographically.

In a child victim, sexual violation prompts skilled investigations. History in this context needs efficient documentation so as to have deductions appropriate to the victim's age. Major reason for paucity of diagnostic findings in such cases is the fact that children being reticent about reporting such conduct and in the event of reporting, frequent delays become instrumental in letting the acute changes fade away. Since it is difficult to visualise hymen in the children (because of its deep seated position), efforts need to be taken so as to record the findings effectively. (It may be necessary to apply either some local anaesthetic solution to the parts, or to administer general anaesthesia.) Additionally, vagina being very small, penetration of adult organ is usually prevented. As such, assaults on children mostly involve only fondling, simulated intercourse such as intercrural connection (i.e., penile friction between the inner thighs and external genitalia), or, oral or anal penetration. Hymen, therefore, is usually found intact and there may be redness plus tenderness of vulva. However, where there has been penetration, bruising and/or tears of anterior and posterior vaginal walls can occur. The hymen may be partially or wholly destroyed or may show bruising and/or lacerations.

In a prepubertal victim, vaginal penetration usually results in tearing of the **hymen** in the posterior 180°, i.e. between 3 and 9 o'clock position (see Case of a child victim as shown in Fig. 20.4). These lacerations/tears may be associated with bruising or abrasions ventrally as well as to the posterior fourchette. The hymenal tears usually heal within 5 or 6 days and become shrunken and look like small tags of tissue after a week to 10 days. **The labia** may be red and inflamed. Usually some oedema of the vaginal introitus is present. Frank injury to the labia is not common, but some scratches/abrasions may be occasioned through scratching the part in case of poor hygiene of the area. Swelling and congestion of the mucosa at the introitus, the clitoris and the labia minora may also be caused by digital stimulation or masturbation. Minor tears may be seen in the regions of fourchette and fossa navicularis produced by excessive stretching of the skin. **Vaginal walls** need careful scrutiny. There may be abrasions, bruising, lacerations

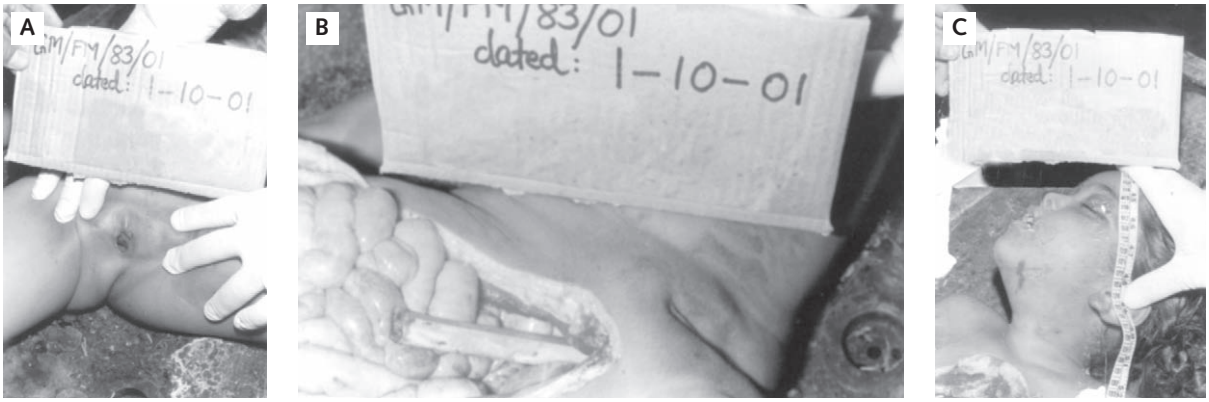


Fig. 20.4 **(A)** Injury to the genital area. **(B)** Abdominal cavity showing presence of 'sugarcane *pori*'. **(C)** Swollen and congested face of the victim showing injury to the inner surface of the lips and side of neck including the adjoining facial area as a result of alleged 'smothering'. The victim, about 10–year-old female child, was taken by the alleged assailant (a known person to the family) who tried to perform sexual intercourse with the victim but however, failed to introduce the organ. He then attempted to widen/dilate the genital passage by introducing '*sugarcane pori*'. To this, the victim cried helplessly. The alleged assailant becoming panicky, smothered the child to death (contributed by Dr. GS Mann, et al.).

or any permutation and combination of these. Bruising of the vagina is seen as dark red areas against overall redness of the vaginal mucosa (within 24 hours, the colour becomes deep red or purple). It is more frequently seen on the anterior vaginal wall in the lower third, and on the posterior wall in the upper third. Bruising of this nature tends to substantiate penile penetration than the digital penetration. Frank lacerations can occur if there is gross disproportion between the penis and vagina, or in cases where some foreign bodies have been inserted into the vagina. Lacerations are usually seen in posterior fornix of the right side of the vault, and less frequently in the left side. In cases where there are no fresh injuries, vaginal examination needs to be conducted to assess (*i*) the laxity of the vaginal orifice, (*ii*) the length of the vagina into the posterior fornix, (*iii*) the number of fingers that can be introduced through the hymenal orifice and (*iv*) the areas and the degree of tenderness, etc. Such examination helps the examiner to assess elasticity of the hymen and to determine the degree of penetration that would be possible without its rupture.

In a postpubertal victim, oestrogenisation of the hymenal tissue provides some protection from the injury associated with intercourse and sexual assault. The oestrogenised hymen is elastic, and penetration can occur without leaving lacerations or disruptions. The degree of any damage will depend upon the elasticity and/or pre-existing dilatation of the hymenal ring, disproportion between the penis and vagina and the amount of force used.

In a postmenopausal victim, injury to the genitalia is frequently sustained because the nonoestrogenised atrophic mucosa is relatively dry and friable, and therefore, gets easily traumatised. Further, as the elderly may develop bruising with less force than do normal healthy younger adults, trauma may be found with greater regularity (however, physical injuries must not be interpreted in a vacuum and their differentiation from changes occasioned through ageing and disease processes need be taken into consideration for ultimate interpretation). Frequent sexual intercourse and parturition usually destroys the hymen which is then represented by several small tags of tissue, called as **carunculae hymeneals or myrtiformes**.

Hymen One area of frequent confusion to medical investigators is normal variation in human morphology. The hymen is no exception (Lincoln C, Genital injury: is it significant? *Med Sci Law* 2001;41(3):206–16). The hymenal tissue undergoes distinct changes from the newborn period to the puberty and afterwards. In the newborn, it is thickened and redundant under the influence of maternal oestrogen, changing to nonoestrogenised state of 'scanty' hymen of childhood. At puberty, oestrogenisation of the hymenal tissue makes the tissue elastic and accommodative as detailed above. More commonly, it is an annular or ring shaped, moderately elastic membrane about 1 mm thick with a connective tissue core and stratified squamous epithelium on either surface. It is usually deficient anteriorly and most pronounced posteriorly. It has a central or ventral opening that in due course provides outlet for the menstrual flow. Several distinct variants have been described:

- **Annular**—a circumferential ring of thin or thick tissue, perforated near the centre.
- **Semilunar**—this common variant has a crescentic shape with concavity upward.
- **Septate**—representing as a band of tissue running down the middle.
- **Cribriform**—multiple naturally occurring perforations through to the vaginal canal.
- **Microperforate**—showing large posterior component, with a tiny opening into the vaginal canal.
- **Fimbriated**—showing fringed edge.
- **Vertical**—with a vertical slit-like opening.
- **Imperforate**—absence of opening.

Studies have shown that notches or clefts are often found on hymenal edge/margin, and the examiner must be careful not to let the normal anatomical variants be a confounding error in the interpretation. Natural notches are usually symmetrical, occur anteriorly, do not extend to the vaginal wall, and are covered with mucous membrane. Tears caused by sexual intercourse or by foreign body are usually situated posteriorly, or in the midline, and usually extend to the vaginal wall and are not covered with mucous membrane.

Rupture of hymen on first penetration is common but not inevitable because the thin elastic membrane is quite capable of stretching to accommodate penetration by erect adult penile organ. Furthermore, nonrupture may be on account of incomplete penetration or tough fleshy nature of the hymen or large hymeneal opening due to practice of masturbation or due to deeper placement of hymen as in young children. Smith and Fiddes report of finding an unruptured hymen in a woman who had been a prostitute for three months. **Contrarily,** there can be causes of rupture of hymen other than the intercourse, since masturbation, trauma (especially in athletes), mechanical dilatation, surgical operation, gynaecological examination, foreign body insertion (sola pith being introduced into the vagina for rendering young girls fit for sexual intercourse—*aptae viris*), ulceration due to any cause, scratching of the parts due to irritation from lack of cleanliness, etc. may affect the condition of the hymen. During forcible penetration, evidence of rupture of hymen may be forthcoming but the character and extent of the injury will vary in different cases depending upon the nature of the hymen, disproportion between the male and female parts, the extent of penetration and the amount of force applied. Rupture of hymen is almost always associated with some degree of bleeding, the amount of which will depend upon the extent of the injury and vascularity of the area. If the quantity of blood found upon the girl's underclothing/clothing and at the place where the crime had allegedly been committed, appears to be greater than would reasonably have been expected from an injury to the hymen, one may be led to suspect that assailant might have received injury to his genitals too. Apart from such injury to the male, coitus may cause considerable bleeding where a small hymenal vessel has been incompletely torn. The severed edges do not unite but become rounded off in the process of healing, which may occur in 2–3 days if the tear is slight but more extensive tears may take a longer period to heal. It is not possible to date the injury of the hymen after it has completely healed. In women who are habituated in sexual intercourse and who have borne children, the remains of the hymen constitute what are known as **carunculae myrtiformes,** which are situated around and close to the vaginal orifice presenting an appearance of small, different sized fleshy projections.

Above all, **it is virtually impracticable to differentiate tears from digital penetration from those of penile penetration,** though it is considered that tears due to digital penetration are often incomplete and not extending to the margins of the hymen but the penile tears due to limited penetration may also show similar findings. Frequently, there may only be abrasion or/and bruising of the hymen without rupture. Fingernail scratches may be seen, as well as general abrasions and redness or intradermal bruising from manual manipulation may be present. Extent of bruising, elasticity of the hymen, size of the hymenal orifice and the size of the penile organ (if the suspected assailant is also available for the examination) should be taken into consideration for differential diagnosis.

From the above description regarding hymen and its rupture/nonrupture, it may be inferred that while recent rupture of hymen would suggest the introduction of an instrument of some kind and while loss of hymen does not necessarily indicate loss of virginity, its persistence does not unequivocally suggest existence of virginity (Table 20.1). **To have some satisfactory evidence of virginity,** various signs like intact hymen, a normal condition of fourchette plus posterior commissure and a narrow vagina with rugose walls must be considered conjointly. Depicting a wide variance in the extent and character of local injury in cases of sexual assaults, a case has been reported in Glaister's *Medical Jurisprudence and Toxicology* (10th ed.) where a girl of muscular build, aged about 16, alleged that forcible and complete intercourse had been performed on her by five men on ten occasions within 4 hours but no recent hymenal tear was demonstrable. In the same book, it has also been reported that in a series of 36 cases involving young females, only 16 showed evidence of recent rupture of hymen. The ages of the girls varied from 13 to 16 years.

Sexually Transmitted Diseases (STDs) The diseases for which the victim appears to be at risk may include (*i*) gonorrhoea, (*ii*) chlamydial infection, (*iii*) syphilis, (*iv*) genital warts, (*v*) genital herpes, (*vi*) chancre and (*vii*) trichomoniasis. Hepatitis B and HIV infection may also be considered under prevailing scenario. To exclude gonorrhoea, chancroid, syphilis, etc., thin films to be made from the smears taken from low and high vaginal passage, gently dried and sent to microbiologist in a sealed container with particulars of the case for further processing and evaluation. Blood sample should be taken to establish baseline and repeated after appropriate interval (depending on incubation period of each disease) to exclude the same. The STD can be attributed to the victim when (*i*) the accused is also suffering from the same disease, (*ii*) the disease appeared in the victim after its known period of incubation after the alleged sexual assault and (*iii*) the victim was not suffering from the disease prior to the assault (incubation period of gonorrhoea is usually 2–8 days but may vary from 1 to 15 days; that of syphilis is 2–8 weeks, the average being 25 days; and that of chancroid varies from 3 weeks to 3 months).

Opinion

Rape is not a medical diagnosis, it is a legal provision enshrined under Section 375 of the Indian Penal Code. No doctor can be

Table 20.1 Differentiating Points between Virginity and Defloration (True and False Virgin)

Virginity	Defloration
May be defined as 'the state of being *virgo intacta*', i.e. a woman who has never had experience of sexual intercourse.	Refers to loss of virginity, i.e. a woman having had experience of sexual intercourse.
Hymen is a membranous structure, varying in position, consistency, structure and shape. **In children,** it appears to be situated deeply because of the rotundity of the labia majora due to their excessive fat content. It barely admits tip of little finger in them. Shortly after puberty, it reaches the adult form and is situated at the orifice of vagina, partially closing it. **In adults,** when the edges of the hymenal orifice/opening are stretched and it barely admits one finger, the presumption is in favour of virginity.	Usually ruptured. **Exception being false virgin** wherein hymen being thick, fleshy, or fibro-elastic, loose and edges undulated, it may remain intact in spite of repeated sexual intercourse. And, the hymenal orifice/opening may allow two fingers to pass through easily. In such cases, **accessory signs of virginity** (as outlined below in this table) need be considered to arrive at some satisfactory opinion as to whether one is dealing with the true virgin or false virgin.
Vagina is a tubular organ about 9 cm long passing from the cervix of the uterus to the vestibule. Vaginal wall is composed of three layers: an inner mucosa, a middle muscularis and an outer fibrous layer. The mucosal layer is thrown into transverse folds called vaginal rugae/rugosities. In a virgin, vagina is pinkish in colour, sensitive to touch, and its walls are approximated. **Vaginal rugae** are well-pronounced.	After repeated sexual intercourse, vagina lengthens into posterior fornix and the rugae/rugosities become less obvious so as to enable one to say that the vagina does or does not appear to be used to sexual intercourse (such changes are not usually produced with the regular use of tampons or digital stimulation).
Labia minora are two thin folds of skin within the labia majora (i.e., they are covered by labia majora). Pink in colour and sensitive to touch.	Enlarged, partly pigmented and partly protrude out through the labia majoras.
Labia majora are thick, fleshy, and both side majoras are in close apposition covering the labia minora.	Less fleshy, slightly absorbed, both sides are not in full apposition exposing the labia minora.
Fourchette (lower or posterior meeting point of labia minoras)—intact and crescent shaped	May show healed tear
Posterior commissure (lower or posterior meeting point of labia majoras)—intact and crescent shaped	May show healed tear
Fossa navicularis (depression between the fourchette and the vaginal opening)—less conspicuous.	More conspicuous after repeated sexual intercourse.
Vestibule (the space between the labia minoras and above the vaginal opening)—narrow	Widen
Breasts—variable in size, firm, hemispherical with pinkish smaller areola and small nipples.	Variable in size, may be flabby or moderately pendulous, with wider areola and large and raised nipples.

Note: In the alleged rape and murder of two Shopian women, the team of doctors from AIIMS, New Delhi could reportedly rule out rape in the younger girl by observing intact hymen and the orifice/opening admitting tip of the little finger, vaginal wall being normal in appearance. Anterior and posterior commissures being normal. The team concludingly remarked (as gathered from the news reports): "there was nothing suggestive of penetration of penis or the like object through the hymenal orifice/opening".

expected to opine as to the consent or nonconsent. All that can be expected from a doctor is the results of his findings and their interpretations. The doctor should make particular observations about the following aspects:

- Any findings indicative of use of force by the assailant.
- Any findings indicative of use of alcohol or any stupefying drugs.
- Any evidence of previous sexual intercourse, i.e. the question of virginity or previous sexual experience may be raised under some circumstances.
- Time elapsed between the examination and the alleged assault.

The first three aspects have already been dealt with. The fourth needs some further elucidation. This is possible from the dating of various injuries present upon the victim and also from the findings present in and around the genitalia. In charges of rape, this aspect is of utmost importance in order to authenticate the victim's statement or to reveal discrepancies in her account. The various points in this concern may be as follows:

- Some engorgement of vaginal mucosa associated with swelling and redness of the introitus and labia minora may be suggestive of sexual contact but not conclusive of recent sexual intercourse.

- Pooling of seminal fluid in the vagina may speak of recent sexual intercourse but this pooling may rapidly drain out if an upright posture is adopted soon after ejaculation had taken place. Furthermore, there may be no pooling at all if a condom was worn or if ejaculation had taken place outside the vagina.
- In the living, the body reactions tend to clear the vagina of the foreign proteins. Motility may be maintained up to a couple of hours after the ejaculation into the vagina with the number of motile sperms gradually becoming less. Samples recovered from vagina may demonstrate identifiable sperms for as long as 48 hours and occasionally, even longer. However, the persistence of motility is variable as it is influenced by a host of factors as enumerated below:

Factors attributable to the assailant:

- Alcoholism
- Drug addiction
- Diabetes
- Inflammation of the seminal vesicles
- Hot bath or hot sponging of the genital area prior to the act. (Temperature of the scrotum is usually 3–8° C lower than the body temperature. Any prior hot bath or sponging may therefore affect the motility of the sperms. The fondness for long hot baths had been blamed by the historians for the downfall of Roman Empire!)

Factors attributable to the victim:

- Different phases of menstrual cycle have different effects on the motility of sperms, between 14 and 18 day of the cycle, motility lasts the longest in the female genital tract.
- Different areas of genital tract also have influence upon the motility of sperms. Sperms retain their motility much longer in the cervix than in the vagina because the acidic pH of vagina may rapidly destroy the motility of sperms. Specimens obtained from the uterine cavity where the pH is alkaline may include living sperms even at the end of a fortnight after insemination.

In some instances, **the accused may be azoospermic,** namely:

- The very old
- The very young
- Those suffering from a variety of diseases involving epididymis, testes or seminal vesicles or some general diseases like tuberculosis, mumps, etc.
- Those who have undergone vasectomy
- Moreover, seminal fluid of a healthy man may be devoid of sperms if he has experienced numerous ejaculations over a relatively short span of time.

Therefore, in a case where the assailant is vasectomised, demonstration of the fluid to be of seminal origin requires demonstration of acid phosphatase enzyme. Prostatic secretion element of the semen contains rich percentage of acid phosphatase than any other body fluid including vaginal fluid/vaginal secretion (vaginal secretions usually contain small quantities of acid phosphatase of the order of 340 international units per litre). It rises to about 3000 international units in about 2–3 hours after the intercourse and gradually returns to normal in about 12–24 hours. Any level higher than 340 IU indicates seminal fluid. However, with the discovery of prostate specific antigen (P-30) and seminal vesicle specific antigen (MHS-5), which are specific to human semen, **acid phosphatase test** is usually done as a screening test. P-30 is present in both normal and aspermic semen. Reportedly, it is detectable in vaginal fluid for a period of about 24 hours after the intercourse as compared to about 12 hours for acid phosphatase. When the fluid has been identified to be of the seminal origin, ultimate specificity (i.e., whether it belongs to the assailant or some other person) can be achieved through DNA testing.

Rape Trauma Syndrome

Sexual victimisation is associated with emotional, cognitive, and behavioural effects. These tend to be more chronic and severe than following other nonsexual violent crimes. The term 'rape trauma syndrome' was first described in 1970s by Burgess and Holstorm. Burgess et al. described two phases of this syndrome:

(i) An immediate or acute phase of disorganisation, characterised by emotional reactions of several kinds like acute tension together with feelings of guilt and humiliation.
(ii) A long-term or delayed phase of reorganisation during which the victim readjusts her life as far as possible. Presently, the syndrome is considered as a variant of 'post-traumatic stress disorder' (PTSD).

In the developed countries, **Rape Crisis Centres** have been established to provide counselling to the rape victims, and friends and relatives of the victim. These centres are largely staffed by volunteers, nonprofessional women (some of whom have been raped in the past), or who have been close to someone who was raped. Of late, the Apex Court of India has come to the rescue of victims of sexual assault and sexual harassment by holding that interim compensation may be awarded to a rape victim by a court of competent jurisdiction during the pendency of criminal trial. In a case from Kohima (1995), a lecturer who had married the victim (his student) and later refused to recognise her as his life partner was asked to pay interim compensation of ₹ 1000 per month to the victim until her charges of rape, cheating, and other criminal offences were decided by the trial court.

Accidents Following Rape

Death may occur as a result of rape from shock due to fright and mental emotions occasioned in an effort to overpower the assailant, or due to excessive bleeding from genital and/or perineal injuries, especially among children. Injuries may cause delayed death from septic infection after several days or weeks.

Cases have been reported where death occurred from suffocation caused by covering the mouth and nostrils or by thrusting a piece of cloth down the throat to prevent the victim from crying for help. At times, the victim is first raped and then killed to destroy the evidence and also prevent identification of the assailant by the victim. On other occasions, it could be due to some misadventure (i.e., as a result of abnormal intercourse). The victim may die of choking especially at the moment of ejaculation during oral intercourse, or be choked by a plug of wool (or similar material) placed in the mouth to retain the seminal fluid. (A case has been reported wherein death had resulted from suffocation. A plug of cotton wool was recovered from the throat at the time of autopsy and was later found to have been contaminated by semen.)

Examination of the Man Suspected of Committing Rape

In cases where the suspect is available for examination, he should preferably be examined by the same doctor who had examined the victim, as it would provide an opportunity for correlating the injuries found on the victim and physical features of the suspect. If this is not practicable, any doctor examining the suspect should follow a set schedule that embraces all the essential points of examination and collection and preservation of samples. The pre-requisites for the examination of a suspect are same as those for the victim except that here the presence of a female attendant is not necessary.

General Examination

Height, weight, general built, routine examination of the systems, body surface from head to foot for any area of soiling, stains or injuries, etc. Presence of any typical abrasions consistent with production by fingernails or any bite marks should also be noted (Fig. 20.5A).

Specific Examination

This examination again may be carried out on some set schedule. Firstly, pubic hair may be observed for any matting, foreign hair, any other trace evidence, etc. Then, penile, scrotal and perineal regions should be inspected (Fig. 20.5B).

The findings upon these regions will more or less be dictated by the time interval between the alleged offence and the time of examination. When the suspect is examined within a few hours of the alleged offence or if he has not washed or bathed since the offence, the following useful findings may be appreciated:

The penis, especially the glans and the prepuce, may appear moist due to vaginal or seminal fluid. A swab should be taken and examined. To demonstrate the presence of vaginal fluid, the glans is soaked with moist blotting paper, which is then exposed to iodine vapours. Brown discolouration of the soaked part of the paper indicates presence of vaginal epithelium, as these cells contain glycogen that turns brown in the presence of iodine vapours.

Abrasions and/or bruises may be observed on the glans and prepuce and also on the frenulum. They may be inflicted by the victim during the struggle for self-protection or for preventing the act to be established or during forcible introduction of the organ into the vagina or due to disproportion between the size of the penis and the vaginal opening.

Swabs from the urethral orifice should be taken. Faecal soiling, blood and foreign hair are most likely to be trapped in the area of the coronal sulcus, particularly in the uncircumcised even if there has been an attempt to wash the genitals after the act.

If the suspect is not circumcised, the presence of smegma around the corona glandis is considered as suggestive of absence of sexual intercourse within the last 24 hours, since it gets rubbed off during the sexual act (smegma is a thick, cheesy secretion of sebaceous glands with a disagreeable odour, consisting of desquamated epithelial cells and smegma bacilli chiefly found beneath the prepuce). Nevertheless, the presence of smegma as proof against sexual intercourse need not be considered carrying any medicolegal value, as legally, mere penetration of the vulva is enough to constitute rape and therefore, it is unlikely that smegma will be rubbed off. Further, smegma accumulates if no bath is taken for 24 hours. Conversely, during customary daily bath, the prepuce is generally retracted for washing and any accumulated smegma gets washed away. It is well-known in the medical world that examination of the smegma loses all importance after 24 hours of sexual intercourse (SP Kohli vs. Punjab and Haryana High Court AIR 1978SC1753, CrLJ 1804).

With the increasing use of condoms, one must look for a used condom. When available, blood stains and vaginal epithelial cells from the outer aspect of the condom and semen from the inner aspect may be obtained. Obviously, the most important identifying element is the documented presence of ejaculate. DNA profiling has made the identification of the assailant possible so that the retrieval of spermatozoa is more critical than ever before. In a case, a used condom containing semen of the possible perpetrator was obtained from the deceased female's genitalia. Such a specimen needs be removed with great care, the end tied so as to retain the contained fluid.

The status of the accused may be examined to determine if there is anything to suggest that he is impotent. However, it has been submitted that potency being the usual and normal state in a man, it will always be presumed to be so, unless the contrary is proved by the accused through medical documentation (see Annexure 5).

History Taking of Alleged Assailant

It also includes general and specific. Specific history here, of course, will be related to the questions regarding male. Explanation for any mark or injury upon the body must be sought.

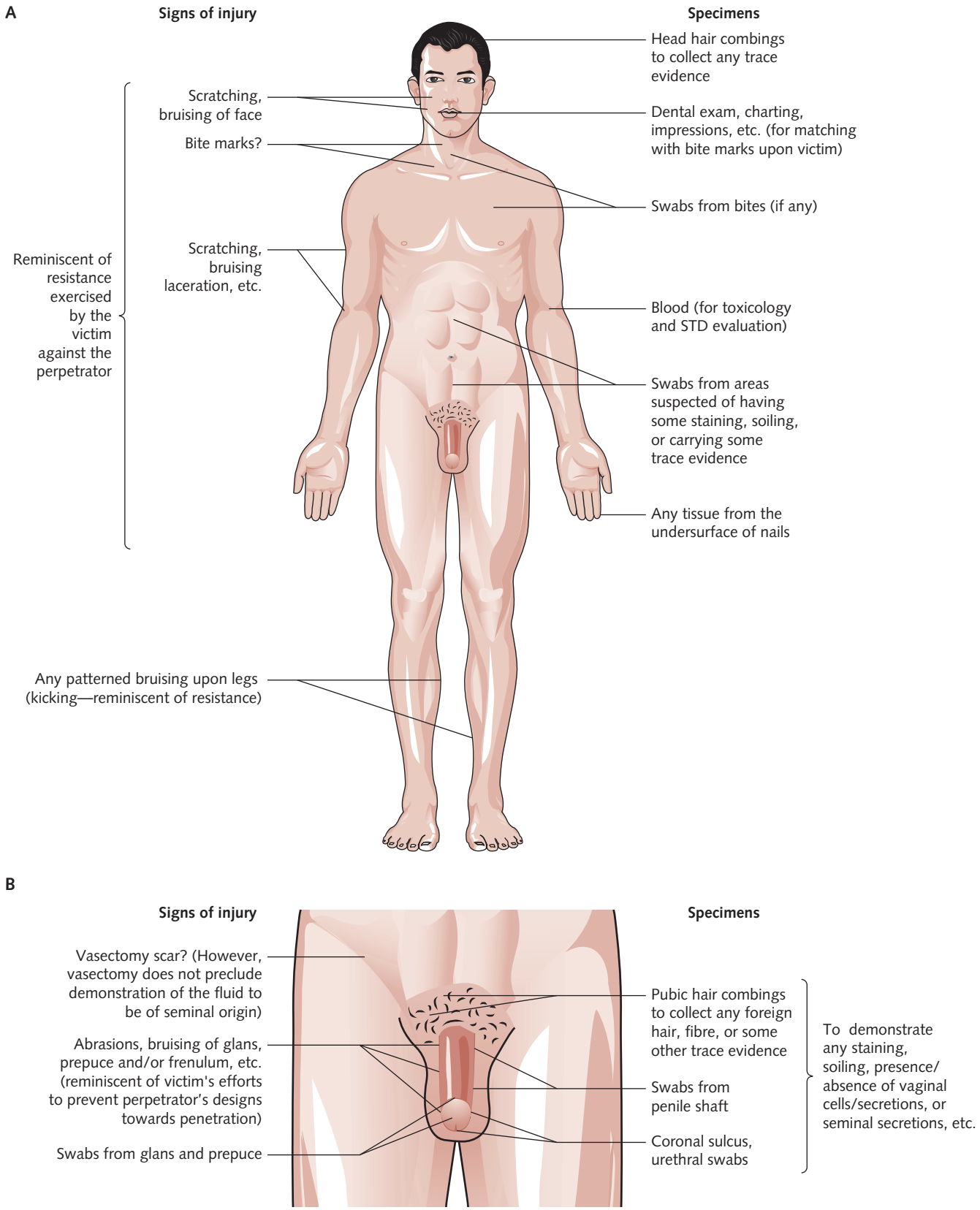


Fig. 20.5 Examination and collection of specimens in an alleged accused of rape: **(A)** general physical examination and **(B)** local examination.

Again, general observation of the individual should be maintained throughout the history taking and examination including his demeanour. Clothing must be assigned due importance as detailed earlier under the heading Examination of the Victim.

Identification of the Alleged Assailant

In the prosecution of a case of rape, the identification of the alleged assailant is a critical component. This may be gathered from the following:

- Evidence of vaginal epithelial cells upon the penis (if present).
- Fragments of skin or hair under the nails of the assailant transferred from the victim.
- Signs of injury on the general body surface and/or upon the local area, particularly scratches and bite marks on the hands, arms and face of the assailant.
- The presence of seminal fluid in vagina may be tested for its compatibility with the semen of the assailant by grouping the samples, similar phosphatase activity levels in the samples and the similar percentage of spermatozoa in the two samples may be indicative of common origin. However, a caution needs to be exercised in this regard because various chemicals may lead to erroneous results, either in a positive or negative direction. Ethanol and fluoride inhibit prostatic acid phosphatase. Many antiseptic solutions contain free phenols, which may contaminate the vagina because the victim's fear of impregnation and of contracting some disease results in rapid washing of the parts with some readily available solutions. These solutions may remove the seminal material or introduce factors that interfere with the detection of the constituents of seminal material. DNA profiling of the samples is the latest achievement towards positive identification.

Opinion

There is no single finding on examination of either the female or male that can point conclusively towards rape. It has aptly been said, **'Rape is an allegation, easily made, hard to prove and harder to disprove'.** The opinion regarding the potency of the accused should be expressed **in a negative form,** i.e. 'from the examination, there is nothing to suggest that the individual is incapable of performing sexual intercourse', if he happens to be a healthy normal individual.

INCEST

This is an act of sexual intercourse by a man with a woman within a certain degree of blood relationship. Incestual practice is prohibited in most of the countries but not in India. In India, though it is not generally an accepted practice, in some parts, it is not only allowed but also promoted by compulsive marriage between the relatives.

From the ancient times, it has been recognised that there were genetic risks in births arising from the mating of close relations and from the Biblical period, lists of 'prohibited degrees' of relationships were declared. The extent of these lists varies from country to country and even between religious and secular codes in the same country. The commonest example is of a father indulging in sexual activities including intercourse with his daughter. Instances of brothers and sisters are less common and, at times, may be due to timid personalities.

A doctor's first duty is the welfare of the patient; therefore, he must evaluate the relative needs of the members of the family. Overriding all the considerations, he should promote the interest of the abused person.

INDECENT ASSAULT

Indecent assault generally means sex-linked misbehaviour towards a person of opposite sex or same sex. The desire may simply be to get sexual gratification but not necessarily aimed at intercourse. Often the acts are intended to insult a person or to embarrass the victim. This can mean many things, from an unproven rape to merely touching the buttocks in a crowded place, fondling of breasts, thighs, perineum, putting hand into the female's skirt or blouse, etc. Only a few have medicolegal aspects when some injuries having sexual attributes occur in the form of bite marks, abrasions, love bites, etc. The offence is punishable under Sections 351 and 354 of IPC.

After the August 1997 judgement of the Supreme Court in Vishakha case, guidelines for the prevention and redressal of sexual harassment of women at workplace were issued, which came to be known as Vishakha guidelines. In that light, a Bill has recently been introduced in the Parliament entitled, 'Protection of Women Against Sexual Harassment at Workplace Bill 2010'. According to this Bill, **sexual harassment** includes such unwelcome sexually determined behaviour, whether (*i*) physical contact and/or advances, (*ii*) a demand or request for sexual favour(s), (*iii*) sexually coloured remarks and/or gestures, (*iv*) showing pornography and (*v*) any other unwelcome physical, verbal or non-verbal conduct of sexual nature. The Bill provides protection not only to women who are employed but also to any woman who enters the workplace as a client, customer, apprentice and daily wage worker or in ad hoc capacity. Students, research scholars in colleges/universities and patients in hospitals have also been covered. Further, the Bill seeks to cover workplaces in the unorganised sectors too.

UNNATURAL SEXUAL OFFENCES

Section 377 IPC deals with unnatural carnal intercourse with any man, woman, or animal. The word 'carnal' is an ancient word which used to denote 'sexual' in the legal parlance. However, the word has been derived from Latin 'caro carnis' meaning 'of the body or flesh'. Therefore, this section, though

generally understood to be applicable to penetration per annum, it also covers acts like putting the organ into the mouth of a human being (coitus per os), performing intercourse in the armpits (playing the bagpipes), submammary fissures or intercrural folds, etc. The Section is wide enough to include a woman also, i.e. carnal intercourse by a woman with an animal is also covered under this Section (Section 47 of IPC defines 'animal' as denoting any living creature, other than a human being).

SODOMY

Sodomy means anal intercourse between two males (homosexual) or between a male and female (heterosexual). It used to be practised in the town of Sodom, from where it acquired its name. The Greeks of the Golden Age also practised it and therefore sometimes it is called *Greek Love*. It is also called **Buggery**.

In the United Kingdom, acts of anal intercourse are no longer criminal offences if they take place between the consenting adult males (of and above the age of 21 years) and the act is carried out in private. As per Indian Law, both the active and passive partners are guilty of the offence, even if the act has been committed with consent and the consenting party is equally liable as an abettor.

The offence mostly involves two males. Sodomy is popularly referred to as **paederasty** when the passive agent is a child, who is known as **catamite**. A paedophile is an adult who frequently engages in sexual activities with children.

A homosexual component exists in everybody, but it varies quantitatively in different individuals and also varies at different epochs of life. The condition may be due to arrested development of mind. In hostels, prisons, military barracks, etc., this may be seen commonly where the boys or prisoners may act alternatively as passive agents for sexual gratification. There is a class of people in India known as **eunuchs,** whose main means of living is passive paederasty. They are therefore known as male prostitutes. Among them, there are two groups—the *Hijrahs* and the *Zenanas*. The *hijrahs* add to their tribes by recruiting boys and castrating them. On healing, the scar invaginates and their external genitals therefore look like those of females on a cursory observation. Being castrated before puberty, they develop feminine characters owing to loss of influence of male hormones. Consequently, they possess feminine voice, feminine type of distribution of fat and hair and develop some breasts. They dress like women, wear ornaments and adopt female tastes and habits. The *zenanas* live separately and their genitals are intact.

Examination of the Passive Agent

Pre-requisites

(a) A requisition for the examination from an authorised person.
(b) Identification of the victim by the parent or guardian and noting of identification marks.
(c) Consent for the examination from the individual or parent or guardian as the case may be.
(d) Presence of a female attendant while examining the female passive agent.

History

General The general history includes the following:

- Details of past illness
- Surgical operations
- Any recent medications or alcoholic intake
- Bowel habits and any operation or instrumentation on bowel
- In case of female passive agents, details of childbirth or any instrumentation during delivery that may alter the normal anatomy of the anal verge and perineum.

Specific The specific history includes the following:

- Date, place and time of the alleged act
- Use of violence
- Use of any lubricant
- Any history of pain, bleeding from the anal canal
- Has the patient defecated since the alleged act
- Has there been any change of clothing
- Has the patient bathed or washed the anal area
- Clothing deserves a special mention. If not changed, they should be removed one by one by making the patient stand on a white sheet of paper so that any trace evidence, if available, may not be lost. Due precautions as stressed under the examination of the victim of rape should be practised in collection and packing of clothing for further laboratory examination. Particular attention should be paid to the crutch areas of under clothing for presence of seminal or blood stains or fecal/lubricant soiling.

Examination

General physical examination on the same lines as mentioned earlier.

Specific examination of the area in and around the anus and genitalia:

Firstly, **pubic hair** need attention. Any matted area, if present, should be cut as close to the skin as possible and sent for laboratory examination. Any other entangled foreign hair or trace evidence should be looked for.

Penis also invites examination because in many cases, oropenile contact is either preliminary to anal intercourse or is actually the entire extent of sexual contact. Swabs should be taken from the penile shaft and glans penis for the presence of salivary traces or any other material.

Perineum needs observation with special attention to the anal verge under good light and patient in knee-elbow position.

Before any digital examination is proceeded, swabs from the perineum, and verge and deeper regions of anus should be obtained.

Anal area: the appearance of anal verge must be noted carefully. Normally, the anal orifice is slit-like, running anteroposteriorly; surrounding skin shows marked natural folds due to the act of corrugator cutis ani muscle. In cases where anal intercourse has taken place, commonly there are changes in the normal anatomy, and the extent of such changes is dependent upon the following factors:

- Frequency of acts of anal intercourse
- Time interval between the last act of intercourse and the examination
- Age, built and the size of the orifice in a particular individual
- Degree of force applied during the act
- Size of the penile organ
- Use of lubricant

First ever intercourse tends to produce changes in the appearance of anal verge, which may vary from overt tearing of anal skin and underlying sphincter muscle or splitting of skin and production of anal fissure or to the mere abrasion/bruising of the verge. Abrasions may be seen frequently that may be superficial or deep and present on any part of the circumference of the anal verge. They can be produced by moderate frictional shearing of the penetrating penis but may also be caused by fingernails during the act of scratching due to poor hygiene of the area or rarely when the impact is upon this area. They may be extensive in cases where there is great disproportion between the anal orifice of the victim and the organ of the accused. Effective lubrication of the part will tend to reduce the production of these abrasions.

Bruising of the area, in and around the anus, may also be present in some cases.

Tearing of the sphincter is rare in case of adults, but can occur in children. Owing to the great contraction of the sphincter, the penis rarely penetrates deeper and consequently the tearing produced, if any, is usually triangular in nature having its base at the anus and sides extending vertically inwards into the rectum. Again lubrication and slow introduction of the organ may prevent these changes to appear.

Anal fissure, if present, are splits in the skin of anal verge and they may be restricted to the external skin only or may extend within the anal canal to the mucocutaneous junction. The situation of the anal fissure is dictated by the muscular support of the skin of the anal verge, and this tends to be the weakest at the posterior quadrant and consequently the fissure is commonly observed in that region. In case of women who have had children, the support is reduced anteriorly as well, and therefore anterior fissuring may also take place.

At the end comes the examination of anal canal and lower rectum with the help of proctoscope. Inner region should be looked for any injury, bleeding or seminal deposition or for deposition of any other material. Swabs may also be taken with the proctoscope in situation. If there is spasm of the sphincter, examination may be carried under anaesthesia.

Signs of Habitual Anal Intercourse

The signs usually met in a passive agent habituated to the act of sodomy may be as follows:

- Shaving of the anal hair but not necessarily the pubic hair.
- Dilated and patulous condition of the anus, as normal folds at the anal verge tend to be lost so that the anal margins appear much smoother.
- Thickening of the skin at the anal margins that may extend into the anal canal up to the mucocutaneous junction.
- Scars of the healed fissures may also be seen.
- In extreme cases of habitual intercourse, the anus may be 'deep-seated' so that the anal area looks as though it is situated in a funnel-shaped depression. But this may be absent in strong healthy individuals habituated to the act as passive agents, while it may be normal within some weak debilitated individuals or old women. The useful guide as to the patient's habituation to anal intercourse is **lateral buttock traction test**, in which the thumb is placed on the cheeks of the buttocks on either side of the anus and gentle lateral traction is applied. In patients who are not accustomed to anal penetration (penile or instrumental or any other), the traction results in reflex constriction of the anal sphincter; the patients who are used to anal penetration react to the lateral traction by relaxation of the sphincter. But history of surgery or anal instrumentation should be excluded. Some natural disease should also be taken into account, if present. Presence or absence of gonorrhoeal discharge, chancre or condylomata should also be looked for. The same principles apply here as mentioned under examination of the victim of rape.

The Opinion

It should be given on the same lines, i.e. interpretation of findings only. All the findings must be weighed together at the end, viz.

- Presence of semen/seminal stains in and/or around the anus.
- Soiling of the anal region with semen, faecal matter or any lubricant.
- Smearing of clothes with semen, blood, lubricants, mud or any other material.
- Injuries in and around the anus.
- Foreign hair or any other trace evidence in or around the anus.
- Changes in the general anatomy of the anal verge and the surrounding area.

However, it may be kept in mind that no sign may be evident when the active agent has used adequate lubricant and has introduced the organ slowly into the anus without using undue

force and the passive agent is a consenting party. Moreover, the acute signs of first ever penetration are usually short-lived and findings may get obliterated within about 24–48 hours. Therefore, the time interval between the alleged offence and the examination is a vital factor in appreciation of the findings.

BUCCAL COITUS (ORAL COITUS OR SIN OF GOMORRAH)

This offence is mentioned in the Bible. It used to be practised in the town of Gomorrah and hence the name 'sin of Gomorrah' is attributed to this practice. Oral coitus may be practised by both sexes. When the male organ is sucked by a female or another male, it is termed **fellatio**. When female sex organs including clitoris are sucked by a male or another female, it is called **cunnilingus**. Thus, it may be a heterosexual or homosexual practice. Either way it is punishable by law under Section 377 IPC as mentioned earlier.

There may not be any sign in the face or mouth of the passive agent due to washing but signs of resistance in the form of minor injuries may be present on the face and elsewhere on the body. The penis of the accused may show abrasions caused by the teeth and may have stains of saliva. In the practice of fellatio, some danger to the victim to be choked is present.

HOMOSEXUALITY

The term 'homosexuality' denotes erotic thoughts and feelings towards a person of the same sex as well as any associated sexual behaviour. The expression of homosexual behaviour varies with age and circumstances. Many people prefer to identify sexual orientation by using terms like **lesbians and gay men**. The terms 'lesbianism' and 'sapphism' have been derived from the island of Lesbos in the Aegean sea, where the practice of female homosexuality was being carried out by an exclusively female population ruled by Queen Sappho.

Psychosexual development of sex-type behaviours spans a broad mix of the elements that comprise '*masculinity*' and '*femininity*'. There is a continuum, with exclusively heterosexual people at one end and homosexual people at the other; between these extremes are the people who engage in varying degrees of both homosexual and heterosexual behaviour and relationships.

In case of men, homosexual physical intercourse includes oral genital contact, mutual masturbation, and less often anal intercourse. And **in case of women,** physical intercourse usually includes caressing, breast stimulation, mutual masturbation and oral-genital contact (cunnilingus). Some women may practice full body contact with genital friction or pressure (tribadism), or use a vibrator or artificial phalluses (dildoes). Active and passive partners are usually exchanged, although one partner may habitually play as active and the other as passive partner. A preferentially active lesbian (who is most often a transvestite or transsexual) is known as a **butch** or **dyke,** while the usual passive agent is called a **femme**. In India, the practice is not unlawful and is not specifically covered under Section 377 IPC.

BESTIALITY

In bestiality, a lower animal is chosen for sexual intercourse, which may be practised either through the anus or vagina of the animal. Though it may be seen in both sexes, it is more common in males. She-goat or a hen may be chosen by a male and a pet dog by a female as these are easily available, relatively docile and convenient in size.

While bestiality may be due to sex starvation, the person is likely to be suffering from some mental aberration. In addition, the superstitious belief that venereal diseases are cured by sexual intercourse with a lower animal may lead to bestiality. The accused is generally a young person employed to look after the animals. While being alone with the animals in the fields, he is tempted to indulge in such a practice.

A male accused, on examination, may show his penis stained with mixture of his semen and the animal dung. Animal hair may be seen sticking to the penis or the surrounding area. In some cases, due to being kicked by the animal, abrasions, bruises and/or even lacerations may be present.

On examination of the animal, human semen may be seen in the vagina or the anus of the animal (or the cloaca in case of a hen). Human pubic hair may be present near the peri-anal area.

Medicolegal Aspects of Semen

Semen is a human body fluid, stains of which are often involved in sexual offences like rape, sodomy, bestiality, attempted rape and sexual murder. Potency of the fluid carries value in civil cases like disputed paternity or nullity when the defence is impotence. Seminal stains are next in importance to blood stains, which are often found simultaneously on the personal garments/fabrics, etc., from the point of view of involvement and probative value.

Semen is a viscid, mucilagenous fluid with greyish-yellow colour and characteristic odour. It is a suspension of **spermatozoa** in a complex medium called the **seminal plasma**. The average volume of a single ejaculate ranges between 1.5 and 5.0 ml. There are usually between 60 and 150 million sperms per millilitre of ejaculate, of which about 80% are motile at the time of ejaculation. The large number of sperms per ejaculate is necessary to overcome anatomical and chemical difficulties encountered by the sperm in its journey to the ovum in the oviduct. The sperms travel through chemically unfavourable vagina (pH-acidic) and pass through the cervix, uterus and oviduct in order to reach the ovum and fertilise it. Survival of the sperm depends upon sperm concentration, motility and rate of passage. The degree of motility may further be reported in terms of hypokinetic, normokinetic and hyperkinetic. The usual rate of speed is 1–4 mm per minute (Table 20.2).

Table 20.2 Composition of Human Semen

Colour: White, opalescent Specific gravity: 1.028 pH: 7.35–7.50	
Sperms: Average count about 100 million/ml	**From testes** (contributes about 10% of total volume)
Fructose (1.5–6.5 mg/ml) Phosphorylcholine Ergothioneine Ascorbic acid Flavins Prostaglandins	**From seminal vesicles** (contribute about 60% of total volume)
Acid phosphatase Spermine Citric acid Cholesterol Phospholipids Fibrinolysin, fibrinogenase	**From prostate** (contribute about 30% of total volume)
Zinc Phosphate Bicarbonate	**Buffers**
Hyaluronidase Neuraminidase Zona lysins	**Acrosomal enzymes**

Thus, the bulk of the semen is seminal vesicle fluid, which is the last to be ejaculated and serves to wash the sperm out of the ejaculatory duct and urethra. The average pH of the combined semen is approximately 7.5, the alkaline prostatic fluid having neutralised the mild acidity of the other portions of the semen. The prostatic fluid gives the semen a milky appearance, while fluid from the seminal vesicles and from the mucous glands gives the semen a mucoid consistency. Also, the clotting enzyme of the prostatic fluid causes the fibrinogen of the seminal vesicle fluid to form a weak coagulum that holds the semen in the deeper regions of the vagina where the uterine cervix lies. The coagulum then dissolves during the next 15 to 30 minutes because of lysis by fibrinolysin formed from the prostatic profibrinolysin. In the early minutes after ejaculation, the sperm remain relatively immobile, possibly because of the viscosity of the coagulum. However, as the coagulum dissolves, the sperm simultaneously become highly motile.

Although sperm can live for many weeks in the male genital ducts, once they are ejaculated in the semen their maximal life span is only 24 to 48 hours at body temperature. At lowered temperatures, however, semen may be stored for several weeks.

STRUCTURE OF SPERMATOZOA

The mature spermatozoon in the human measures about 60 μm in length and is divided for descriptive purposes into a **head, neck and tail** region (Fig. 20.6). The **head** has a flattened pyriform shape and measures 8–10 μm approximately. The nucleus, which occupies major portion of the sperm head, has a large amount of chromatin comprising deoxy-ribonucleoproteins. Anteriorly, covering like a cap is the acrosome, which is a highly modified lysosome, derived from the Golgi apparatus during spermatogenesis. It consists of a membrane-bound sac of hydrolytic enzymes and is completely enclosed within the plasma membrane of the sperm cell. The acrosome aids the sperm in penetrating the layers around the ovum.

Acrosomal contents and their functions:

- **Hyaluronidase** is a hydrolytic enzyme.
  (i) It lyses the glycosaminoglycans in the extracellular matrix holding the cells of the corona radiata together. As the coronal cells become more loosely associated, sperm cells can propell themselves inward towards the zona pellucida.
  (ii) Hyaluronidase may also be involved in breaking down the zona pellucida.
- **Neuraminidase,** also a hydrolytic enzyme, removes neuraminic acid (sialic acid) from glycoproteins. In experimental studies, a neuraminidase-treated zona pellucida cannot be penetrated by sperm cells. Thus, the acrosomal neuraminidase may aid in preventing more than one sperm from entering an ovum (polyspermy).
- Zona lysins are proteolytic enzymes that are capable of degrading the zona pellucida, perhaps easing the passage of sperm cells through to the ovum.

The neck is a short region containing the connecting piece composed of segmented columns and the proximal centriole.

The tail measures about 50–60 μm in length and is composed of a middle piece, a principal piece and an end piece. The middle piece is about 10–12 μm in length. The core structure is the axoneme, which is surrounded by outer dense fibres and more externally by a sheath of helically arranged mitochondria. The principal piece is about 40–50 μm in length and is slightly narrower than the middle piece. The fibrous sheath, composed of two longitudinal columns and connecting ribs, is the characteristic component of this segment. The end piece is about 5–10 μm in length and contains only the axoneme and associated cytoplasm.

COLLECTION, PRESERVATION AND DESPATCH OF SPECIMENS

Materials/objects suspected of containing semen are usually sent to the Chemical Examiner of the State Government. Clothes stained with semen should be dried well before despatch. Moisture will facilitate disintegration of spermatozoa through decomposition. If vaginal contents are to be examined, the specimen should be obtained from the posterior fornix. A small amount of normal saline may be put into the posterior fornix before it is aspirated with a pipette. If a culture is required, the specimen should be obtained with sterile instruments. Several vaginal smears should be made at the same time on clean glass slides. The smears should be immersed immediately

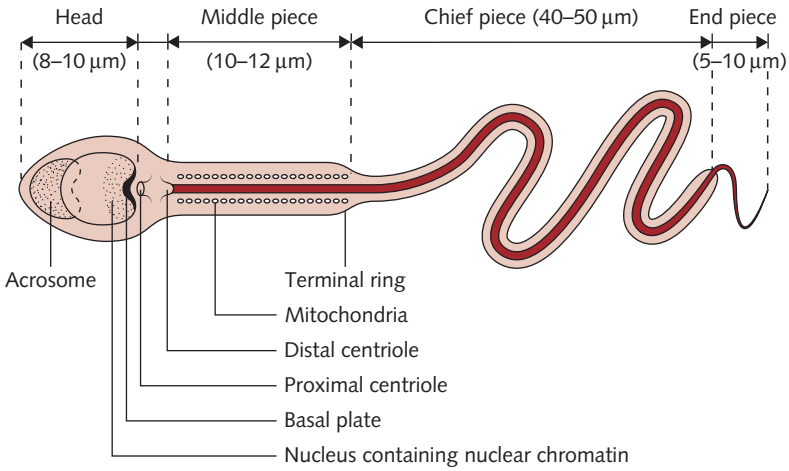


Fig. 20.6 Ultramicroscopic structure of human sperm.

in a fixative that contains equal quantities of absolute alcohol and ether. Several vaginal swabs should also be taken and placed in clean test tubes. The swabs should be kept in a refrigerator until they can be examined for acid phosphatase. Suspected stains on pubic or perineal hair should be collected by clipping the hair. Dry stains on the skin are gently wiped with wet swabs, which are then air dried. Every item should be labelled separately before dispatch; the label should mention the particulars of the case, the site from where it is collected, and the date and time of collection.

SEMINAL IDENTIFICATION

Whether the material/stain is of seminal origin may be judged through preliminary scrutiny by physical and chemical examination.

Physical Examination

The naked eye appearance of seminal stains on the fabric depends upon the background on which it is located. Stains on white fabric appear yellow with borders appearing darker than the centre. On coloured or dirty fabrics, no colour may be appreciated. On nonabsorbent surface such as skin, leather, etc., the stain presents a scaly starchy appearance. On absorbent surfaces such as cotton, silk or wool, the stain may be colourless or grey. When examined under ultraviolet light, seminal stains exhibit strong bluish-white fluorescence. Stains mixed with blood may not fluoresce. Fluorescence depends upon the choline of the semen. Substances such as food stains, vaginal secretions, urine, pus, etc. may also show fluorescence. Stains on artificial silk do not show fluorescence. The residues of modern detergents on clothes also fluoresce strongly.

Chemical Examination

The rapid tests are **Florence test** (based on the exhibition of dark brown rhombic crystals of choline iodide, resembling haemin but are larger, arranged in clusters, rosettes or crosses, etc.) and **Barberio test** (showing yellow needle-shaped crystals of spermine picrate). Both these tests are simply suggestive of presence of some vegetable or animal substance. A negative test, however, reveals that the stain is not of seminal origin.

Another chemical test helping to establish seminal origin of the fluid is the demonstration of acid phosphatase enzyme in the fluid. However, presently it is being used as only screening test because of discovery of P-30 and MHS-5 secreted by prostate and seminal vesicle, respectively (as already discussed under the heading 'Rape'). It may be worth adding here that in the medicolegal context, factors affecting stability of this enzyme in the vaginal passage need to be kept in mind while interpreting results, viz. (*i*) somato-sexual disorders (account for lower values than normal), (*ii*) chronic prostatitis (leads to low levels of the enzyme in the semen), (*iii*) amount of seminal fluid deposited into the vagina, (*iv*) victim's activities (whether she has washed, douched, or done much walking, etc.) and (*v*) vaginal environment (bacteria, hydrogen ion concentration, etc.).

CONFIRMATORY TESTS FOR SEMEN

Microscopic Examination

The morphology of intact spermatozoa of man is unique and distinguishable from that of animal spermatozoa. After the spermatozoa are damaged and broken up, this morphology is lost and not available for the detection of species origin. The microscopic detection of seminal stains based on morphology of spermatozoa thus offers an added advantage of their species origin. The extraordinary diversity of shape and structures encountered among the spermatozoa of different species prompted Wagner and Leuckart (1852) to state, "one may often safely venture to infer from the specific shape of these elements, the systemic position and the name of the animals investigated".

It serves as a confirmatory test for semen wherever the sperms are demonstrable, and obviously also valuable in determining the potency. When one unbroken sperm is found, it is a proof of seminal fluid. In relation to determination of potency, sperm count and sperm motility should be taken care of. A potent fluid should normally contain not less than 60 million sperms per millilitre but counts within a wide range are consistent with potency. Complete absence of sperms (azoospermia) must not be assumed unless confirmed by an examination of at least three ejaculates.

Difficulties may arise when disconnected heads and tails are seen. Yeast, trichomonas, monilial spores, and isolated cell nuclei, etc. may simulate spermatozoa. A definite opinion in these cases can be given only by an expert. Spermatozoa usually undergo disintegration within a few months of deposition. In exceptional cases, they may be seen even in very old stains. Gordon and Shapiro were able to identify complete spermatozoa in a seminal stain 5 years after its deposition on a clean cotton material.

Proof of Semen

Prostate specific antigen (P-30) and seminal vesicle specific antigen (MHS-5) are specific to human semen and are produced by epithelial cells of prostate and seminal vesicle, respectively. Antibodies specific to these antigens can be detected by various immunological tests like ELISA, immunodiffusion and agglutination-inhibition of human seminal plasma (HSP). These antigens have been reported to be present in the semen of aspermic persons as well.

DNA Testing

Recent developments have now added the DNA technique to the armamentarium of forensic scientists. This is a remarkable advance in the forensic field, particularly in the sexual offences where the problems posed by admixture of semen and vaginal secretions can now be solved in substantial number of cases. Seminal fluid in the vagina of the victim of murder/rape can be matched against the blood DNA pattern of a suspect—there is no need to match semen against semen, as DNA from all sources in a given person must be identical. In sexual offences associated with homicide, as much material as can be obtained from the vagina should be collected either by pipette or by multiple swabs from different areas of vagina. These swabs should be frozen at –20° C if there is likely to be some delay in transmitting these to laboratory.

Diagnosis of Pregnancy

Although the diagnosis of pregnancy is not difficult in most cases, sometimes where a positive diagnosis is of utmost importance—more specifically in the early months—difficulties may be encountered.

MEDICOLEGAL IMPORTANCE OF PREGNANCY

The medicolegal importance of pregnancy is manifold. A medical person may be requested to examine a woman to ascertain whether pregnancy exists or not under the following circumstances:

- A woman may plead pregnancy as a bar to hard labour or execution. If a woman sentenced to death is found to be pregnant, the High Court shall commute the sentence to imprisonment for life [CrPC (Amendment) Act, 2008 (w.e.f. 31.12.2009)].
- A woman may advance pregnancy as an excuse to avoid attendance in a court of law as a witness. Pregnancy per se is not a sufficient excuse for not attending the court. She will be excused only when a physician certifies that delivery is imminent or that some serious complications may ensue if she were to attend the court.
- A woman may feign pregnancy and delivery after the death of her husband and produce a fictitious heir (supposititious child) to the estate that belonged to her husband. In such cases, i.e. the person who would have naturally succeeded thereto, may apply to the court for an enquiry to be made into the alleged pregnancy and delivery.
- Occasionally, it may be alleged that a woman living apart from her husband is pregnant. The allegation may be put forward in support of a suit for divorce. Sometimes, the woman defamed may be an unmarried female or a widow who seeks to clear her name.
- The question of pregnancy may be raised in criminal abortion. According to Sections 312 and 511, to cause or attempt to cause a woman 'quick with the child' to miscarry is a graver offence than if she is not 'quick with the child'. It is important to decide whether or not she was 'quick with the child' at the time when abortion was attempted or caused.
- A woman may claim to be pregnant and file a suit in a court of law for break of promise of marriage or for seduction.
- A woman may allege that she is pregnant in order to get greater compensation from a person or persons through whose culpable neglect her husband died.
- Pregnancy may well be a motive for the suicide or murder of an unmarried woman or widow.
- The question of whether a woman has given birth to a child recently or not, may be raised in an alleged case of infanticide and concealment of birth.
- At the time of marriage, if a woman is pregnant, marriage may be declared null and void.
- When the pregnancy is followed by death of the husband, the widow may claim a greater share in the ancestral property.

- Pregnancy beyond the scope of lawful wedlock generates many issues concerning the child.
- Working pregnant women are allowed additional leave facilities.

DIAGNOSIS

In medicolegal cases, diagnosis of pregnancy must be based upon history, physical signs and laboratory investigations. One may prefer to categorise the various steps as presumptive, probable and positive instead of classifying them under three trimesters. Before examination, the doctor must obtain the **consent of the woman** in the presence of witnesses. Examination without consent will lead to civil action for damages as well as criminal action for assault. The request of the members of the family or any other interested person does not give him the authority to proceed with the examination. It will be proper for a doctor to examine an arrested person even without consent when requested by a police officer not below the rank of Sub-inspector, provided there are reasonable grounds for believing that the examination of her person will afford evidence as to the commission of the offence (Section 53 of CrPC). When a female is to be examined under this Section, it shall be done only by or under the supervision of a female registered medical practitioner. If the woman is mentally defective or a minor, consent may be obtained from the parent or guardian or next of kin.

PRESUMPTIVE SIGNS OF PREGNANCY

Amenorrhoea

Amenorrhoea may be the first warning symptom in normally menstruating women exposed to the probability of pregnancy. This is, however, not wholly reliable as amenorrhoea may result from chronic debilitating diseases, emotional stress and other factors. Moreover, cyclical bleeding can occur in the first 8–12 weeks of pregnancy either from a bicornuate uterus or uterus didelphys and even from a normal uterus prior to the fusion of the decidua vera and capsularis. Pathological lesions in the genital tract can also give rise to bleeding during pregnancy.

Morning Sickness

In the early weeks, nausea and vomiting are common. 'Morning sickness' generally starts in about 4–6 weeks of pregnancy and may continue till about the 16th week. Usually, it is present in the early hours of the morning and shows signs of abatement as the day progresses. In some cases, however, sickness may continue throughout the day. Sometimes, nausea is more persistent than vomiting. In some cases, there may not be any morning sickness.

So long as it does not affect the general health, morning sickness is an ordinary physiological phenomenon associated with pregnancy. Occasionally, it may become a pathological symptom, when the nausea and vomiting become too excessive as to prevent the possibility of any nourishment being retained or even taken by the patient. This is called hyperemesis gravidarum.

Salivation and Changes in Disposition

Salivation is an early symptom and is pronounced in certain cases. The changes in disposition may be shown by a change in the temperament, resulting in the patient becoming irritable and capricious. She may evince a desire for articles of food quite at variance with her ordinary preferences. These have been termed the longings or pica of pregnancy; they are not of diagnostic value, as they are purely subjective and may occur in various neurotic conditions as well.

Irritability of the Bladder

Frequency of micturition is sometimes complained of, and is due to the pressure exerted on the bladder by the growing uterus. As the uterus increases in size and becomes an abdominal organ, this pressure is relieved and the symptoms gradually disappear.

Changes in the Skin

Pigmentation is one of the characteristic changes that take place in pregnancy. This is more marked on the forehead and cheeks in the form of dark brown patches, more noticeable in those who are fair skinned. Pigmentation and striae may also be seen on the breasts and over the abdominal wall. A linear pigmented area stretching from the umbilicus to the symphysis pubis is of deeper colour and is known as the **linea nigra**.

The abdominal wall distends as the pregnancy advances, and grows thinner, especially around the umbilicus. The skin over the abdomen shows depressed lines, pinkish or slightly bluish in appearance. These lines are called the **striae gravidarum**. They are curved, irregular, arranged more or less concentrically, sometimes radially, around the umbilicus, gradually becoming broader and deeper near Poupart ligament. They may also be found over the thighs on the anterior aspect, sometimes on the posterior aspect as far as the knees, as well as under the breasts. These lines are caused by the rupture of the subcuticular elastic fibres, and after delivery they heal, leaving pearly white or silvery bright lines, now known as **linea albicantes**.

Changes in the Breasts

Changes in the breasts are marked, particularly in primigravidae. There is a general enlargement with prominence of the veins and increased pigmentation, forming the characteristic primary and secondary areolae. The nipples also become more

prominent, erectile and turgescent. **Montgomery's follicles** appear first on the primary areolae and later on the secondary areolae. The secondary areolae develop from the 20th week onwards, while the other changes generally take place during the first trimester—from the fourth to the 12th week of pregnancy. Presence of little fluid in the breast can usually be detected from the 12th week onwards by gently squeezing the breast in the direction of the nipple. The fluid is clear and contains some colostrum corpuscles.

In multiparae, the changes in the breasts are not of much diagnostic value because pregnancy may take place in a lactating woman, while pigmentation of the areola and the milky secretion in the breasts may persist after a previous pregnancy. While the absence of these signs does not prove the nonexistence of pregnancy, their presence cannot help one to a positive conclusion unless supplemented by other signs.

Bluish Discolouration of the Vagina

This sign is generally detected between the fourth and 8th weeks of pregnancy. The discolouration increases in intensity up to the 16th week, when it has perhaps reached its maximum. It persists throughout pregnancy. The vulva and the vaginal mucous membranes, consequent upon the congestion of the blood vessels, present a violet or light blue tint, and later a purplish or deep blue tint. This sign was first described by Jacquemier, and later emphasised by Chadwick, and is therefore known as **Jacquemier sign** or **Chadwick sign**.

Quickening

An important symptom that may be felt during the second trimester is quickening. The active foetal movements are generally felt by the mother first at the end of the 16th week; the term 'quickening' is applied to the first recognition. The movements become more vigorous and may sometimes be painful. They may cease entirely in some cases although the foetus continues to be alive. Their sudden and complete cessation, however, is suggestive of death of the foetus in utero.

Quickening is important from the medicolegal point of view in the sense that when a condemned woman pleads pregnancy as a bar to hard labour or execution, the court may postpone the execution of the sentence of death or commute it to life imprisonment. The usual certificate required from a doctor in such a case is—whether the woman is 'quick with the child' or not. Also in cases of criminal abortion upon a woman who is 'quick with the child', punishment is enhanced.

PROBABLE SIGNS OF PREGNANCY

Hegar Sign

Softening and compressibility of the isthmus or lower uterine segment constitutes Hegar sign. This is of great value and has been observed from about the 6th or 8th week to the 12th week of pregnancy. This sign is more difficult to recognise in multiparae than in primiparae.

Braxton Hicks Sign

Intermittent uterine contractions are known as Braxton Hicks sign, and it is found irrespective of whether foetus is alive or dead. It may be detected by palpation as early as in the 16th week. These contractions, as a rule, occur throughout pregnancy at fairly long intervals and last for a few seconds. They may be easily elicited by keeping the hand in full contact with the abdominal wall over the uterus, when the gradual relaxation and contraction of the uterine musculature will be felt. Similar contractions are sometimes noticed in cases of haematometra and occasionally with soft myomas.

Ballottement

Internal and external ballottements are objective signs of pregnancy, which can be elicited during the fourth or fifth month of pregnancy. Ballottement tests may be performed by external means as well as internal means. Accordingly, the tests are termed as internal ballottement test and external ballottement test. In **external ballottement test**, grip of two fingers is applied over the lower part of uterus, the woman being in semi-inclined position. As the foetus takes lowest position inside the uterus in this posture of woman, it is closer to the fingers. By exerting thrust with the help of the fingers, the foetus can be made to move up in the amniotic fluid. It may sometimes be elicited in cases of fibroids or ovarian tumours associated with ascites. It is difficult to be elicited when the abdomen wall is thick and fatty. After a while, the foetus resettles again in the lower part of the uterus. In **internal ballottement**, the test is performed by pressing two fingers on the sides of the fornix and imparting a force for the upward movement of the foetus, which also resettles at the lower part of the uterus after a while. This test gives satisfactory positive result by the fourth or fifth month of pregnancy when the quantity of amniotic fluid is comparatively more and the foetus can thus be made to move freely. After fifth/sixth month, difficulty may be faced in eliciting these tests due to proportionate decrease in the quantity of amniotic fluid in comparison with the size of the foetus. This may not be demonstrable in conditions associated with a deficiency of the liquor amnii, where the foetus is not presenting by the cephalic pole.

Uterine Changes

The uterus is perhaps the most important organ to undergo remarkable changes due to pregnancy, which are more easily appreciable in the early months as compared to later months.

Uterine Souffle

With increase in the size of uterus and the foetus inside, the circulation of blood in uterus is also increased. This increase in

circulation causes flow of more amount of blood inside the uterus through the uterine vessels; thus, when the lateral aspects of fundus is auscultated, murmur is heard, which synchronises with the mother's pulse beat.

It becomes appreciable from the end of the fourth month. This type of sound may also be heard with the help of a stethoscope, when there is increased blood supply in the uterus due to any reason like a new growth inside the uterus.

Uterine souffle must not be confused with foetal heart sound, which is more rapid in its rate and does not synchronise with mother's pulse.

Enlargement of Uterus

Under normal conditions, the uterus enlarges uniformly and its height is proportionate to the period of pregnancy.

The anteverted fundus of pregnant uterus becomes just palpable at the symphysis at about 10 weeks. By the 12th week, it is palpable above the symphysis; it is halfway between the symphysis and umbilicus at 16 weeks and at the level of the umbilicus at 20 weeks (Fig. 20.7). The fundus then rises one 4th of the way to the xiphisternum each month until the ninth, when at term it sinks to the level it occupied at the 8th month but there is a falling forward of the fundus. Determining the duration of pregnancy by this method cannot be accurate because of the inconsistency of the location of the umbilicus, thickness of the abdominal wall, the amount of liquor amnii, the size of the foetus and the possibility of multiple pregnancy. Despite these, at times the estimate is fairly accurate.

The height of the uterus is usually smaller when compared to the period of amenorrhoea in cases of ectopic gestation, retroverted gravid uterus, intrauterine death of the foetus and oligohydramnios. On the other hand, it is larger in vesicular mole, hydramnios, multiple pregnancy, and concealed accidental haemorrhage and in tumours complicating pregnancy.

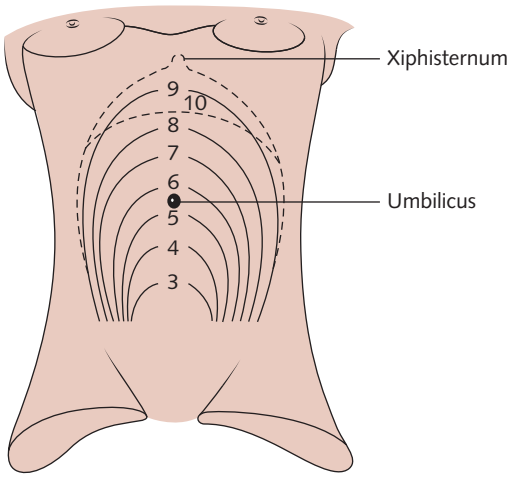


Fig. 20.7 Relative height of the fundus at the various lunar months of pregnancy.

Pregnancy Tests

The need for more rapid and accurate diagnosis of pregnancy soon after conception has become more urgent keeping in view the liberalisation of the abortion law, as pregnancy terminations carried out soon after conception carry little or no risk. None of the physical signs and symptoms at so early a period of pregnancy can definitely confirm it; hence, the need for laboratory procedures.

Laboratory Tests

- Bioassay
- Immunoassay
  - Haemagglutination inhibition test (Pregnosticon) or flocculation inhibition of hCG-coated particles (Gravindex test)
  - Radioimmunoassay (RIA)
- ELISA test

Bioassay The bioassay techniques that have been employed in the past to detect hCG in the urine of pregnant women are not routinely employed now.

Immunoassay Human chorionic gonadotrophin (hCG) has both (alpha and beta) subunits. Antigen–antibody reactions are utilised to detect its presence in the serum and urine. The tests using hCG antibodies for alpha subunits are not specific. Cross-reaction with luteinising hormone occurs as the alpha subunit is identical for both. However, antibodies raised against the beta subunits of hCG are highly specific and do not cross react with luteinising hormone. RBC or latex particles coated with hCG are used as the indicator to detect antigen–antibody reaction. Levels of 1.5–3 IU/ml of hCG can be detected.

- The principle of haemagglutination inhibition of erythrocytes or prevention of flocculation of hCG-coated latex particles is employed. **There are two basic categories of immunoassays**—the tube test and the slide test.
  - *Tube test:* For carrying out the test, a small quantity of urine (5–8 ml) is filtered. About 0.1 ml of the urine is pipetted into the ampoule provided with the Pregnosticon kit, and 0.4 ml of distilled water is added. The ampoule is shaken for 1 minute and placed in the test rack. The test rack holding the ampoules is left to stand for 2 hours without disturbing it in any way. Then the result of the test can be read off.
  - *Slide test:* Gravindex and Pregnosticon slide tests are the ones in vogue. They substitute latex particles sensitised with hCG for the red blood cells used in most of the tube tests. A drop of the woman's urine is mixed with antibody on a slide, and latex particles are added. Results are read in 1–3 minutes. Agglutination indicates absence of hCG required to neutralise the antibody, suggestive of nonpregnancy. Lack of agglutination indicates that

the level of urinary hCG is high enough, on which one may assume pregnancy.

The tube tests are more sensitive than the slide tests, as the tube tests are capable of diagnosing pregnancy as early as in 4–7 days following a missed period. They are, however, more reliable beginning the 2nd week after a missed period.

Because of the limited sensitivity in detecting early pregnancy, immunoassays are associated with a high incidence of false negatives. It is always advisable, under the circumstances of a negative test, to repeat it a week later.

- In radioimmunoassays, iodo-hCG (I^{125}) is used as the radioligand for antibodies raised against hCG. The RIA is dependent on the displacement of the radioligand by non-radiolabelled hCG in the biological fluid. The 'free' and 'bound' iodo-hCG are separated. Radioactivity of the bound form is measured, and from a standard chart, the hCG value is obtained.

ELISA Test The enzyme-linked immunosorbent assay (ELISA) is based on a principle similar to that of radioimmunoassay, i.e. competitive inhibition of a labelled hormone to its antibody by an unlabelled hormone. The label in RIA is radioactive, while that in ELISA is a plastic or certain inert particle or enzyme-end point. Clear blue colour means positive, colourless means negative.

POSITIVE SIGNS OF PREGNANCY

Palpation of the Foetal Parts

About the middle of pregnancy, the foetus is generally increased to a size when it can be recognised by abdominal palpation. As pregnancy progresses, this sign is of great value, not only in detecting pregnancy but also in ascertaining the various positions of the foetus in utero.

Auscultatory Signs

Auscultation over the abdomen during pregnancy is useful to elicit various sounds, some of which are of great importance in the positive diagnosis of pregnancy.

The foetal heart sounds can be heard from about the 17th week to 20th week of pregnancy using Pinard's fetoscope. This is the only sign of pregnancy that by itself and in the absence of all others is perfectly reliable for the diagnosis of pregnancy. The point of greatest intensity of the foetal heart sounds will vary with the position of the child in utero. Ordinarily, the foetal heart beats 120–160 times a minute.

It is possible to hear foetal heart sounds using the ultrasound Doppler technique. The ultrasound wave reflected by the moving blood flow undergoes a shift in pregnancy, the echo of which is detected by the receiving crystal, adjacent to the transmitting crystal. This method to detect foetal heart sounds becomes useful by the 10th week.

Echocardiography can be used to detect the foetal heart movement as early as in 48 days after the last menstrual period.

Radiological Diagnosis

The foetal skeleton can be made out in a good radiograph as early as in 16–18 weeks and, when seen, is conclusive evidence of pregnancy. Radiography is not of use prior to 16 weeks. When the abdominal wall is thick or when there is hydramnios or multiple pregnancy, the foetal skeleton may not be seen clearly. Realisation of the hazards of radiation in pregnancy has, however, made the obstetrician restrict its use considerably, especially in early pregnancy.

Ultrasonography

Ultrasonography by the pulse-echo sonar method is now being widely applied for diagnostic purposes in obstetrics. The gestational sac is detected as a well-defined white ring by the 6th week. Foetal echo within the sac appears by the commencement of the seventh week. Foetal heart reaction can be detected by the end of the 7th week. In 8–14 weeks, the crown rump length of the embryo allows accurate estimation of gestational age. Sonography allows early identification of dead embryo when the gestation sac appears ill-defined with absent foetal echo. The earliest identification of multiple pregnancy is possible by this technique. Also, successful recognition of a vesicular mole, the placental site, foetal anomalies and, subsequently, hydramnios and the assessment of foetal growth are possible with this method without submitting the patient to the risks of radiation.

MEDICOLEGAL IMPLICATIONS OF DURATION OF PREGNANCY

It is not possible to fix the actual date of conception even when the exact date of fruitful coitus is known, because the union of spermatozoon and ovum occurs at a variable time after coitus. In some it may occur within a few hours, while in others after a few days. The spermatozoon may retain its motility in the vagina up to a couple of hours, though it can be present as long as 2–3 days after intercourse. The spermatozoa may retain their motility in the cervical canal and uterus for 5–7 days, but the power to fertilise is usually retained for not more than 48 hours. The ovum can survive in a fertilisable form only for about 24 hours and probably only for 8–12 hours after it leaves the ovary. The time taken by spermatozoa to travel from the vagina to the tubes is 6–24 hours, but may be as short as 1–2 hour. Ovulation usually takes place 2 weeks before the onset of the next menstrual cycle. However, pregnancy may occur on any day of the cycle and even during menstruation. Therefore, it is impossible to fix the time of conception accurately. At most, the examiner can calculate 280 days from the first day of the last menstrual period and the date so obtained will be somewhere in the middle of the last 2 weeks of pregnancy. The time of fixation of pregnancy is further complicated by

the fact that the pregnancy may occur when menstrual flow is suppressed because of other causes. On the other hand, menses have been known to continue in spite of pregnancy.

Generally speaking, the duration of pregnancy is 280 days in a woman with a 28-day menstrual cycle, but it may last only for 240 days or extend to 300 days or more depending on the duration of the menstrual cycle. In India, England, USA and elsewhere, the law does not lay down any limit for the duration of pregnancy. In the case of Gaskill vs. Gaskill, the Lord Chancellor accepted 331 days as the period of gestation. In another case, Lord Simonds of the House of Lords did not accept 360 days as the period of gestation and came to the conclusion that the woman had committed adultery and the child was not that of the husband. It is clear therefore that each case will be dealt with on its own merit.

Children born at or after 210 days of intrauterine life are viable, but those born after 180 days may also be viable and capable of separate existence. The size of the child assumes importance while considering protracted or shortened pregnancies. As a rule, the longer the period of gestation, the larger the infant. It is by no means unusual to find relatively larger infants in short period of gestation as in diabetic mothers and comparatively smaller infants in protracted pregnancy when there is, for example, multiple pregnancy and malnutrition in the mother.

DIFFERENTIAL DIAGNOSIS OF PREGNANCY

Pseudocyesis (False or Spurious Pregnancy)

This condition may occur in women who have an intense desire to become pregnant. Most frequently, it is observed in a woman who is approaching the menopause, when her menstrual flow has become scanty or has ceased for a time. A deposit of fat takes place in the anterior abdominal wall and omentum, and the intestines become distended with flatus. In such cases several of the doubtful signs and symptoms of pregnancy may be present, e.g. menstruation may cease, the mammary sign of gestation may appear and the abdomen may become progressively prominent. The patient may imagine that she feels foetal movements; striae may appear both on the abdomen and breasts. The diagnosis of this condition is not difficult, but the physician should be on guard in assessing any statements the patient may offer in regard to her condition. Ultrasound is useful in such cases; needless to say, the biological test will invariably be negative.

Superfoetation and Superfecundation

Superfoetation means the fertilisation of an ovum from a subsequent ovulation in a woman who is already pregnant, and the consequence is the birth of two children at the same time, one of whom may be mature and the other immature. They may however be born at full term, one few weeks ahead of the other. The condition, though not impossible, is difficult to prove. Pregnancy does not close the cervical or tubal orifices immediately. The ovulation usually stops with the onset of pregnancy, but should it occur after the onset of pregnancy as ovulation may take place particularly during first trimester of pregnancy until the decidua vera comes into apposition with decidua reflexa and the decidual cavity gets obliterated, fertilisation of the newly released ovum may take place following coitus.

Superfecundation means multiple pregnancy resulting from the fertilisation of two or more ova liberated in the same menstrual cycle by spermatozoa introduced during successive acts of coitus. The condition is possible but difficult to prove since both fertilised ova develop as twins and go to full term at about the same period. If the mother has had sexual relation with two men of different race and two children are born at term, having different racial characters, it is substantial indication of superfecundation.

SURROGATE MOTHERHOOD

A surrogate mother is one who is hired to bear a child whom she turns over at birth to her employer. The surrogate mother, therefore, has no genetic contribution to make. In spite of the fact that the surrogate mother makes the much larger contribution towards the birth of the baby, the baby is considered illegitimate, if the mother is not the legal wife of the man. Some agencies advocate that the surrogate must be married and be a mother of at least one healthy child who should be medically as well as psychologically fit. She should not indulge in cigarettes, alcohol or any other drug during pregnancy and must agree to give up her rights after the baby is born. Her husband must also pass tests. The agency arranges the contract, life insurance for the surrogate's family (if she happens to die during pregnancy or childbirth) and life insurance or a 'will' for the child, should the (contracting) couple die before the child is born.

Surrogate may be compelled to terminate pregnancy if so wished by the contracting couple. There have been instances where the contracting individual has specified the sex of the baby as well and refused to take the baby, if it was not normal and filed a suit against the surrogate alleging that she had broken the contract. A New Jersey court opined that the time difference between producing semen and producing a child is enough to destroy the analogy. What surrogates sell is not their labour but their body itself, and every act that they perform may be under the scrutiny of the contracting couple.

The surrogacy may be employed in a variety of events. For instance:

- A couple (Mr. and Mrs. A) cannot have a child because Mrs. A is unable to receive the fertilised ovum into her womb and nourish it there due to a malformation of the womb or as a consequence of a disease. They, therefore, resort to extraction of Mrs. A's ovum to be fertilised by Mr. A's sperm in the laboratory. She has, therefore, played an important role in the production of this baby. The resultant embryo is implanted into the womb of another woman (Ms. B) who carried the foetus to full term on the understanding that on

birth, she will hand over the baby to Mr. and Mrs. A; Ms. B is surrogate mother.

- Another form of surrogacy leads to even greater problems. Mrs. A does not have viable eggs in her ovaries. Ms. B is therefore artificially inseminated, using Mr. A's sperms. In this case, Mrs. A has played virtually no role and Ms. B is the genetic mother as well as the surrogate mother (by agreement).

Surrogacy has become controversial from the time it involved money. It involves lawyers, contracts and highly paid go-betweens and anonymous payers too. That is why surrogacy is sometimes called **'baby selling'** and surrogates as **'whores'**. It may be viewed as a mode of exploiting women for the benefit of men who ensure that the baby has their genes. The rights of the contracting father are considered as paramount. The contracting couple adopts the baby soon after its birth so that they become legal parents of the child. Unlike adoption, a contract is signed before the baby is conceived.

TRAUMA AND PREGNANCY

Pregnancy is accompanied by unique physiologic changes geared towards accommodating and delivering the growing foetus. In majority of cases, the body is able to adapt to these changes. However, severe life-threatening complications may arise at occasions, leading to death of the mother (maternal mortality is defined as the death of a woman during pregnancy or up to 5 months after delivery). Two of the most common complications presenting as sudden and unexpected maternal death are embolic in nature—(*i*) pulmonary artery thromboembolism and (*ii*) amniotic fluid embolism.

The pregnant uterus by acting as a hydraulic shock absorber for forces directed at the anterior abdominal wall and dissipating them in its elasticity provides protection for the remainder of the abdominal organs. Furthermore, at least until 12th to 13th gestational week or so, the foetus is situated low within the mother's bony pelvis, providing an added element of protection from direct impact injury. **Traumatic uterine rupture needs be differentiated from nontraumatic rupture**. In the former, uterus is often ruptured in the fundic region. It is theorised that during blunt force impact, amniotic fluid will distribute pressure relatively equally in all directions and the rupture, if it occurs, will be at the weakest point, which is most commonly at the fundus. In the latter, rupture preferentially occurs in the regions of already weak uterine wall (such as presence of scar). Risk factors for nontraumatic/therapy-related uterine rupture may include prior caesarean section delivery, increased gestational age, and the use of uterotonic drugs such as oxytocin and prostaglandin and obstetric interventions like forceps delivery or breech extractions, prolonged labor with cephalopelvic disproportion, etc. **The placenta**, in contrast to the uterine wall, is nonelastic and cannot contract or expand to the same extent as the uterine wall to which it is attached. Placental abruption resulting from maternal trauma usually manifests within minutes to a few hours of the injury. Where the abruption is delayed, one needs to consider nontraumatic aetiologies such as maternal hypertension, cigarette smoking, cocaine abuse and prior abruption or still birth, etc. The criteria suggestive of the probable causative relationship between a traumatic event and a subsequent abortion have been reported as including (*i*) the traumatic event was followed by a process that led ultimately to abortion; (*ii*) the foetus and placenta were studied pathologically and found to have been normal; (*iii*) the appearance of the foetus and placenta were compatible with the stage of gestation at which the traumatic event occurred; and (*iv*) absence of factors that are known to cause abortion, such as uterine abnormalities, chronic infection in the mother and a history of exposure to abortifacients or a physical attempt to induce abortion, etc.

Child Abuse

United Nations Convention on the Rights of the Child sets out basic rights and standards for judging the welfare of children, including the maltreatment. It encompasses both the maltreatment of children within family settings and that occurring through group processes and social forces. Countries vary in their approach to the problem of child maltreatment. In the United States, if any professional entertains any suspicion as to the maltreatment of the child, he is required by law to report the things to the local child welfare agency (mandatory reporting). In countries like Belgium and Holland, maltreatment cases are dealt with confidentially through health and social workers. The United Kingdom lies between these extremes. In India, any doctor who has reason to suspect about the maltreatment of the child is required to report the matter to the police. The Juvenile Justice (Care and Protection of Children) Act, 2000 provides for taking special measures towards the care and protection of children.

HISTORICAL BACKGROUND

In the Eastern culture, cases of child abuse are rare because the children are considered as the gifts of God. However, instances of ill-treatment of young children who work as domestic servants are not uncommon. Though assault and murder of children is well-known in every country, the battered child syndrome is different from the usual homicide. The syndrome was first mentioned in 1946 when an American Radiologist, Caffey, wrote a paper in a radiological journal drawing attention to the peculiar association of subdural haemorrhages with fractured long bones. Initially, it was thought to be related to increased fragility of bones. About a decade later, he wrote a second paper in which he proposed that parental abuse might be the cause of the lesion. From that time onwards,

an avalanche of articles led to the present awareness of the problem. The problem has variously been known as child abuse syndrome/battered baby syndrome/shaken baby syndrome/nonaccidental injury in childhood, etc. The children afflicted are usually young, usually less than 4 or 5 years of age. On the whole, child abuse is a matter of Clinical Forensic Medicine as abusers seldom intend to kill their victims. Deaths are, in a way, accidental, but may sometimes be processed under charges of 'culpable homicide not amounting to murder' or 'death due to rash or negligent act' depending upon the circumstances.

POINTS FOR SUSPICION

The patterns of injury are fairly constant and mainly comprise bruises and fractures. The classic feature is repetition of injuries. Major suspecting points may include:

- delayed reporting of injuries,
- injuries not corresponding to those that would be anticipated from the explanation given,
- injuries may be of recent origin and/or different ages, and
- sudden death in case of fatality.

TYPES OF ABUSE

Many types of abuse can be encountered and there may be overlapping to varying extent. **Physical abuse** has been explained as physical assault to a child by any person having custody, care or charge of that child. Methods may include hitting, throwing, inducing burns or scalds, biting, poisoning, suffocating and drowning [Meadow, R (1997). *ABC of Child Abuse* (3rd Ed.)]. Some rare conditions may also be pointed out here:

- **Munchausen syndrome by proxy**—when a parent or carer feigns an impression or produces a state of ill health in a child, it is labelled as factitious illness by proxy or Munchausen syndrome by proxy. Common features of presentation include factitious epilepsy, nonaccidental poisoning or multisystem disorders. Fabrication may result in harm to the child or have an impact on child's physical and emotional development.
- **Gentle homicide**—this term may be used when the child is killed through asphyxia, specifically 'smothering', which may be carried out with a pillow or bed clothes. Occasionally, the perpetrator may press the face of the child against his chest, or hands may be used to clamp the nose and mouth of the child. Diagnosis can be approached by taking into account the circumstances and detailed autopsy findings. **Sexual abuse** has been explained as sexual activities that involve a child and an adult, or a significantly older child [Finkelhor D. (1994) Current information on the scope and nature of child sexual abuse]. It may be in the form of contact sexual activities or noncontact sexual activities. **The former** may include penile, digital or object penetration and nonpenetrative acts like touching or kissing of sexual parts of the body of the child or making the child touch sexual parts of the abuser's body. **The latter** may include exhibitionism or encouraging the children to have sex together. **Child neglect** refers to the under-provision of the child's basic needs, both physical and psychological. It may occur through parents or through institutions, i.e. in orphanages, nurseries, educational establishments, etc. Various types of neglect may be present:
  - **Physical neglect**—principally involving lack of provision of the child's basic physical requirements like food, shelter, clothing, etc., and also includes failure to protect the child from physical harm or danger.
  - **Emotional neglect**—involving inattention to the child's emotional needs, i.e. failure to give due love and affection.
  - **Medical care neglect**—involving failure to provide the necessary medical/surgical treatment (including immunisations).
  - **Educational neglect**—comprising inattention towards the education of the child.

RANGE OF INJURIES

The most common mode of death is the head injury. Next in frequency is the injury of some abdominal organ/viscus, and the remaining account for some small percentage. Many of the injuries might not have themselves been fatal, but recognition of their mode of production will go a long way in distinguishing an accident from deliberate maltreatment. In this context, the words of forensic pathologists, Johnson, Cameron and Camps act as guiding force—"The skin and bones tell a story which the child is either too young or too frightened to tell". The following types of injuries may help in strengthening the diagnosis of abuse/maltreatment (Fig. 20.8).

Bruising (of recent origin and/or of different ages showing variance with the history) may be encountered as given below:

- Around the limbs, especially the wrist and forearms, upper arms, thighs and ankles, etc., because these places form convenient sites for the grip.
- On the buttocks due to hand smacks or beating with a strap or some similar object. Presence of bruising on the inner side of thighs may indicate possible sexual interference.
- Face, cheeks and mouth often may be bruised due to slapping. Forehead and ears are the other sites in this region.
- Scalp bruising is better appreciated through palpation and is often a part of the deeper head injury. More often, it will be localised and inconsistent with a simple fall.
- Chest, abdomen and neck may show bruising resulting from finger pressure (usually in the form of small discoid lesion, once called as 'six penny bruises' from the size of the coinage in Great Britain).

Skeletal damage (of recent origin and/or different ages demonstrating variance with the history) is often encountered.

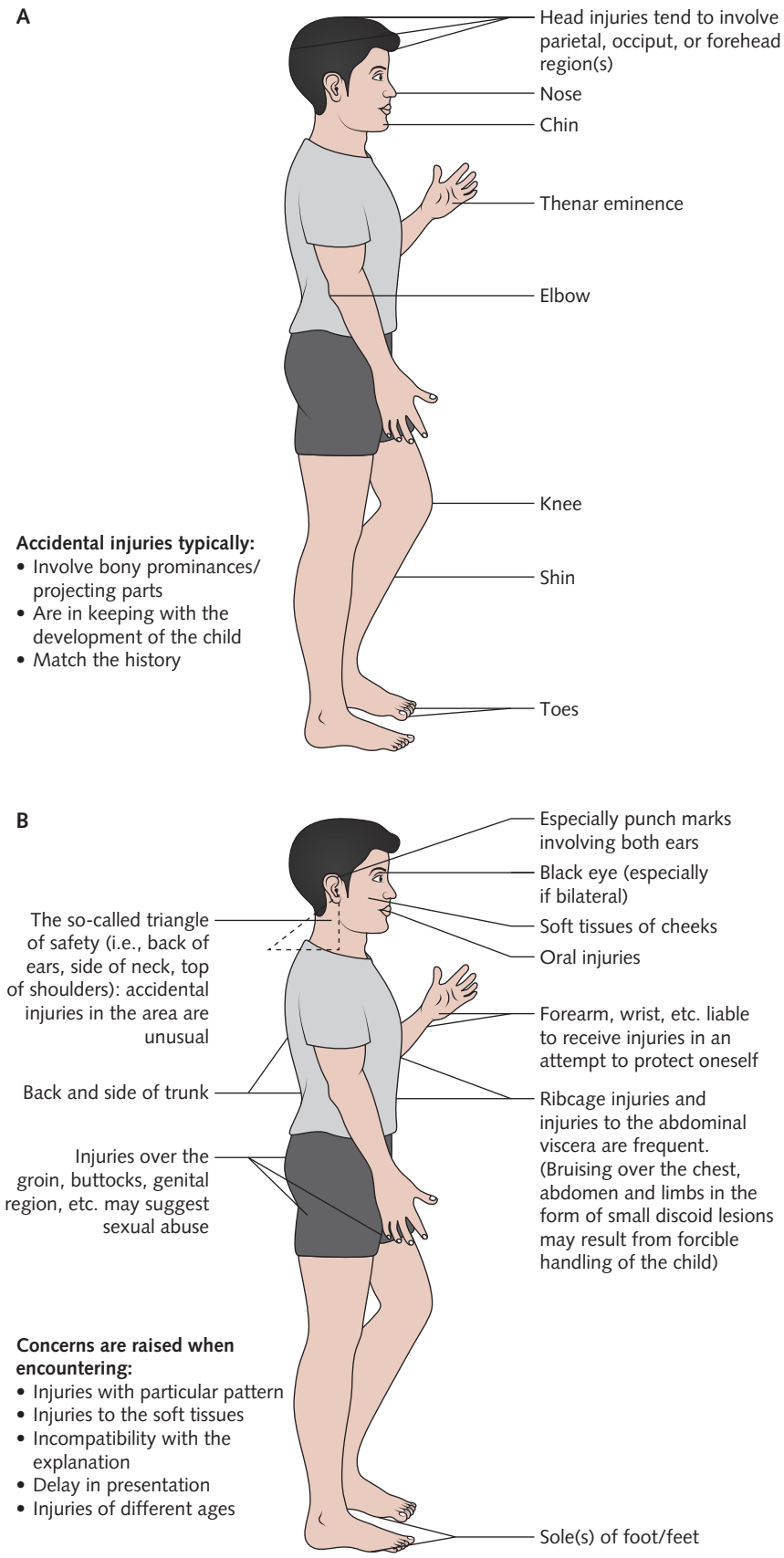


Fig. 20.8 **(A)** Typical sites for accidental injuries. **(B)** Typical sites for nonaccidental injuries.

Diagnosis largely depends upon radiology. Common types of fractures encountered in such situations are as under:

- **Skull fractures,** with or without intracranial haemorrhage, usually subdural, have been reported. At autopsy, any haemorrhage needs detailed description relating to site, amount, colour and adhesiveness. Careful differentiation between coup and contrecoup lesions will help to determine if the injury resulted from a moving object striking a fixed head or a moving head striking a fixed object. Harwood-Nash et al. found that out of 4465 childhood head injuries, 1187 had skull fractures and subdural haemorrhage was twice in incidence in the nonfracture cases. The commonest site being the occipitoparietal area. The differentiation that whether the fracture has occurred from accidental fall or from deliberate impact is warranted. Notwithstanding the flexibility, it has been reported that the infant skull gets fractured with application of much less mechanical force than would be needed to fracture a mature skull. However, brain injury is not a necessary accompaniment of the fracture of skull and it is impossible to forecast the consequences following any fall, though of minor magnitude.

  The pattern of fracturing is somewhat peculiar because of the presence of open sutures and fontanelles in the children. Fracture lines tend to end at sutures. However, if the fracture crosses sutural line, there is usually a lateral displacement so that the two limbs of the fracture are not in line, the so-called 'side-stepping'. (Sutural 'diastasis' [separation] may occur with or without fractures, the loosely knit skull plates being easily displaced by distortion of the calvarium.) Another common fracture that may be resulted from a blow or a fall on the side or top of the head is in the form of a horizontally placed fissured fracture running backwards from the frontoparietal suture, often turning down towards the base of the skull. In the event of an impact on the vertex of the skull, such fractures may occur bilaterally due to cracks occurring along the lines of the maximum stress.
- **Fractures of the extremities** are often caused by indirect force, i.e. the bone damage being produced by stress from abnormal angulation, torsion or traction, rather than from a direct impact upon the bone. Swinging the child by the writs or ankles, dragging them by arm or violent shaking using limbs as the 'grasping/griping sites' are the usual mechanisms. Swinging, wrenching or twisting can lead to avulsion of metaphysis. Chipping of the edges of the metaphyses or epiphyses may also occur. Periosteum being loosely attached to the bone can get easily lifted during shearing or traction effects.
- **Chest cage injures**, in general, are not usually of accidental origin in children. Involvement of several consecutive ribs on one or both sides is often encountered in child abuse. Damage may be fresh or old demonstrating different radiological appearances. (Nobbing fractures is the term used to denote fractures occurring due to the assailant/abuser holding the child with hands and shaking it violently or squeezing from side to side, thus fracturing ribs on both sides close to the spine, giving a nobbing appearance. Such fractures may be produced due to rib being levered against the transverse process to the extent that breakage occurs. They are much more appreciable after a week or two, when callus gets formed.) Rib fractures in the axillary line may be the result of anteroposterior pressure, rather than side-to-side squeezing. Fractures on other sites are usually the result of direct impact such as fist blow or kick.

Damage to the **internal organs** is almost always confined to the abdominal viscera. Forcible impact on the lower chest or the abdominal wall is responsible for such injuries. An excuse that the child fell or tripped upon some protruding obstruction might be forwarded by the parents. It is a matter of fact and interpretation as to whether the injuries are compatible with the explanation given or not. The central tissues/organs are the main sufferers. Liver is the frequent sufferer, the most common lesion being a laceration, which may be superficial or deep seated involving one or both lobes depending upon the manner of impact. Other common sufferers are (*i*) second part of the duodenum that crosses mid-line and is liable to be 'sandwiched' between the compressed anterior abdominal wall and the promontory of the lumbar spine and (*ii*) jejunum that may or may not be accompanied by laceration of the mesentery. **Simpson records a case** of a child wherein the external evidence of injury was confined only to some trivial bruises of the face. However, autopsy revealed 17 fractured ribs and liver plus spleen floated out through the incision made down the trunk. The mother, at first denied such violence, but later admitted losing her temper and sweeping the child by the legs against the bed rail.

Thermal injuries may sometimes be encountered. These may be scalds or dry burns. Scalds are usually produced by dipping the child in hot fluid. The term 'punished child' is sometimes used in this context when the child is dipped in hot water as a punishment for having become soiled. The distribution of burns is self-explanatory. (As the child is lowered into water, he involuntarily flexes his legs. The knees are brought up against the abdomen and lower legs folding back against the thighs. The child is then lowered into water up to his waist. Because of such attitude, there is often sparing of the skin in the inguinal region and popliteal fossae.) Dry burns may be produced in a variety of ways. A particular type of burn may be a 'cigarette burn'. Such burns are most often seen over the parts not normally covered by clothing. The mark may be rounded or bizarre in appearance. Fresh cigarette burns are red/reddish. On healing, they become pinkish and later have a silvery sheen on the surface.

21
CHAPTER

Complications of Trauma: Was Wounding Responsible for Death?

After going through this chapter, the reader will be able to describe: Immediate causes of death—primary or neurogenic shock; injury to vital organ(s); haemorrhage; air embolism | Delayed causes of death—secondary shock; wound infection; pulmonary thromboembolism; fat and bone marrow embolism; crush syndrome; exacerbation of pre-existing disease | Trauma and operation/anaesthesia | Weapon and its implications

'To complicate' means to make complex/difficult/perplexing, etc. Therefore, 'complication' in the medical science implies an added disease or an accident superimposed upon another without being specifically related, yet affecting or modifying the prognosis of the original disease/condition. Where wounds have been criminally inflicted and death ensues, a doctor is expected to report contribution of wounding towards death. There may be one principal cause of death, but other circumstances/complications may have contributed to the fatal outcome. A wound is designated as **dangerous** only when the danger is imminent. Such a wound is either extensive or serious with relation to the organ or part wounded. The term should not be employed to designate a wound, which is originally simple in character but becomes dangerous from unexpected complications. A **mortal/fatal** wound is one that almost immediately after its causation or within a short time thereafter leads to death by interfering with the functions of a vital organ or with the general functions of the body. However, unexpected recoveries have been reported. Singleton described cases in which the recovery followed the rupture of a wound in the wall of the left ventricle, wounding of the pericardium (without operation), and the lodgment of a bullet in the right ventricle (the patient remaining well after a period of 2 years). Causes of death from wounds may be enumerated as under:

- **Primary/Immediate/Direct:**
  - Primary/neurogenic shock
  - Injury to vital organ(s)
  - Haemorrhage
  - Air embolism
- **Secondary/Delayed/Indirect:**
  - Secondary shock
  - Infection
  - Thrombosis and embolism
  - Fat embolism
  - Crush syndrome
  - Development of separate pathological state
  - Exacerbation of pre-existing disease
  - Operation and/or anaesthesia
  - Neglect of/by the patient/victim

Primary/Immediate/Direct Causes

PRIMARY/NEUROGENIC SHOCK (VASOVAGAL SHOCK OR REFLEX CARDIAC ARREST)

The nervous control of cardiac and vascular functions is regulated by autonomic nervous system, and involves an optimum balance of two mutually antagonistic divisions, i.e. **sympathetic and parasympathetic**. The former stimulates the cardiovascular system, whereas the latter exerts a tonic and inhibitory control over it. The functioning of the cardiovascular system may be reflexly affected through the autonomic nervous systems by factors like:

- **Emotions (psychic shock)** may lead to parasympathetic inhibitory influence, whereas fear and pain lead to sympathetic stimulation.
- **Pressure changes in the carotid sinus** can lead to corresponding changes, i.e. increased pressure in this sinus causes stimulation of parasympathetic system, and decreased pressure stimulates sympathetic system.
- **Disturbed gaseous concentration in the blood** can also have its effect, i.e. low oxygen and high carbon dioxide may

lead to sympathetic stimulation through direct action on medullary centre and through chemoreceptors situated in the aortic and carotid bodies.

- **Excitation of peripheral nerves**, i.e. stimulation of pressor and depressor afferent fibres. Freeman reviewed the physiological mechanism of this form of sudden death from minor stimuli or injury at places/areas of the body carrying wide net of 'receptor' nerve endings forming the 'afferent pathway' for the reflex action mediated through the vagus nerve. Fatal cardiac inhibition can occur after slight traumatic stimuli applied to such parts of the body, e.g. passing a catheter into the bladder or an instrument into the uterus. It has also been reported from a slight blow on the throat or testicles and similar 'receptive spots'. (Sudden blow on the carotid region of the neck, as may be practised by commandos during training tactics for hand-to-hand encounter, may precipitate a transient or even fatal vagal inhibition without any obvious mark of injury, i.e. death can occur due to functional effects of injury/injuries. The finding on the skin may be negligible in the form of some abrasion or discolouration, but these are not necessary accompaniments of such an event.)

An interesting case has been cited in context with the death by fright wherein an elderly man was the victim of robbery; during the course of which, he suddenly died. Meticulous autopsy supplemented the circumstantial findings. In a subsequent manslaughter trial in Canada, the following criteria were used to fulfill the medicolegal requirements for homicide by fright (JAJ Ferris, First Asian-Pacific Congress on Legal Medicine and Forensic Sciences, 18–22 September, 1983, Singapore):

- The cause of death must be clearly identified.
- The assault must have been of such a type that it can be reasonably related to the mechanism of death and that such a mechanism must be based on acceptable scientific concepts.
- If there was no direct physical injury to the victim, then the circumstances of assault should be of such a nature as to be commonly accepted as highly emotional.
- The deceased must have exhibited no clinical or pathological alteration in his physical state prior to the assault to indicate that death would have occurred without the influence of assault.
- Death or change in the victim's condition that directly led to death must have taken place within a reasonable and acceptable pathological or physiological response time period.

INJURY TO VITAL ORGAN(S)

Though it is usual that injuries are accompanied by considerable effusion of blood, yet there may be circumstances wherein the quantity of blood lost may not be significant and the death ensues from damage to some vital organ(s). It needs be borne in mind that even when some vital organ is injured severely, a great variation exists in the ability to perform volitional acts before death ensues. An illustrative instance of this aspect is to be seen in a case cited by a Canadian pathologist in a conference wherein it was communicated that a man whose legs and abdomen had been crushed away under a 'cow catcher' train fender was able to speak jocularly, expressing his desire (as being unable) to pass a motion some 3 hours later while he was lying (with his mangled lower half) on the floor of the operation theatre before development of decompensated shock and death.

HAEMORRHAGE

Loss of blood lowers the blood pressure and may produce fatal shock. It may also menace life by disturbing the functions of some vital organ or part into which it is effused. Thus, several ounces effused into pericardium may arrest the action of heart, whereas a similar loss into the abdomen would be negligible. Surprisingly large volume of blood and fluid may get effused beneath the skin and among the muscles and operate as fatally as if it had flowed from an open wound as was seen in R vs. Hopeley, wherein the master had severely beaten a youth of 16 years with a rope and a stick. Though the external wounds were slight, considerable bruising and crushing of the muscles and soft parts was demonstrable at autopsy. Death was attributable to the cumulative effects of violence (there was no mortal wound in the common sense of the term). In an altercation, the accused stabbed the victim on the left forearm. Radial artery was pierced in consequence thereof, and the victim died of haemorrhage sometime later. It was held that forearm was not a vital part, and the offence was covered under Section 320 of IPC. The accused was sentenced to rigorous imprisonment for 3 years [KE vs. Kottengodan Alaxi (1939) 40 CrLJ 308 (Mad HC)].

The quantity of blood loss proving to be fatal depends upon multiple factors. Inhalation of blood in case of wounds of the face or throat can cause death by asphyxia. The young, the aged, and those carrying reduced vitality from some disease may die from loss of blood earlier than those who are healthy and vigorous. In adults, it seems likely that a rapid loss of over 2 litre of blood constitutes an immediate danger to life. Sudden loss of blood has a much more serious effect than the same quantity lost slowly; for example, wounds of large artery or a large vein may speedily end life, as open veins are also liable to admit air into the circulation causing death by air embolism irrespective of the volume of blood lost. Cases of ruptured internal organ leading to concealed haemorrhage pose difficulty owing to the lapse of time before the volume of lost blood produces signs. This may be seen particularly in traffic accidents where organs such as liver or spleen may be injured within their intact capsules. The organ may go on enlarging in size due to accumulation of blood, and continued bleeding may strip more capsule from the parenchymal surface. Eventually, the capsule may rupture and the bleeding now

may pour into the peritoneal cavity. (**Primary haemorrhage** is one that occurs immediately after an injury, whereas **secondary haemorrhage** may occur after several hours or even days after the injury from the same site. This may be due to rise in blood pressure during recovery and muscular movements that loosen the blood clot and the erosion of vessel wall(s) subsequent to infection.)

At occasions, injuries involving arteries may not show immediate copious haemorrhage because of retraction and invagination of musculo-elastic wall. However, slight muscular exertion may cause it to recur and eventually lead to death. Occasionally, even gross injuries (such as amputation of a limb by train wheel) may be almost devoid of significant haemorrhage. Here, it may be explained due to combined effects of crushing with that of arterial wall retraction. If the injury is involving the blood vessel, seat of haemorrhage is obvious and one can evaluate quantity of blood collected in some cavity. However, modification in opinion can be considered in reference to 'open wounds'. Under such a situation, factors like absence of knowledge about the quantity of blood being present at the scene of crime, removal of blood by washing/mopping, or blood-soaked dressings/clothing being destroyed, etc. may prevent any concrete opinion to be furnished. Further, uncertainty as to the amount of blood leaked from the divided vessel after death may be another complicating factor (as soon as the action of heart ceases, the arteries cease to spurt, but so long as the blood remains fluid—or after it is again liquefied—it continues to flow from the divided vessels. Scalp injuries are quite notorious in this aspect, and copious external bleeding can continue after death, particularly if the head is in dependent position at the time of death). **Pathological findings** in death due to gross haemorrhage usually include pallor of the skin, mucous membranes and of the viscera; especially the pallor of the renal cortex, showing an attempt towards compensation for a falling blood pressure. Subendocardial haemorrhages on the septum and papillary muscles of the left ventricle are also attributed to either sudden hypotension or intracranial injury (see ahead also).

Stages of Haemorrhagic Shock

Deterioration of the circulation in shock is a progressive phenomenon and can be divided arbitrarily into three stages:

Nonprogressive Stage (Compensated/ Reversible)

In the early stage of shock, an attempt is made to maintain adequate cerebral and coronary blood supply by redistribution of blood so that brain and heart are adequately perfused and oxygenated. This is achieved by activating various neurohormonal mechanisms producing:

- widespread vasoconstriction (especially in the vessels of the skin and abdominal viscera, whereas regional blood flow to the heart and brain is preserved by vasodilatation of the coronaries and cerebral circulation occurring in response to hypoxia and acidosis), and
- fluid conservation by the kidney through release of aldosterone from the hypoxic kidney and reduced glomerular filtration rate due to arteriolar constriction.

Progressive Stage

This is the stage wherein progressive deterioration occurs that may be due to inadequate initial compensatory mechanisms as described above or due to some accompanying additional factors like pre-existing disease, weak physical status of the individual, etc.

Decompensated Stage (Irreversible)

When the shock is so severe that in spite of compensatory mechanisms and therapy, no recovery is being affected, it is called as decompensated or irreversible stage of shock. A number of factors have been described in irreversibility of shock. These may include progressive fall in blood pressure due to deterioration of cardiac output attributed to release of myocardial depressant factor. **Heart** is more vulnerable to the effects of hypoxia than any other organ. It gets affected in cardiogenic as well as in other forms of shock. Peculiar finding is the development of subepicardial and subendocardial haemorrhages. These haemorrhages are often seen in the left ventricle, on the interventricular septum, and on the opposing papillary muscles. Such haemorrhages can appear extremely rapidly. Bernard Knight reports occurrence of such haemorrhages in a heart that was avulsed from its base during the crash of a military aircraft, obviously causing almost an instantaneous death. Sheehan described these haemorrhages during 1930s to be associated with cases of abortion and pregnancy and therefore, formerly known as Sheehan haemorrhages. They provide an indication as to some catastrophic event shortly before death. Examples may include the following:

- Subsequent to sudden profound hypotension;
- Following intracranial damage;
- Some poisoning, especially acute heavy metal poisoning like arsenic;
- Obstetrical catastrophes have been reported to be particularly showing these haemorrhages, as found by Sheehan.

The mechanism of production of these haemorrhages has been suggested to be mediated through autonomic nervous system. The suggestion is supported by the fact that they 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.

Severe Adult Respiratory Distress Syndrome (ARDS)

It is known by various names such as shock-lung syndrome, diffuse alveolar damage (DAD), acute alveolar injury (AAI),

traumatic wet lungs and post-traumatic respiratory insufficiency. Causes may include the following:

- Shock due to sepsis, burns, trauma
- Diffuse pulmonary infections (chiefly viral pneumonia)
- Inhalation of toxins and irritants (smoke, war gases, metal fumes, nitrogen dioxide, etc.)
- Oxygen toxicity
- Narcotic overdose and drugs like salicylates, colchicine
- Aspiration pneumonitis
- Fat embolism, etc.

Events in the production of this syndrome and the ensuing changes in the lungs are shown in Flowchart 21.1. Depression of vasomotor centre results in **cerebral ischaemia**. Falling of blood pressure below 50 mmHg, as occurring in systemic hypotension in prolonged shock, leads to serious ischaemic damage to the brain tissue. The areas supplied by the most distal branches of the cerebral arteries suffer the most. Usually the changes get noticeable under microscope if the ischaemia is prolonged (for 12–24 hours). Severe **metabolic acidosis** due to anaerobic glycolysis (excessive accumulation of lactic acid in the blood in prolonged shock) enhances the release of catecholamines into the circulation, which leads to effects like release of clot promoting factor, thromboplastin, platelet aggregator factor, and ADP, etc. Excess of lactic acid in the blood can also cause endothelial injury and therefore, can initiate thrombus formation. Consequently, microthrombi may appear and impair blood flow. Persistence of **widespread vasoconstriction** (occurring initially as a protective mechanism) can cause anoxia of the tissues and organs like liver, spleen, kidney and intestines. Consequently, further worsening of functions of these organs occurs.

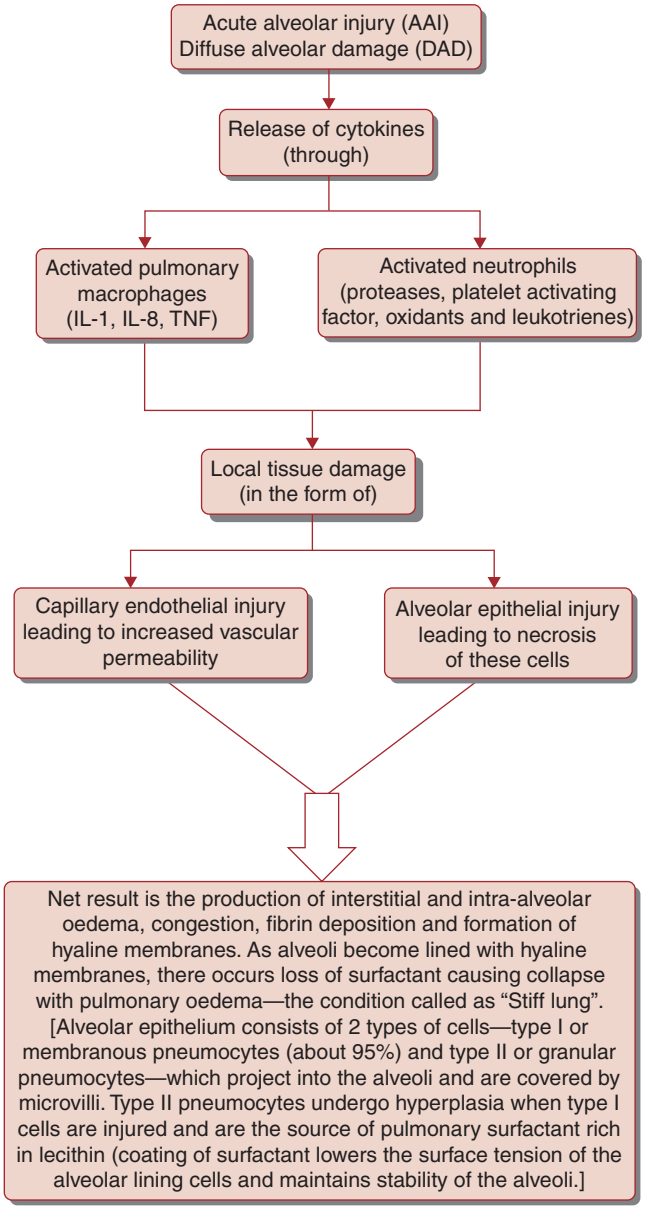
Liver, due to severe hypoxia, becomes helpless in inactivating 'vaso depressor material' (VDM—it is a substance produced by the spleen and skeletal muscle and is normally inactivated in the liver) so that the blood levels of VDM rise, which leads to peripheral vasodilatation and thus diverts blood from the systemic circulation leading to deterioration of the effective circulatory blood volume.

Kidneys undergo acute tubular necrosis (ATN). It is an important complication of prolonged shock, first noted in persons who sustained crush injuries in collapse of buildings in air raids in the World War II. The condition is popularly termed as 'shock kidney'. It can occur following other causes besides shock.

Adrenals show stress response in shock leading to:

- release of aldosterone in response to hypoxic kidney;
- release of glucocorticoids from adrenal cortex and
- release of catecholamines (like adrenaline) from the adrenal medulla.

Intestines undergo haemorrhagic necrosis that results in loss of blood and plasma, causing further reduction in the effective circulatory blood volume. It may result in mucosal and mural infarction called the haemorrhagic gastroenteropathy. In shock due to burns, acute stress ulcers of the stomach and/or duodenum may occur, which are known as Curling ulcers.



Flowchart 21.1 Events in the production of ARDS and ensuing changes in lungs.

AIR EMBOLISM

Entry of air into the circulation is usually resulted from trauma, sometimes surgical or therapeutic, and at other times, from pressure changes (barotrauma) or criminal intervention (criminal abortion). Air embolism comprises of an interruption of the circulatory system by air bubbles (or other gas) that gain access to the circulation, usually through the venous side. The air entering the venous side gets sucked towards the right heart through pulmonary trunk and arteries, rarely emerging on the pulmonary vein side.

In the surgical context, air embolism is produced when negative intrathoracic pressure draws air into an open vein, an event most likely to occur during operations on the head or neck (especially thyroid and neurosurgical procedures) wherein the patient/victim sits upright and the operative wound is above the level of the heart. Other therapeutic procedures like tubal insufflation, pneumoencephalography, instrumental interference of pregnancy, etc. can also lead to introduction of air into the venous circulation (criminal abortion used to be a known reason for deaths occurring due to air embolism from the insufflation of the uterus by a Higginston syringe. The mechanism is obscure but may be attributable to muscular rebound of the uterus sucking air into the cervix).

Accidents may occur during transfusion or infusion, and may form the basis of negligence. Due to use of flexible, collapsible fluid or blood containers in the present times, the danger has minimised. However, where rigid bottles are still used, the need for a vent tube may allow air to enter the connecting tubing when the bottle is allowed to empty completely. And, when the fresh bottle is then connected, the new flow will drive all the air in the tubing into the vein, the so-called 'empty bottle syndrome'.

Injuries to veins of the neck or chest can also lead to air embolism wherein the air gets sucked in due to negative pressure. Some homicides (including 'mercy killings') by deliberate injection of air into the peripheral vein have been reported.

The volume of air necessitating fatal embolism has been debated. Estimates varying from 10 to 480 ml have been reported by Polson (1963). However, in general, embolism by about 100 ml or more has been considered to be fatal. In the rare event of air gaining access to the arterial system, presumably much less would be required to be effective.

Barotrauma is the injury produced in tissues as a result of the inability of gas-filled body cavities such as lungs, middle ears, and sinuses to equalise their internal pressure with the changing ambient pressure. The dissolution of gases into circulation and tissues is governed by Henry's Law, which states that the amount of gas dissolved in a liquid is directly proportional to its partial pressure. Other factors contributing towards dissolution of a gas may include solubility of the gas in the blood and tissues, the blood flow through the tissues and the duration for which gas is breathed at increased pressure. It has been held that air breathed at a depth of 50 m is six times as dense as that at the surface.

Pulmonary barotrauma is an example of barotrauma that produces life-threatening extra-alveolar air syndrome (EAAS). Rapid or panicked ascent (i.e. any ascent greater than 18 m/minute) by a diver while holding breath and closing glottis can easily lead to production of EAAS. This is because the sudden closure of the glottis or the air trapping effect of the lung disease causes the lungs to over-expand (as per Boyl's law), resulting in alveolar rupture (the transpulmonic pressure differential is the pressure difference between the intratracheal and intrapleural environments; the critical level has been documented as 80 mmHg, beyond which alveolar rupture would occur). The air emerging out of the ruptured alveoli can dissect into the pulmonary interstitium, pleural cavities (producing pneumothorax) mediastinum (producing pneumomediastinum), pericardium (producing pneumopericardium), subcutaneous tissues (producing subcutaneous emphysema), etc. Rupture into the pulmonary veins can lead to systemic embolisation with consequent production of arterial gas embolism, which could produce coronary or cerebral artery gas embolism (CAGE).

The other consideration is about the atmospheric pressure outside the body approaching 1 atmosphere at the surface, whereas the gas pressure inside the body tissues and fluids is sum total of the pressures of oxygen, nitrogen, carbon dioxide and water vapour, i.e. considerably greater than the pressure outside the body. Hence, gases escape from the dissolved state and form bubbles and can get removed through the reverse respiratory process. However, this removal takes hours to occur and the eliminating period occasions multiple problems, collectively called **decompression sickness**. (That is why the divers are not advised to board an aircraft within 24 hours after the dive so that he/she may not be subjected to further bubble formation through being exposed to a reduced atmospheric pressure in the aircraft).

Most of the symptoms of decompression sickness are caused by gas bubbles blocking blood vessels in the different tissues. Initially, smaller vessels are blocked by minute bubbles. However, as the bubbles coalesce, progressively larger vessels are affected. **Tissue ischaemia** and, at times, tissue death occurs. Pain in the joints and muscles of the legs or arms is a prominent symptom in most of the sufferers. The joint pain accounts for the term **bends**, often applied to this condition. It often commences within 6 hours, but may occur as late as 36 hours. Micro-occlusion of vessels within the central and peripheral nervous systems produces symptoms such as progressive numbness, dizziness, vertigo, confusion, paresthesiae, ascending motor weakness with urinary and rectal incontinence, paraplegia, unconsciousness, and coma. Pulmonary manifestations can occur shortly after ascent and include dry cough, retrosternal discomfort with shallow and rapid breathing, the so-called **chokes**. Bubbles can also interfere with the coagulation system causing platelet aggregation and disseminated intravascular coagulation (DIC) [blood–gas interaction is capable of activating the intrinsic pathway of the clotting cascade through activation of the Hageman factor]. **Fat embolism** may occur both in post-descent shock and in decompression. Origin of fat embolism in decompression states has been argued. Some claim that it is resulted from air bubble disruption of fat depot tissues, whereas others point to the redistribution of blood lipids, inter-relating with disseminated intravascular coagulation and fibrin production.

Autopsy

Postmortem examination in such fatalities invites thorough appraisal of victim's previous and current medical history and

circumstances in which the death occurred. Autopsy needs be conducted as soon as possible to minimise the effects of decompression occurring postmortem. Apart from general requirements and technicalities involved in an autopsy, some particular steps need emphasis in such cases.

External examination invites critical evaluation. The body needs to be examined while still clothed and equipped (where possible) or such information may be collected from those who have the same. A close observation of the evidence of trauma or hypothermia (hypothermia is sometimes an associated hazard of diving) should be carried out. Perceptibly trivial lesions, including abrasions and/or bruises, may correspond in location and pattern to parts of the diving apparatus, giving an insight into the idea of their origin from excessive pressure or forceful movement at the time of the incident. Subcutaneous emphysema in the face, neck and upper chest, probably, is indicative of pulmonary barotrauma. Otoscopic examination is needed for assessment of barotrauma of the ears. **Radiological examination** prior to dissection is invited. A view taken in the lateral decubitus position may be helpful in demonstrating pneumothorax. The examination must cover the head, neck, chest and abdomen. Radiography or computed axial tomography (CAT) can assist in diagnosing extra-alveolar air in the pleural or pericardial cavities or intravascular locations more easily than the standard postmortem examination. **Evaluation of presence of air in the heart** has been advised through opening of ventricles by incising across the apex with the heart in situ. **Brain and spinal cord** need be preserved for detailed study in cases where neurological dysfunction was recorded antemortem.

Medicolegal Considerations

Most deaths associated with diving are caused by drowning. At occasions, barotrauma might have occurred or contributed towards drowning. Victims of sudden cardiac death may have significant structural disease that has been clinically silent and unidentified through usual screening. In such cases, some form of enquiry is often launched and suits for compensation may creep up. As reported, virtually all of the few cases of arterial air embolism have arisen in relation to dysbarism, wherein bubbles have either been generated within the vessels by decompression, or have gained entry into the arterial system from tears/ruptures of the alveolar capillaries. In such cases, the obvious signs of pulmonary barotrauma (such as subcutaneous or mediastinal emphysema and pneumothorax, etc.) are often absent.

Rarely, investigations as well as examination might reveal no explanation for the death. In such cases, the possibility of under water dysrhythmia may be considered. Another phenomenon known as 'essoufflement' needs be kept in mind. In this, death may result from hypercapnoea. Explanation may be sought in the abnormal rapid shallow breathing under water leading to hypercapnoea in the absence of hypoxia. The triggering factors for this abnormal respiration are believed to include mental stress, elevated breathing load under water, fatigue, cold and diving inexperience.

Wounds Indirectly Fatal/Delayed Causes of Death

Wounds/injuries may not be immediately followed by fatal results, and the injured may develop serious complications leading ultimately to death. Events, in such cases, may conveniently be demonstrable to bear connectivity to the consequences of the injury/wounding. The delay may be short or it may be progressively longer. Wounds of the head and spine are especially liable to cause ultimate death. Whether death is to be attributed to the wounding or not is a matter to be considered from the attending circumstances and detailed autopsy. **In one case,** as reported in the literature, a 51-year-old man was shot in the back and became paraplegic, with subsequent complications including decubitus ulcers, osteomyelitis (requiring a right above-knee and a left below-knee amputation); and neurogenic bowel plus bladder problems. Death occurred 12 years later to the receiving of the bullet injury and was erroneously labelled as 'death due to urosepsis'. Once the remote gunshot wound history was uncovered, the body was requested for proper certification. The autopsy disclosed an ascending purulent meningitis that was associated with sacral osteomyelitis due to large sacral ulcer. The old projectile was recovered from the spine and receipted to the police. The death was certified as due to ascending purulent meningitis following gunshot wound of the back. In the present scenario, such pitfalls may be encountered especially when a network of healthcare workers has been attending to a victim, overlooking the initial condition or injury that initiated the sequence of events leading to the medical complications and ultimately death of the patient/victim. Various causes leading to delayed death may include the following.

SECONDARY SHOCK

Shock is defined as a clinical state of cardiovascular collapse characterised by an acute reduction of effective circulatory blood volume leading to inadequate perfusion of cells and tissues. The ultimate result is the cellular hypoxia and, if uncompensated, may lead to impaired cellular metabolism and death. Secondary shock is the formal shock that occurs due to haemodynamic derangements leading to hypoperfusion of the tissues. If not specified, the term 'shock' is commonly referred to this type of shock and is commonly classified into three major aetiological forms:

- **Hypovolaemic shock**—causes may include severe haemorrhage (trauma, surgery, etc.), fluid loss (dehydration), burns, etc.
- **Septic shock**—due to severe bacterial infections or septicaemia, which may be due to gram-negative micro-organisms

(endotoxic shock) or due to gram-positive micro-organisms (exotoxic shock).

- **Cardiogenic shock**—may be due to deficient emptying of heart (e.g., myocardial infarction, cardiac arrhythmias, etc.); deficient filling of heart (e.g., cardiac tamponade); obstruction to outflow (e.g., pulmonary embolism).

WOUND INFECTION

Wound infection has probably been a major complication of surgery and trauma. It has been documented for at least 4000–5000 years. Egyptians were able to prevent putrefaction as testified by their skills of mummification. The Hippocratic teachings described the use of antimicrobials such as wine and vinegar, which were widely and successfully used to irrigate open, infected wounds before secondary closure at a later date.

Louis Pasteur recognised that microorganisms were responsible for spoiling wine, turning it into vinegar. The concept of 'magic bullet' (Zauber Kugel) that could kill microbes but not their host, first became a reality with the discovery of sulphonamide chemotherapy in the mid-twentieth century. The discovery of antibiotic penicillin is attributed to Alexander Fleming but it was isolated by Florey and Chain. The first patient to receive penicillin was a police constable Alexander in Oxford. Since then, there has been proliferation of antibiotics with activity against a range of bacteria.

Humans come into contact with a variety of microorganisms in every walk of their life. Indeed, many microorganisms exist as 'commensals' or normal flora of the skin, oropharynx, respiratory tract, gastrointestinal tract, and genital tract, etc. Bacteria are normally prevented from causing infection due to factors, namely (*i*) intact epithelial surface; (*ii*) low gastric pH; (*iii*) presence of antibodies, complement and opsonins; and (*iv*) presence of phagocytic cells, macrophages and killer lymphocytes, etc.

Disease/infection ensues when this delicate host–parasite relationship gets perturbed in favour of the microorganisms. However, usually there is a delay before the host defences can become mobilised after a breach in the epithelial surface (whether caused by trauma or surgery). The acute inflammatory, humoral and cellular processes take up to 4–6 hours or so, to mobilise the body's response to a breach in its defences. This is known as the **decisive period** during which the invading bacteria avail the golden period to get established in the tissues. This is the basis for giving antibiotics prophylactically. The susceptibility of the individual depends upon factors like virulence of the microorganism, the dose of the infectious agent, immune status of the individual, malnutrition (obesity, weight loss, etc.), metabolic disturbances (diabetes, uraemia, etc.), colonisation and translocation in the gastrointestinal tract, poor perfusion (systemic shock or local ischaemia) and poor care during or after surgery.

Sources of infection may be **endogenous** (primary infection), i.e. when normal commensals become brave enough to invade the tissues, or **acquired** (secondary infection), i.e. when organisms from outside invade the body as from operating theatre or ward (nosocomial or hospital acquired). Cell injury produced by infectious agents, particularly bacteria, is attributable to toxins (i.e., exotoxins and endotoxins elaborated by these biologic agents). **Exotoxins** are diffusible bacterial polypeptides secreted by the infectious organisms into the surrounding tissues. Bacterial exotoxins may be further divided into:

- **cytolytic**, which interfere with permeability properties of the cell membranes and cause membrane damage through degrading membrane phospholipids, or enhancing pore formation, or
- **bipartite**, which bind to a specific receptor on the target cell by the binding region (B region) of the toxin molecule, and then enter the cell to cause injury by release of toxin.

Endotoxins of bacteria are lipopolysaccharides (LPS) in nature and are constituents of outer membrane of gram-negative bacteria. LPS may induce an array of biological changes including activation of complement, induction of cytokines, and activation of clotting mechanisms. Gram-negative septicaemia may lead to endotoxic shock because of release of LPS. **Bacteraemia** is considered as presence of small number of bacteria in the blood and often do not show significant multiplication. They are commonly not detected by direct microscopy and are demonstrable through blood cultures. **Septicaemia**, however, implies presence of rapidly multiplying, highly pathogenic bacteria in the blood. Septicaemia is generally accompanied by systemic effects like toxaemia, neutrophilic leucocytosis, and disseminated intravascular coagulation (DIC), etc.

Viruses can injure and kill cells by two mechanisms:

(i) By **direct cytopathic effect** that may be the result of the virus co-opting the host cell macromolecular synthetic machinery for its own use and impairing the host transcriptional and translational processes.

(ii) By **indirect cytopathic effect** through triggering a series of immune responses. The viruses replicate, and viral capsular proteins are synthesised in the susceptible cell. The progeny viruses are then released from such a cell without affecting the cell's survival.

PULMONARY THROMBOEMBOLISM

Thrombosis is the process of formation of solid mass in the blood stream (circulation) from the constituents of flowing blood. The mass itself is called the **thrombus**, whereas the mass of coagulated blood formed in vitro is called the **clot**. At times, the thrombus or its part may get dislodged and be carried along in the blood stream as embolus to lodge in a distant vessel. The effects of pulmonary embolism depend mainly on the size of the occluded vessel, the number of emboli, and the cardiovascular status of the victim. Occasionally, a large embolus may get impacted at the bifurcation of the main

pulmonary artery (saddle embolus), or may be found in the right ventricle or its outflow tract. Very rarely, **paradoxical embolism** may occur by passage of an embolus from the right heart into the left heart through atrial or ventricular septal defect. In this way, pulmonary emboli may reach the systemic circulation. Massive pulmonary embolism may result in almost instantaneous death.

Immobility of body parts due to various causes leads to reduced venous return and stasis because of decreased muscular massage of the leg veins, resulting in thrombus formation in deep veins of the legs. Less common sources include thrombi in varicosities of superficial veins of the legs and pelvic veins. Though it is more likely to occur in the ipsilateral limb, yet it can occur contralaterally or bilaterally. Higher incidence of its occurrence in the left lower extremity deep veins may be due to frequent compression of the left common iliac vein between the lumbosacral spine posteriorly and the left common iliac artery anteriorly. **Even prolonged sitting** can lead to deep vein thrombosis, as was observed in persons sleeping in deck chairs in air-raid shelters in the war, and more recently it has been described as a hazard of long air flights or prolonged sitting and working at the computer (e-thrombosis). Various aetiological factors showing inclination of deep veins towards thrombus formation may be enumerated as under:

- Prolonged venous stasis in the lower extremities due to surgical procedures (especially involving head injuries, injury to lower limbs, or pelvic region, etc.) leads to stasis of blood. The relationship of stasis and hypercoagulable states has been studied by Stead and demonstrated radiographically with femoral vein flow measurements and with radioisotopic techniques. It is logical that reduced velocity of venous return prolongs the contact time of activated platelets and clotting factors with the vein wall, thereby encouraging thrombus formation.
- Soleal sinuses have been documented as the principal site of venous thrombosis. Venous blood normally flows from the superficial to deep venous system (perforating veins connect the superficial to the deep venous system, whereas communicating veins connect veins within the superficial or deep venous system). Gastrocnemial or soleal sinusoids of the calf muscle are devoid of any true valves, but small venous channels between these sinusoids help in preventing the reflux.
- Role of injury to venous wall has been well-documented. (Animal models have been used to demonstrate endothelial damage after operations. These endothelial lesions occurred as multiple microtears within the valve cusps and extended through the endothelium and through the basement membrane, exposing subendothelial collagen. Thus, blood elements come in contact with the collagen, which triggers coagulative process.) The triad proposed by **Virchow** (German Pathologist, 1821–1902) more than 140 years ago still carries significance. It narrates three major factors contributing towards thrombus formation, namely (*i*) slowing of circulation; (*ii*) local injury to the vessel wall and (*iii*) injury to the vascular and other tissue. Such factors become instrumental in inducing coagulability of blood that may last for several weeks, the peak being considered as between 1 and 2 weeks. Dating of thrombus/embolus can be of considerable medicolegal importance so as to know if the embolus arose prior to subsequent to some traumatic event.

Dating of the Deep Vein Thrombi and Pulmonary Emboli

Presence of thrombus may be confirmed by transverse cuts across the calf or thigh muscles containing the veins; for this purpose, soleal and gastrocnemius muscles need be transected to examine their contained veins. As thrombo-endothelial junction provides the most information, a segment of the thrombosed vein (if necessary with adjacent muscle) needs to be dissected out of the leg. The thrombus-containing vein is then sent for histological examination. As derived from the literature, an approximate idea of the duration of thrombosis can be gathered as follows:

- Polymorphs may be seen within first day, but they vanish rapidly, often by the next day when mononuclears take their place.
- Beginning of haemolysis in the red cells is usually manifested between 24 and 48 hours.
- Initiation of endothelial proliferation in the form of buds is demonstrable about the second day and enhanced proliferation during the first week.
- Fibrin may be seen as purplish strands using phosphotungstic acid-haematoxylin stain (PTAH), on the first day and spread into sheets by 4th day.
- Haemosiderin may be demonstrable by Perl reaction as blue granules by the end of the first week, reaching maximum in 3 weeks.
- Fibroblasts tend to appear towards the end of the 1st week and reaching a maximum at about 4 weeks. Elastic fibres usually do not appear before 4 weeks. They reach their maximum density in about 2 months.
- Capillaries begin to appear as endothelial buds on the second day but usually are devoid of red cell until about 2 weeks. Endothelial thickening and haemosiderin deposition in the wall of the vein may constitute permanent evidence of a former thrombus.

FAT AND BONE MARROW EMBOLISM

Materials other than thrombotic types may gain access to the blood stream and be carried to a site distant from their point of origin. Nonthrombotic emboli may include fat, bone marrow, air, calcific fragments, portion of atheromas, amniotic materials, and foreign bodies/material such as fragments of bullet, oil, talc, and prostheses, etc. The consequences of these emboli depend upon the nature of material/chemical, the vascular system involved, and the physical status of the victim.

Fat embolism is most often seen after fractures of long bones or compression/laceration of adipose tissue. Abrupt pressure changes during fracture of the bone(s) rupture the thin-walled venous sinuses in the marrow and force the marrow fat into them. In addition, level of plasma triglycerides, free fatty acids and lipase rise as part of the stress response. Also, stress is instrumental in releasing some factor activating disseminated intravascular coagulation (DIC) and aggregation of fat emboli. Other circumstances of fat embolism include orthopaedic procedures, such as joint replacement involving insertion of an intramedullary rod, operations like liposuction and mastectomy, acute pancreatitis, extensive burns, diabetes mellitus, and during introduction of extrinsic fat or oil(s) into the body. After entering the venous system, fat globules usually embolise to the lungs, i.e. appear in the pulmonary capillaries and may somehow leak through the lungs into the systemic circulation causing severe disability in the event of their impaction in the brain, heart, or kidney. Not only fat, but cellular haematopoietic tissue from the bone marrow (especially from the haematopoietically active marrow) can be liberated into the venous system and reach the lungs. This is called **bone marrow embolism**. The emboli usually travel to the lungs at the time of trauma, but the symptoms get delayed, probably because of the time required for hydrolysis of the embolised fat to release fatty acids, which damage the endothelium and activate blood coagulatory process as has been detailed earlier. This so called 'lucid interval' may be confused with the development of an extradural or subdural haemorrhage in cases where cerebral fat embolism is suspected too.

Autopsy Findings

These are typically characterised by petechial haemorrhages. These are caused by the impaction of fat droplets in small venules and may be seen in the skin of any part of the body, especially on the front of chest, face and eyelids. Internally again, they are widespread, but are typically seen in the white matter of the brain, both cerebral and cerebellar hemispheres, as well as the brain stem. Mason found fat in the lungs of 20% of his series of **nontrauma deaths**, but emphasised that quantitatively the amount was small as compared to that found in cases of fatal trauma. He used a simple scale for assessing the histological severity of embolism as seen in Oil Red-O frozen sections of the lung, viz.:

Grade 0: No emboli seen
Grade 1: Emboli found after some searching
Grade 2: Emboli easily seen
Grade 3: Emboli present in large numbers
Grade 4: Emboli present in potentially fatal numbers

Medicolegal Considerations

Mild to moderate degrees of pulmonary fat embolism may not be attached undue weightage since the small fat globules are not likely to appreciably obstruct the vast pulmonary vascular bed. The effects depend upon the size and quantity of fat globules, and whether or not the emboli pass through the lungs into the systemic circulation. Widespread obstruction of pulmonary circulation due to extensive pulmonary embolism can occur and result in unexpected rapid death. The picture is that of ARDS (marked pulmonary oedema is the pathological marker for this syndrome as the small blood vessels of the lungs are chemically injured by high plasma levels of free fatty acid, resulting in increased vascular permeability and consequent pulmonary oedema). Pulmonary infarction is usually not a feature of fat embolism because of the small size of globules. In routine stains, the fat globules in the pulmonary arteries, capillaries and alveolar spaces appear as vacuoles. Frozen section is essential for confirmation of globules by fat stains such as Sudan dyes (Sudan Black, Sudan III and IV), Oil Red-O and osmic acid. **At occasions** (e.g., in a body recovered from water), fat or marrow embolism can be used as a marker for differentiating antemortem and postmortem fractures, because any trauma inflicted/sustained after stoppage of cardiac function (which may be the situation where dead body drifts/thrown in water and injuries are sustained/received while in/under water, including some fracture) cannot transmit fat or marrow to the pulmonary capillaries.

CRUSH SYNDROME

The association between crush injury, rhabdomyolysis and acute renal failure was first reported during the World War II in victims trapped during the 'London Blitz'. It has been reported in earthquake and mining accident survivors, after excessive exercise and when limbs have been forced into abnormal postures for prolonged periods, extensive burns, and certain poisons such as mercuric salts or carbon tetrachloride, etc.

Crushed muscle also sequesters many litres of fluid, reducing the effective intravascular volume, which results in renal vasoconstriction and ischaemia of the kidney. Myoglobin (released from the crushed muscles) gets concentrated in the tubules, leading ultimately to tubular necrosis. However, this theory of tubular damage has been questioned since the biopsy materials have been found to show minimal tubular damage. The true mechanism is still obscure but has been suggested to be due to involvement of juxtaglomerular apparatus and the renin–angiotensin system as well as disseminated intravascular coagulation effects on the glomeruli.

SUPERVENTION OF SEPARATE PATHOLOGICAL STATE

Occasionally, though an injury by itself is not fatal in the usual sense, the development of some other pathological state may prove fatal. For example, development of a fibrous scar in the hollow muscular organ may produce stricture and obstruction, fibrous scar in the wall of an artery may bulge into a traumatic aneurysm, damage of an artery by a bullet/missile may produce false aneurysm (which may rupture later), direct injury to the

coronary artery through thoracic trauma may be followed by coronary thrombosis, etc. A wound of the abdominal wall may be followed by strangulated hernia with fatal results. The circumstances of connectivity of the development of the condition to that of injury may be cloudy. The proof of injury and appearance of disease within a reasonable lapse of time in an already healthy individual need to be documented unambiguously. **Ewing's postulate** showing relationship between trauma and new growth consists of:

(i) evidence of previous integrity of the part affected,
(ii) undeniable and adequate trauma must be proven,
(iii) a reasonable—neither too short nor too long—time interval should elapse before symptoms of growth/tumour develop,
(iv) the disease must develop in the exact location of the injury, and
(v) the nature of the tumour must be proved microscopically, if possible.

EXACERBATION OF A PRE-EXISTING DISEASE

Some pre-existing disease may get accelerated by trauma/assault. For example, a diseased spleen or an aneurysm may rupture following an injury/assault, wherein the assault or the injury may be a precipitating factor in hastening/accelerating death. Explanation I attached to the Section 299 IPC dealing with 'culpable homicide' speaks of this concept. The explanation reads as, "A person who causes bodily injury to another who is labouring under a disorder, disease or bodily infirmity, and thereby accelerates the death of that other, shall be deemed to have caused his death." The scenario may be quite knotty, especially for the doctor, as the relatives who are commonly unaware of any pre-existing ill health of the victim or the deceased are usually disinclined to accept the medical findings. Under such circumstances 'cause and effect' relationship becomes a complicated problem. The issues needing consideration under such circumstances may include the following:

- Whether the disease was solely responsible for death, and the death would have occurred irrespective of the injury.
- Whether the injury was entirely responsible for death and the death would have occurred whether or not the disease was present.
- Whether the death was caused by a combination of these.

The common situations involve coronary artery disease, pulmonary embolism and subarachnoid haemorrhage (for details, see Chapters "Sudden and Unexpected Death", "Complications of Trauma" and "Regional Injuries", respectively).

OPERATION AND/OR ANAESTHESIA

A doctor cannot swear that an operation would have saved the life of the victim. He can only affirm that it might have provided the victim a better prospect of recovery. The law regards an operation necessitated on account of an injury to be a consequence of that injury, and the person responsible for the injury is held to be responsible for all natural consequences of the injury (including complications arising out of the operation or anaesthesia). However, the factors like (*i*) necessity for the operation, (*ii*) surgeon's competency and (*iii*) sufficiency of the injury to prove fatal in the ordinary course of nature, etc. are accorded due weightage by the courts in arriving at the ultimate outcome of the case.

In R vs. Draper, the accused through his careless driving knocked down the deceased wherein a wheel passed over his left arm just above the elbow. The surgeon advised immediate amputation of the arm. The victim refused to submit to the operation. The arm was therefore dressed and splint applied. However, a week later, the victim consented to the operation. The arm was amputated, but the victim died of pyaemia (one of the complications of the operation). Though the medical opinion forwarded was that the victim's life could have been saved if he had allowed immediate amputation, yet the judge directed the jury to look into the fact of sustaining the injury by the victim through the negligence of the accused, and ultimately leading to the death of the victim (refusal by the victim for submitting to an operation was another matter to be dealt under appropriate provisions of the law). Explanation 2 attached to Section 299 IPC dealing with 'culpable homicide' supplements this concept. The explanation reads as, "where death is caused by bodily injury, the person who causes such bodily injury shall be deemed to have caused the death, although by resorting to proper remedies and skillful treatment, the death might have been prevented".

NEGLECT OF/BY THE INJURED

Rarely, death may occur from the complications arising out of an injury that are otherwise not sufficient to lead to death in the ordinary course of affairs. This may happen due to improper treatment, or negligence on the part of the doctor, or to the negligence or willful disobedience on the part of the victim/patient. In such cases, the issue of negligence/contributory negligence may creep up.

While discussing complications of trauma/injuries and their implications towards legal outcome, **it is prudent to discuss the role of the weapon or instrument causing the wound/injury**. So far as the medicolegal expert is concerned, he/she has to bear in mind that the type of the injury caused depends upon the way in which it is caused, which includes the type of the weapon. Thus, the type of the injury often throws light on the incriminating weapon, i.e. the nature of weapon as ascertainable from the nature of injury is useful as the gravity of offence and the severity of punishment are guided by these factors. **The word weapon** may be taken to mean (as derived from various dictionaries) 'thing designed or used for causing physical harm' or 'a means employed for trying to gain the advantage in a conflict' or 'any means that may be used against

an adversary' or 'any article made or adapted for use for causing physical harm to the person or intended by the person having it with him, for such use by him', etc. Thus, in Phool Kumar vs. Delhi Administration, it was held: "It is not necessary that deadly weapon must be actually used by the culprit in the robbery or dacoity by way of causing hurt or brandishing the same and that it is 'used' within the meaning of Section 393, IPC, if the deadly weapon is merely held out for terrorising or frightening a victim to obtain property" [AIR 1975 SC 905: 1975 SCC (Cr) 336: (1975) 1SCC 797: 1975 CrLJ 778].

An opinion as to whether or not a particular weapon could have caused a certain injury can usually be answered without much difficulty. It is a fact that a weapon seldom matches wounds with fingerprint accuracy unless some part of it has been left in the wound/track. At occasions, weapon is distinctive enough in its configuration so as to enable the witness to state that there is a fairly good match between the wound and the weapon, but the things are seldom unequivocal (the cases like purposeful implantation of pellets need be born in mind). Sections 323 and 325 IPC are general Sections for providing punishment for voluntary causing hurt and grievous hurt, respectively. Sections 324, 326, 327, 328, 329 and 330 deal with the offences committed under certain aggravating circumstances. (Section 324 IPC describes **dangerous weapons or means**. The culprit is liable to more severe punishment where the harm caused has the differentia of one of the modes of infliction described in these. Such weapons or means described are any instrument for shooting, stabbing or cutting; or any instrument that, 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 that is deleterious to human body to inhale, to swallow, or to receive into the blood; or by means of any animal.) Some interesting points about weapons and their implications appear as under:

- Wounds of different character may enable separate populations of wounds to be distinguished, implying the use of two or more different weapons, although the number of assailants is not easily ascertainable.
- Glass is a good example of material that may lacerate or incise according to the circumstances (Brawler's beer glass that may lay open the face cleanly but, at the same time, mangle nearby tissues).
- There is a spectrum of injuries between linear laceration and ragged incise wound (determined by the extent of sharpness of the blade of the instruments/weapons). It is important to make the distinction, because the resulting opinion regarding the nature of weapon will heavily weigh to the legal outcome of the case.
- A stick becomes a dangerous weapon by reason of an iron knob at its top, and when that part of a stick is used as a weapon of offence, it is likely to cause death, and thus the offence committed with such an instrument would fall under Section 324, IPC [Koli Gator Sura vs. State of Gujarat, AIR 1966 Guj 221].
- Death caused by squeezing of testicles is not an injury that is sufficient to cause death, but it is one likely to cause death [(1975) 2 SCC 7: 1975 SCC (Cri) 384, Bai Fatima].
- In deaths caused by hitting on the head by the wrong side of an axe, intention to kill cannot be inferred though knowledge that the injury caused would lead to death in the ordinary course of nature can be attributed [(1975) 3 SCC 751: 1975 SCC (Cri) 211, Mohinder Singh].
- Where the accused continued beating the deceased with a hot iron '*chimta*' (iron rod used in kitchen) to take out an evil spirit, it was held that the large number of injuries inflicted with a hot iron '*chimta*' had certainly endangered the life of the deceased and as such the accused was liable under Section 326 IPC (Pratap Kumar vs. State of Punjab, 1976 CrLJ 818).
- The fist blow caused by the accused resulted in subdural haematoma that led to the death of the victim, but it could not be said that the accused could be attributed with the knowledge that by such act he was likely to cause death, nor could it be said that the accused intended to cause the particular injury that he actually caused, it was held that accused could be convicted only under Section 323 and not Section 300 [Thomas vs. State of Kerala, 1992 CrLJ 581 (Ker)].
- The accused threw acid on faces of their victims. Medical evidence showed that the injuries caused on the faces and eyes were not sufficient to cause death, conviction of the accused under Section 307 was altered to one under Section 326. The court observed that unless it can be shown that the intention or knowledge of the accused was to cause such bodily injury as would come within one of the four clauses of Section 300, he cannot be held guilty of an offence under Section 307 [Kulmani Sahu vs. State of Orissa, 1994 CrLJ 2245 (Ori)].
- Where the accused gave a push on the chest of the deceased, and the victim fell on a stone resulting in death, conviction was recorded under Section 323 [Pichapillai vs. State, 1996 CrLJ 3634 (Mad)].
- It was contended that knife was found from a nearby water pit after about 24 hours, no blood would have remained on it, cannot be accepted as the evidence shows that the knife was closed before it was thrown in the water pit. It follows that one must search blood on body, under nails, clothes, shoes, button holes, turn of pants, seams, inside the pockets, heels of shoes, etc.; and in case of weapon, on the blades, cervices and cracked handles, etc. [Mahipal alias Mahaveer Singh vs. State of Rajasthan, AIR 1998 SC 864: 1998 SCC (Cr) 707: 1998 Cr LJ 1257: JT (1998) 2 SC 661: 1998 Cr LR (SC) 350].

22 CHAPTER

Medical Education vis-à-vis Medical Practice

After going through this chapter, the reader will be able to describe: Ethics and moral in relation to medical practice | Various 'codes' of medical ethics | Constitution and powers of the Indian Medical Council | Constitution and powers of the State Medical Council | Code of ethics by Medical Council of India | Duties of doctors towards patients and towards each other | Duties of doctors towards the state | Professional secrecy and privileged communication | Professional misconduct/infamous conduct/malpractice | Difference between unethical conduct and misconduct | Red Cross emblem policy

The word **'ethics'** is derived from the Greek term 'ethikos', which stands for rules of conduct that govern natural disposition in human beings; the word **'morals'** is taken from the Latin 'mores' concerning with goodness or badness of a human character or behaviour. These words are virtually synonymous although their meanings have been distinguished in common parlance. For example, 'morals' in popular sense has been narrowed mainly to matters of sexual or social behaviour, and 'ethics' is most often applied to matters of professional behaviour. Medical ethical problems that once were no more than entertaining speculations about the future are now a reality, and medical ethics can no longer be regarded as dessert in the smorgasbord of medical education. The contemporary construction of bioethics was first well-formulated in the United States in the mid-1970s by the statement of four principles of bioethics elucidated by Beauchamp and Childress:

(i) **Nonmalfeasance** (duty to endeavour to do no harm)
(ii) **Beneficence** (duty to do good, be caring, etc.)
(iii) **Respect for autonomy** (duty to respect the human dignity, freedom and free choice of the individual and more importantly, to facilitate in every reasonably possible manner the making of such a free choice)
(iv) **Natural and distributive justice** [i.e. ensuring equal share in the distribution of healthcare (equality) and priority to be given to those who are in need, in proportion to their need (equity)].

Ethics, morality and law are closely related but are not synonymous. Morals are mainly derived from religious percepts and practices, and therefore are not open to logic and argument. Flouting of morals may result in feelings of guilt or may invite censure from society. Ethics, on the other hand, are intellectually derived by a specific group/profession for its specific needs and can be changed or modified with the change in situation. The code of ethics generally retains the moral guidelines and cannot run contrary to the generally accepted morals of the society. Needless to say, the ethical code must also be in conformity with law of the land. For instance, if euthanasia is not allowed by law in a country, no medical group can include euthanasia as an acceptable practice in that country. Code of ethics is, however, different from law since nonadherence to the prevailing ethical standards may not be considered as an offence by law.

Apart from the control of medical profession by the legislatory actions of the government through the various legal provisions, other controlling influence upon the medical practitioners is through the 'medical ethics', which is a code of behaviour imposed by the profession itself depending upon the moral values. Disobedience and violation of it, though not an offence legally, will be considered unprofessional or unethical from the moral point of view. However, it is important to distinguish between an **ethical dilemma** and an **ethical violation**. For example, one is faced with an ethical dilemma between two ethically legitimate alternatives, such as preserving the patient's confidentiality or protecting third parties likely to be affected by the patient's behaviour. Under such circumstances where confidentiality interests of the patient conflict with the safety interest of another person, a duty is usually imposed on a doctor to warn the individual(s) who is/are the subject(s) of threat(s) even if it does not seem likely that threat(s) will actually be carried out. This erring on the side of prevention of harm has been favoured by most ethicists.

Codes of Medical Ethics

Although the laws enacted by the Central and State Governments exist to regulate the medical practice in India, yet there remains a large sphere that is left to the conscience of the doctors to be governed by the moral values, where the limits or prohibitions are set not only by fear of law or by censure of the Medical Councils but by the good reputation that doctors wish to enjoy in the eyes of their professional colleagues.

The first and basic code of medical ethics is almost 2500 years old. This is the universally known **Hippocratic Code**, laid down by Hippocrates and his school in ancient Greece. Hippocrates was the 'Father of Western Medicine' and lived in the Island of Kos in Greece in the fifth century. The well-known Hippocratic Oath is still subscribed to by students in many medical schools or if they do not actually take the oath, they accept its basic philosophy.

The Declaration of Geneva (1948)

After the second World War, the World Medical Association at its third General Assembly at Geneva in September, 1948 devised a modern version of the Hippocratic oath, known as the Declaration of Geneva.

The Declaration of Geneva, 2006, reads:

- I solemnly pledge to consecrate my life to the service of humanity;
- I will give to my teachers the respect and gratitude that 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 that are confided in me, even after the patient has died;
- I will maintain by all the means in my power, the honour and the noble traditions of the medical profession;
- My colleagues will be my sisters and brothers;
- I will not permit considerations of age, disease or disability, creed, ethnic origin, gender, nationality, political affiliation, race, sexual orientation, social standing or any other factor to intervene between my duty and my patient;
- I will maintain the utmost respect for human life;
- I will not use my medical knowledge to violate human rights and civil liberties, even under threat.

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

Medical Councils

No one can hold himself out to the public as being a 'Registered Medical Practitioner' unless he has proper qualifications from a recognised medical college and has got himself registered by one of the Medical Councils of India. This regularisation was begun in 1916, when Government of India passed a law confining the grant of titles implying qualifications in western medical science by their Act No. VII of 1916, called the 'Indian Medical Degrees Act'. This prohibited the use of a title implying these qualifications by unqualified persons.

Indian Medical Council Act, 1933

This was passed by the Indian Legislative Assembly in 1933 to safeguard the statutes of various medical degrees awarded by different Indian universities. The Act also envisaged to maintain uniform standard of medical education in the country.

Indian Medical Council Act, 1956

This Act was enacted by repealing the Act of 1933 to include the following amendments:

(a) To give recognition for representation of the licentiate medical practitioners.
(b) To provide registration of foreign medical qualifications.
(c) To provide for formation of a committee to help in reorganising postgraduate medical education in the country.
(d) To maintain Medical Register containing names of registered medical practitioners all over India.

But the Act was amended in 1964 to reconstitute the Medical Council of India and reorganise medical education of the country. The Act extends all over India except the State of Jammu and Kashmir.

Constitution of the Indian Medical Council

(a) One member from each State other than a Union Territory to be nominated by the Central Government in consultation with the respective State Government.
(b) One member from each University, to be elected from amongst the members of Medical Faculty of the University by members of its Senate.
(c) One member from each State where a State Medical Register is maintained, to be selected by persons amongst themselves, enrolled in the said Register.
(d) Seven members are to be elected from amongst persons enrolled in any of the State Medical Register.
(e) Eight members are to be nominated by the Central Government. The President and Vice President of the Council

are to be elected from amongst the members of the Council. The members of the Council will hold office for not more than five years.

The Executive Committee of the Council to execute the functions of the Council is to be constituted from amongst the members, comprising President, Vice President and 7–10 other members. The Council appoints a Registrar and a Secretary for its day-to-day functioning. Dr. BC Roy was the first Indian to be the President of the MCI in 1939.

FUNCTIONS OF THE INDIAN MEDICAL COUNCIL

Maintenance of Medical Register

The Register contains the names, addresses and qualifications of the medical practitioners who are registered with any State Medical Council. It is known as the Indian Medical Register. Removal or erasure of the name from the Register of the concerned State Medical Council will tantamount to its removal from the Indian Medical Register. The Register will be considered as a public document within the meaning of Indian Evidence Act.

Regulation of Standard of Undergraduate and Postgraduate Medical Education

The Council has an undergraduate and postgraduate committee for this purpose. The Council prescribes courses and criteria that a medical institute or college should fulfill for launching a particular course of study. For this purpose, the Council will send its inspectors to see that the college is adequately spaced, staffed and equipped as per the Medical Council of India stipulations. The inspectors will also enquire about the courses of study, teaching and training of the students and may also visit the institution during the examinations to assess the standard of examinations. On the basis of reports of the inspectors, the Medical Council recommends to the Central Government for the recognition or nonrecognition of the medical qualifications given by that university or medical institution.

Recognition of Foreign Medical Qualification

The Council may permit registration of Indian citizens who have obtained foreign medical qualifications and is also empowered to negotiate with the respective authority of any country for mutual recognition of the qualifications.

Derecognition of Medical Qualification

The Council can make representation to the Central Government to withdraw recognition of a medical qualification of any college or university if, on receipt of report from the inspectors, it feels that the standards of resources/training/teaching are not satisfactory. The Council, on getting assurance for future rectifications of all defects and deficiencies by the institution, can send its recommendation to the Central Government for reconsideration. The Central Government will again refer it to the Council which, in turn, will send its inspectors to visit the institution for verifying the implementation of the assurances and will send their reports accordingly. Until recognised, holders of such medical qualifications from the said college or institution are not held at par with those having recognised qualifications.

Appellate Powers

When a registered medical practitioner, being dismayed by the disciplinary action taken against him by the State Medical Council, appeals to the Central Government, the Central Government consults the Medical Council of India which, in turn, gives its recommendations after going through the circumstances, which is a binding to the appealing party and the concerned State Medical Council.

Warning

The Council can issue 'Warning', i.e. a cautionary notice on finding a doctor guilty of some unethical conduct after due enquiry. This needs be differentiated from 'Warning Notice', which comprises of information about certain unethical practices known as 'infamous conduct' in professional respect. This list is given to doctors at the time of registration with the council, and later periodically. Through this, doctors are self-warned about such offences and likely consequences.

Composition of the State Medical Council

- Medical teachers from different universities of the State elected by the teachers of the different medical institutions.
- Registered medical practitioners elected by the registered medical practitioners of the State.
- Some members nominated by the State Government.

The members of the State Medical Council elect a President and a Vice President from amongst themselves. They also elect the members of the Executive Committee, Penal and Ethical Committee, etc. A Registrar is appointed to look after the day-to-day working of the Council, who also acts as its Secretary.

FUNCTIONS OF THE STATE MEDICAL COUNCIL

Maintenance of Medical Register

The Registrar of the Council maintains a register to provide for the registration of medical practitioners within its jurisdiction. On payment of prescribed fees, the name, address and qualifications are entered in the Register. A provisional registration is granted to a doctor who has passed the qualifying examination but has to undergo a certain period of training (Rotatory Internship for one year) in an approved institution and permanent

registration is granted after that training period. It is the duty of the Registrar of the State Medical Council to inform the Indian Medical Council about the additions or deletions or amendments made in the State Medical Register, from time to time.

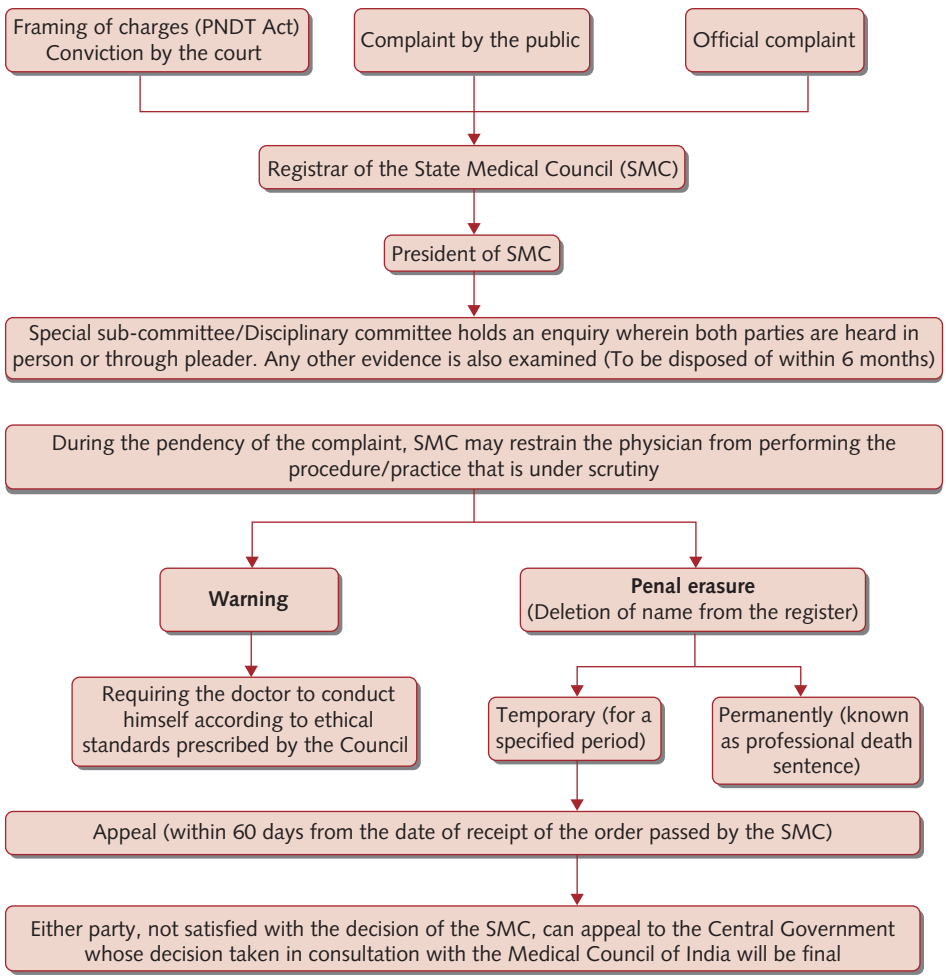
Exercise of Disciplinary Control

The State Medical Council is entrusted with the disciplinary control over the registered medical practitioners of the State enrolled in the Register. The council by itself does not start any proceedings but takes cognisance of any misconduct (professional) of a medical practitioner and deals with the complaint through its Disciplinary/Ethical committee. Various steps have been outlined in the Flowchart 22.1.

Professional Secrecy

The secrets confided to a doctor by the patient during the course of his professional service tantamount to an implied contract between the parties, which is known as "professional secrecy". Any divulgence, therefore, will amount to breach of contract, trust and confidence, and will render the doctor liable for damages. The secrets may be learnt from the patient or found on examination or noticed in the usual privacies of domestic life. Consequently, most commentators include **privacy and confidentiality** in this context. **The former** is a broader right that everyone enjoys with respect to personal/private information. Right to privacy may be considered as person's right to be free from unwarranted publicity and intrusions. It usually comprises of unauthorised dissemination of private information about the person or the publication of material or records. **The latter** is somewhat narrower right as it is specifically used in relation to dissemination of medical information in the course of diagnosis and treatment of an illness or injury. The concept of confidentiality also carries significance towards the well-being of the patient in the sense that when the patient is not apprehensive of leakage of information, he/she is less likely to conceal information, including even the intimate matters concerning sex life, social and moral behaviour, etc.

Further, sharing of the health information amongst the healthcare professionals on 'need to know' basis is now understandable since presently, medical care has become more complex and relies upon 'an enlarging patient care team' (necessitating access to patient's record amongst doctors, nursing staff,



Flowchart 22.1 Usual steps adopted by the State Medical Council in dealing with a complaint.

technicians, dietitians, physiotherapists, social workers, etc.). This is especially so in hospitals where 'resident' and other coverage is arranged in shifts. This has evolved the concept of 'necessity to realise' on the part of the patients that any confidential information given to an attending doctor will probably pass through other doctors and health professionals. In addition, requests for information from insurers, employers, police, lawyers and Government agencies for statutory, legal, financial or other reasons have increased. Influence of factors such as freedom of information, concepts of openness and accountability, quality assurance, close involvement of patients in understanding their illnesses and making decisions to accept or refuse treatment, etc. is progressively heaping up. In the light of foregoing discussion, it is well-recognised that some overriding public interests may require breaches of these parameters, and in appropriate circumstances the autonomy of the individual must yield to the higher interests of the public and the State, especially in the prevailing societal shake-up involving third-party payers, fourth-party auditors, and the varied legitimate needs for the healthcare information (see 'Privileged Communication' ahead). A few circumstances for exercising caution are enumerated as under:

- The doctor is not to discuss the illness of the patient with anyone else without his consent.
- He should not reply to queries of others including near relatives about the nature of illness, its sequelae, without the express consent of the patient.
- The doctor is not to disclose the nature of illness of the patient, even when requested by a public or statutory body except in case of notifiable diseases, without his consent. When the patient is minor or insane, the consent of the guardian has to be taken.
- The doctor is not to unveil secrets confided and nature of illness detected in respect of the husband to his wife and vice versa, without the expressed desire of the person concerned.
- The doctor should not reveal any secrets relating to the nature of illness in respect of a servant examined to his master without the consent of the servant, even though the master may be paying the doctor's fees.
- When the doctor is asked by the government to examine a patient, who happens to be a government servant, he will examine him but not disclose the nature of his illness to the government without the express consent of the patient, though in certain respect, the report may be privileged.
- The doctor, when examining an under trial prisoner in police custody, is not permitted to disclose the nature of illness of the patient to the authorities, unless expressly so desired by the patient himself. But convicted person has no such right to prevent the doctor from disclosing the nature of illness or result of the examination to the authorities. Under Section 53 CrPC, an arrested person may be examined forcibly by the doctor using reasonably necessary force at the request of the police to elicit material evidence, if any.
- Similarly, a farm or factory medical officer cannot disclose the results of examination of a farm or factory worker to the factory authorities without the consent of the employee.
- When a person undergoes medical examination to undertake Life Insurance Policy, his conduct conveys voluntary consent to the disclosure of the findings to the authorities concerned. Any information about a deceased patient, when treated before, can be given when asked by an insurance company only after obtaining proper consent from the legal heirs of the deceased.
- The doctor should not divulge any information about any patient without his/her express consent in cases of divorce and nullity.
- Maintenance of secrecy of medicolegal/postmortem is implied and the medical officer should see that this secrecy is maintained.
- While reporting a case in any medical journal, care should be taken not to disclose the name or photograph of the patient, unless he extends express consent in the matter.

PROFESSIONAL SECRECY IN CRIMINAL MATTERS

Though the doctor owes to his patient the obligation of professional secrecy, yet under Section 202 IPC, he is required to bring to the notice of the police any information about any criminal act that might come to his knowledge in the course of his professional service. A person might come with a wound (a firearm wound or a stab wound) that supposedly had been sustained during some scuffle; the doctor would be wrong in suppressing the facts. In such matters, he cannot claim protection under the garb of professional secrecy. Similarly, he should see that a case of 'rape' or 'assault' is brought to the notice of the police preferably by the victims themselves, if not, he can inform the police himself to safeguard the interest of the community at large.

The practitioner's responsibility in respect of cases of abortion is somewhat peculiar. If he is called to treat a woman upon whom criminal abortion has been attempted or performed, he should treat the case and make a detailed record and should preferably consult another practitioner. If the condition of the woman is serious, police should be informed and dying declaration may be recorded.

Privileged Communication

This is bona fide unprejudiced statement of a doctor on a subject matter of public interest to the concerned authority as a part of his duty to protect the interest of the community at large or of the state.

A medical person though not expected to communicate the secrets divulged to him by the patient during his treatment, yet he is privileged to do so in the interest of the community at

large. This sort of communication is to be unbiased, bona fide and without malice. **A 'privileged communication' may thus be regarded as** a communication extended by a doctor to a proper authority who has corresponding legal, social and moral duties to protect the public. When the privilege is claimed, the facts alone would determine the issue. Such a privilege can be claimed and disclosure of professional secrets is justified under the following circumstances.

IN RESPECT OF EMPLOYEES AND EMPLOYERS

A doctor is duty bound to inform the Superintendent of a hostel if any of the servants of the hostel suffers from any communicable disease, such as an open case of tuberculosis, syphilitic ulcers, gonorrhoea, etc.

A doctor should report to the respective employer if he finds a car/bus/tram driver, a ship's captain or a pilot suffering from epilepsy, high blood pressure, drug addiction, alcoholism, colour blindness, etc. He should first try to persuade the patient to change his employment because of the inherent dangers to himself and to others. If the persuasion fails, he should inform his employer that the said patient is unfit for that kind of employment.

DISCLOSURE IN PATIENT'S INTEREST

Disclosure of the disease to some other person may force the patient to get proper treatment. It is to be done ordinarily with the patient's tacit approval. But, at times, the doctor will have to inform the guardian or other relatives of the patient about the state of his melancholia or suicidal tendency without informing him.

WHEN SUFFERING FROM VENEREAL DISEASES

If a syphilitic patient is about to marry, the doctor should try to persuade him not to marry before he is cured, but if he does not listen, the doctor can divulge the matter to the woman he is going to marry or to her guardians. When a married partner is found to suffer from syphilis, the attending doctor should insist on cessation of sexual intercourse by him until the disease is cured, without giving any hint about it to the other partner. Persons suffering from syphilis and/or gonorrhoea in communicable form should be prohibited to use swimming pools.

WHEN SUFFERING FROM INFECTIOUS DISEASES

As part of statutory duty, a doctor is duty bound to notify infectious diseases to the public health authorities. When a cook, a waiter or a nurse is found to be suffering from an infectious disease like smallpox, cholera, typhoid, etc., he should not be allowed to work until he becomes noninfectious. If the patient does not listen to the advice of the doctor, then the doctor can communicate the matter to the employer of the patient.

DISCLOSURE IN DOCTOR'S SELF-INTEREST

Both in civil and criminal suits by the patient against the doctor, evidence about his condition may be furnished unhesitatingly. Where a doctor owes a statutory duty to the State, he must notify births, deaths, communicable diseases, food poisoning cases, etc. to the concerned authorities.

IN COURTS OF LAW

The doctor would not ordinarily divulge professional secrets but if insisted by the Courts of Law, he should request the judge to allow him to give the information in writing. However, he should not voluntarily disclose any information either in the Court or out of it. Under such situations, the doctor should write the facts about the patient in a paper, which is to be enclosed in an envelope, and forwarded to the proper authorities, inscribing on its top, 'Privileged Communication'.

PROFESSIONAL SECRECY IN RESPECT OF DEAD

An autopsy surgeon while examining a dead body may come to learn certain facts that, on disclosure, may affect the reputation of the deceased, socially or materially, or it may cause mental suffering to the relatives. Therefore, it is expected that he should maintain secrecy about these facts, from ethical and moral point of view as he does in respect of the living.

PROFESSIONAL SECRECY AND INSURANCE REPORTS

When any doctor is appointed on the panel of the Life Insurance Corporation, he should furnish all information about the patient without reservation, however detrimental this report may be to him. The report by the doctor on the health of the proposer is privileged. However, there may be cases where the proposer had been previously treated for some ailment and there may be no signs of it at the time of examination. Here the doctor need not report the disease from the previous knowledge as it would amount to breach of professional secrecy.

Unethical Conduct and Misconduct Differentiated

'Ethics' stand for rules of conduct concerned with the distinction between right or wrong (good or bad) that govern natural disposition in human beings. These may be regarded as intellectually derived principles by a specific group/profession aimed at differentiating between right or wrong, good or bad and so on. These are amenable to change/modification with the change in situation/circumstance. Therefore, it is hard to find any medical activity that does not have some ethical considerations, varying from informed consent to doctor–doctor relationship,

from medical confidentiality to research on human beings, from accidents to custodial torture and so on. Despite all this, the basic nature of ethical behaviour remains the same and all medical ethics can be said to rest on the principle that "the patient is the reason for the doctor's existence and all other matters must be subservient to this fact". In other words, a doctor's over-riding consideration remains towards the patient, while also accepting for his duty towards colleagues and to the community at large. Disobedience/violation of ethics, though not an offence legally, yet it will be considered unethical from the moral point of view and invite action from the Medical Council. **'Misconduct'** is a generic term and means 'to conduct amiss; to mismanage; wrong or improper conduct'. It includes malfeasance, misdemeanour, delinquency and offence. It is something more than accident or negligence/carelessness and is the culpable neglect of an official/professional in regard to the office/profession. It implies some degree of 'mens rea' on the part of the person concerned or, at any rate, a very grave degree of negligence, or serious failure to carry out instruction.

The expression 'Infamous Conduct' was first used in the Medical Act 1858 of England. In 1894, Lord Justice Lopes considered the meaning of the expression and said, "If it is shown that a medical man in the pursuit of his profession has done something with regard to it that would be reasonably regarded as disgraceful or dishonourable by his professional brethren of good repute and competency, then it is open to the General Medical Council to say that he has been guilty of **infamous conduct in a professional respect**." This concept, sometimes called as **'pear conduct'**, holds true even today, and it is impossible to describe any universal rules because of varying systems of licensing and registration in the various countries. Nevertheless, some general ethical standards, morality and competence need be adhered to. These standards are not born of snobbery or elitism but of practical necessity. Presence or absence of 'misconduct' needs be construed with reference to the subject matter and the context wherein the term is being applied/constructed (Table 22.1).

Professional Conduct, Etiquettes and Ethics for Registered Medical Practitioners

MEDICAL COUNCIL OF INDIA NOTIFICATION

New Delhi, dated 11th March, 2002 No. MCI-211 (2)/2001/ Registration. In exercise of the powers conferred under Section 20A read with Section 33 (m) of the Indian Medical Council Act 1956 (102 of 1956), the Medical Council of India, with the previous approval of the Central Government hereby makes the following regulations relating to the Professional Conduct, Etiquette and Ethics for registered medical practitioners, namely.

SHORT TITLE AND COMMENCEMENT

1. These regulations may be called the Indian Medical Council (Professional conduct, Etiquette and Ethics/Regulations, 2002).
2. They shall come into force on the date of their publication in the Official Gazette.

CHAPTER 1: CODE OF MEDICAL ETHICS

A. Declaration: Each applicant at the time of making an application for registration under the provisions of the Act shall be provided a copy of the declaration and shall submit a duly signed declaration. The applicant shall also certify that he/she had read and agree to abide by the same.

B. Duties and responsibilities of the physician in general:

1.1 Character of physician (Doctors with qualification of MBBS or MBBS with postgraduate degree/diploma or with equivalent qualification in any medical discipline):

1.1.1 A physician shall uphold the dignity and honour of his profession.

1.1.2 The prime object of the medical profession is to render service to humanity; reward or financial gain

Table 22.1 Differences Between Professional Negligence and Professional Misconduct

Professional negligence	Professional misconduct/infamous conduct
Concerned with the duties of a medical man towards patients.	Concerned with violation of 'codes' and 'ethics' of medical practice.
Involves dereliction of duty in treatment leading to damage/harm to the patient.	There may not be dereliction of duty and damage to the patient.
Charge is brought before the appropriate court/forum.	Charge is brought before the State Medical Council.
Doctor may be liable to pay compensation as in other civil cases or be punishable as per Indian Penal Code as in other criminal cases.	Doctor may be given 'warning' or his name may be erased from the register of State Medical Council either temporarily or permanently depending upon the gravity of misconduct.
Appealable in the higher courts.	Appealable to the Govt. of India whose decision taken in consultation with Medical Council of India will be final.

is a subordinate consideration. Whosoever chooses his profession, assumes the obligation to conduct himself in accordance with its ideas. A physician should be an upright man, instructed in the art of healings. He shall keep himself pure in character and be diligent in caring for the sick; he should be modest, sober, patience, prompt in discharging his duty without anxiety; conducting himself with propriety in his profession and in all the actions of his life.

1.1.3 No person other than a doctor having qualification recognised by Medical Council of India and registered with Medical Council of India/State Medical Council(s) is allowed to practice Modern System of Medicine or Surgery. A person obtaining qualification in any other system of Medicine is not allowed to practice Modern System of Medicine in any form.

1.2 Maintaining good medical practice:

1.2.1 The principal objective of the medical profession is to render service to humanity with full respect for the dignity of profession and man. Physicians should merit the confidence of patients entrusted to their care, rendering to each a full measure of service and devotion. Physicians should try continuously to improve medical knowledge and skills and should make available to their patients and colleagues the benefits of their professional attainments. The physician should practice methods of healing founded on scientific basis and should not associate professionally with anyone who violates this principle. The honoured ideals of the medical profession imply that the responsibilities of the physician extend not only to individuals but also to society.

1.2.2 Membership in Medical Society: For the advancement of his profession, a physician should affiliate with associations and societies of allopathic medical professions and involve actively in the functioning of such bodies.

1.2.3 A physician should participate in professional meeting as part of Continuing Medical Education Programmes for at least 30 hours every five years, organised by reputed professional academic bodies or any other authorised organisations. The compliance of this requirement shall be informed regularly to Medical Council of India or the State Medical Councils as the case may be.

1.3 Maintenance of medical records:

1.3.1 Every physician shall maintain the medical records pertaining to his/her indoor patients for a period of 3 years from the date of commencement of the treatment in a standard proforma laid down by the Medical Council of India.

1.3.2 If any request is made for medical records either by the patients/authorised attendant or legal authorities involved, the same may be duly acknowledged and documents shall be issued within the period of 72 hours.

1.3.3 A registered medical practitioner shall maintain a register of medical certificates giving full details of certificates issued. When issuing a medical certificate he/she shall always enter the identification marks of the patient and keep a copy of the certificate. He/she shall not omit to record the signature and/or thumb mark, address and at least one identification mark of the patient on the medical certificates or report.

1.3.4 Efforts shall be made to computerise medical records for quick retrieval.

1.4 Display of registration number:

1.4.1 Every physician shall display the registration number accorded to him by the State Medical Council/ Medical Council of India in his clinic and in all his prescriptions, certificates, money receipts given to his patients.

1.4.2 Physicians shall display as suffix to their names only recognised medical degrees or such certificates/ diplomas and memberships, honours that confer professional knowledge or recognises any exemplary qualification/achievements.

1.5 Use of generic names of drugs: Every physician should as far as possible, prescribe drugs with generic names, and he/she shall ensure that there is a rational prescription and use of drugs.

1.6 Highest quality assurance in patient care: Every physician should aid in safeguarding the profession against admission to it of those who are deficient in moral character or education. Physician shall not employ in connection with his professional practice any attendant who is neither registered nor enlisted under the Medical Acts in force and shall not permit such persons to attend, treat or perform operations upon patients wherever professional discretion or skill is required.

1.7 Exposure of unethical conduct: A physician should expose, without fear or favour, incompetent or corrupt, dishonest or unethical conduct on the part of members of the profession.

1.8 Payment of professional services: The physician engaged in the practice of medicine shall give priority to the interests of patients. The personal financial interests of a physician should not conflict with the medical interests of patients. A physician should announce his fees before rendering service and not after the operation or treatment is underway. Remuneration received for such services should be in the form and amount specifically announced to the patient at the time the service is rendered. It is unethical to enter into a contract of 'no cure no payment'. Physician rendering service on behalf of the state shall refrain from anticipating or accepting any consideration.

1.9 Evasion of legal restrictions: The physician shall observe the laws of the country in regulating the practice of medicine and shall also not assist others to evade such laws. He should be cooperative in observance and enforcement of sanitary laws and regulations in the interest of public health. A physician should observe the provisions of the State Acts like Drugs and Cosmetics Act, 1940; Pharmacy Act, 1948; Narcotic Drugs and Psychotropic Substances Act, 1985; Medical Termination of Pregnancy Act, 1971; Transplantation of Human Organ Act, 1994; Mental Health Act, 1987; Environmental Protection Act, 1986; Prenatal Sex Determination Test Act, 1994; Drugs and Magic Remedies (Objectionable Advertisement) Act, 1954; Persons with Disabilities (Equal Opportunities and Full Participation) Act, 1995; and Bio Medical Waste (Management and Handling) Rules, 1998 and such other Acts, Rules, Regulations made by the Central/State Governments or local Administrative Bodies or any other relevant Act relating to the protection and promotion of public health.

CHAPTER 2: DUTIES OF PHYSICIANS TO THEIR PATIENTS

2.1 Obligations to the sick:

2.1.1 Though a physician is not bound to treat each and every person asking his services, he should not only be always ready to respond to the calls of the sick and the injured, but should be mindful of the high character of his mission and the responsibility he discharges in the course of his professional duties. In his treatment, he should never forget that the health and the lives of those entrusted to his care depend on his skill and attention. A physician advising a patient to seek service of another physician is acceptable; however, in case of emergency, a physician must treat the patient. No physician shall arbitrarily refuse treatment to a patient. However, for good reason, when a patient is suffering from an ailment that is not within the range of experience of the treating physician, the physician may refuse treatment and refer the patient to another physician.

2.1.2 Medical practitioner having any incapacity detrimental to the patient or which can affect his performance vis-á-vis the patient is not permitted to practice his profession.

2.2 Patience, delicacy and secrecy: Patience and delicacy should characterise the physician. Confidence concerning individual or domestic life entrusted by patients to a physician and defects in the disposition or character of patients observed during medical attendance should never be revealed unless their revelation is required by the laws of the State. Sometimes, however, a physician must determine whether his duty to society requires him to employ knowledge, obtained through confidence as a physician, to protect a healthy person against a communicable disease to which he is about to be exposed. In such instance, the physician should act as he would wish another to act towards one of his own family in like circumstances.

2.3 Prognosis: The physician should neither exaggerate nor minimise the gravity of a patient's condition. He should ensure himself that the patient, his relatives or his responsible friends have such knowledge of the patient's condition as will serve the best interests of the patient and the family.

2.4 The patient must not be neglected: A physician is free to choose whom he will serve. He should, however, respond to any request for his assistance in an emergency. Once having undertaken a case, the physician should not neglect the patient, nor should he withdraw from the case without giving adequate notice to the patient and his family. Provisionally or fully registered medical practitioner shall not willfully commit an act of negligence that may deprive his patient or patients from necessary medical care.

2.5 Engagement for an obstetric case: When a physician who has been engaged to attend an obstetric case is absent and another is sent for and delivery accomplished, the acting physician is entitled to his professional fees, but should secure the patient's consent to resign on the arrival of the physician engaged.

CHAPTER 3: DUTIES OF PHYSICIAN IN CONSULTATION

3.1 Unnecessary consultations should be avoided:

3.1.1 However in case of serious illness and in doubtful or difficult conditions, the physician should request consultation, but under any circumstances such consultation should be justifiable and in the interest of the patient only and not for any other consideration.

3.1.2 Consulting pathologists/radiologists or asking for any other diagnostic laboratory investigation should be done judiciously and not in a routine manner.

3.2 Consultation for patient's benefit: In every consultation, the benefit to the patient is of foremost importance. All physicians engaged in the case should be frank with the patient and his attendants.

3.3 Punctuality in consultation: Utmost punctuality should be observed by a physician in making themselves available for consultations.

3.4 Statement to patient after consultation:

3.4.1 All statements to the patient or his representative should take place in the presence of the consulting physicians, except as otherwise agreed. The disclosure of the opinion to the patient or his relatives or friends shall rest with the medical attendant.

3.4.2 Differences of opinion should not be divulged unnecessarily but when there is irreconcilable difference

of opinion, the circumstances should be frankly and impartially explained to the patient or his relatives or friends. It would be opened to them to seek further advice as they so desire.

3.5 Treatment after consultation: No decision should restrain the attending physician from making such subsequent variations in the treatment if any unexpected change occurs, but at the next consultation, reasons for the variations should be discussed/explained. The same privilege, with its obligations, belongs to the consultant when sent for in an emergency during the absence of attending physician. The attending physician may prescribe medicine at any time for the patient, whereas the consultant may prescribe only in case of emergency or as an expert when called for.

3.6 Patients referred to specialists: When a patient is referred to a specialist by the attending physician, a case summary of the patient should be given to the specialist, who should communicate his opinion in writing to the attending physician.

3.7 Fees and other charges:

3.7.1 A physician shall clearly display his fees and other charges on the board of his chamber and/or the hospitals he is visiting. Prescription should also make clear if the physician himself dispensed any medicine.

3.7.2 A physician shall write his name and designation in full along with registration particulars in his prescription letter head.

Note: In Government hospital where the patient load is heavy, the name of the prescribing doctor must be written below his/her signature.

CHAPTER 4: RESPONSIBILITIES OF PHYSICIANS TO EACH OTHER

4.1 Dependence of physicians on each other: A physician should consider it as a pleasure and privilege to render gratuitous service to all physicians and their immediate family dependants.

4.2 Conduct in consultation: In consultations, no insincerity, rivalry or envy should be indulged in. All due respect should be observed towards the physician in charge of the case, and no statement or remark be made that would impair the confidence reposed in him. For this purpose, no discussion should be carried on in the presence of the patient or his representative.

4.3 Consultant not to take charge of the case: When a physician has been called for consultation, the Consultant should normally not take charge of the case, especially on the solicitations of the patient or friends. The consultant shall not criticise the referring physician. He/she shall discuss the diagnosis and treatment plan with the referring physician.

4.4 Appointment of substitute: Whenever a physician requests another physician to attend his patients during his temporary absence from his practice, professional courtesy requires the acceptance of such appointment only when he has the capacity to discharge the additional responsibility along with his/her other duties. The physician acting under such an appointment should give the utmost consideration to the interests and reputation of the absent physician, and all such patients should be restored to the care of the latter upon his/her return.

4.5 Visiting another physician's case: When it becomes the duty of a physician occupying an official position to see and report upon an illness or injury, he should communicate to the physician in attendance, so as to give him an option of being present. The medical officer/physician occupying an official position should avoid remarks upon the diagnosis or the treatment that has been adopted.

CHAPTER 5: DUTIES OF PHYSICIAN TO THE PUBLIC AND TO THE PARAMEDICAL PROFESSION

5.1 Physicians as citizens: Physicians, as good citizens, possessed of special training should disseminate advice on public health issues. They should play their part in enforcing the laws of the community and in sustaining the institutions that advance the interests of humanity. They should particularly cooperate with the authorities in the administration of sanitary/public health laws and regulations.

5.2 Public and community health: Physicians, especially those engaged in public health work, should enlighten the public concerning quarantine regulations and measures for the prevention of epidemic and communicable diseases. At all times, the physician should notify the constituted public health authorities of every case of communicable diseases under his care in accordance with the laws, rules and regulations of the health authorities. When an epidemic occurs, a physician should not abandon his duty for fear of contracting the disease himself.

5.3 Pharmacists/nurses: Physicians should recognise and promote the practice of different paramedical services such as pharmacy and nursing as professions and should seek their cooperation wherever required.

CHAPTER 6: UNETHICAL ACTS

A physician shall not aid or abet or commit any of the following acts that shall be construed as unethical.

6.1 Advertising:

6.1.1 Soliciting of patient directly or indirectly by a physician, by a group of physicians or by institutions or organisations is unethical. A physician shall not make use of his/her name as subject in any form or manner of advertising or publicity through any mode either alone or in conjunction with others which is of such a character as to invite attention to him or

to his professional position, skill, qualification, achievements, attainments, specialties, appointments, associations, affiliations or honours and/or such character as would ordinarily result in his self-aggrandisement. A physician shall not give to any person, whether for compensation or otherwise, any approval, recommendation, endorsement, certificate, report or statement with respect of any drug, medicine, nostrum remedy, surgical or therapeutic article, apparatus or appliance or any commercial product or article with respect of any property, quality or use thereof or any test, demonstration or trial thereof, for use in connection with his name, signature or photograph in any form or manner of advertising through any mode nor shall he boast of cases, operation, cures or remedies or permit the publication or report thereof through any mode. A medical practitioner is however permitted to make a formal announcement in press:

(i) on starting practice,
(ii) on change of type of practice,
(iii) on changing address,
(iv) on temporary absence from duty,
(v) on resumption of another practice,
(vi) on succeeding to another practice and
(vii) for public declaration of charges.

6.1.2 Printing of self photograph or any such material of publicity in the letter head or on signboard of the consulting room or any such clinical establishment shall be regarded as acts of self-advertisement and unethical conduct on the part of the physician. However, printing of sketches, diagrams, picture of human system shall not be treated as unethical.

6.2 Patent and copyrights: A physician may patent surgical instruments, appliances and medicine or copyright applications, methods and procedures. However, it shall be unethical if the benefits of such patents or copyrights are not made available in situations where the interest of large population is involved.

6.3 Running an open shop (dispensing of drugs and appliances by physicians): A physician should not run an open shop for sale of medicine for dispensing prescriptions prescribed by doctors other than himself or for sale of medical or surgical appliances. It is not unethical for a physician to prescribe or supply drugs, remedies or appliances as long as there is no exploitation of the patient. Drugs prescribed by a physician or brought from the market for a patient should explicitly state the proprietary formula as well as generic name of the drug.

6.4 Rebates and commission:

6.4.1 A physician shall not give, solicit or receive nor shall he offer to give, solicit or receive, any gift, gratuity, commission or bonus in consideration of or return for the referring, recommending or procuring of any patient for medical, surgical or other treatment. A physician shall not directly or indirectly participate in or be a party to act of division, transference, assignment, subordination, rebating, splitting or refunding of any fee for medical, surgical or other treatment.

6.4.2 Provisions of para 6.4.1 shall apply with equal force to the referring, recommending or procuring by a physician or any person, specimen or material for diagnostic purposes or other study/work. Nothing in this Section, however, shall prohibit payment of salaries by a qualified physician to other duly qualified person rendering medical care under his supervision.

6.5 Secret remedies: The prescribing or dispensing by a physician of secret remedial agents of which he does not know the composition or the manufacture or promotion of their use is unethical and as such prohibited. All the drugs prescribed by a physician should always carry a proprietary formula and clear name.

6.6 Human rights: The physician shall not aid or abet torture nor shall he be a party to either infliction of mental or physical trauma or concealment of torture inflicted by some other person or agency in clear violation of human rights.

6.7 Euthanasia: Practicing euthanasia shall constitute unethical conduct. However, on specific occasion the question of withdrawing supporting devices to sustain cardiopulmonary function even after brain death shall be decided only by a team of doctors and not merely by the treating physician alone. A team of doctors shall declare withdrawal of support system. Such team shall consist of the doctor in charge of the patient, Chief Medical Officer/Medical Officer in Charge of the hospital and a doctor nominated by the in charge of the hospital from the hospital staff or in accordance with the provisions of the Transplantation of Human Organ Act, 1994.

6.8 Code of conduct for doctor and professional association of doctors in their relationship with pharmaceutical and allied health sector industry.

6.8.1 In dealing with pharmaceutical and allied health sector industry, a medical practitioner shall follow and adhere to the stipulations given below:

(a) **Gifts:** A medical practitioner shall not receive any gift from any pharmaceutical or allied healthcare industry and their sales people or representatives.

(b) **Travel facilities:** A medical practitioner shall not accept any travel facility inside the country or outside, including rail, air, ship, cruise tickets, paid vacations, etc. from any pharmaceutical or allied healthcare industry or their representatives for self and family members for vacation or for attending conference, seminars, workshops, CME programme etc. as a delegate.

(c) **Hospitality:** A medical practitioner shall not accept individually any hospitality like hotel accommodation for self and family members under any pretext.

(d) **Cash or monetary grants:** A medical practitioner shall not receive any cash or monetary grants from any pharmaceutical and allied healthcare industry for individual purpose in individual capacity under any pretext. Funding for medical research, study etc. can only be received through approved institutions by modalities laid down by law/rules/guidelines adopted by such approved institutions, in a transparent manner. It shall always be fully disclosed.

(e) **Medical research:** A medical practitioner may carry out, participate in, work in research projects funded by pharmaceutical and allied healthcare industries. A medical practitioner is obliged to know that the fulfillment of the following items (1) to (vii) will be an imperative for undertaking any research assignment/project funded by industry for being proper and ethical. Thus, in accepting such a position a medical practitioner shall:
   (i) Ensure that the particular research proposal(s) has the due permission from the competent concerned authorities;
   (ii) Ensure that such a research project(s) has the clearance of national/state/institutional ethics committee/bodies;
   (iii) Ensure that it fulfills all the legal requirements prescribed for medical research;
   (iv) Ensure that the source and amount of funding is publicly disclosed at the beginning itself;
   (v) Ensure that proper care and facilities are provided to human volunteers, if they are necessary for the research projects;
   (vi) Ensure that undue animal experimentations are not done, and when these are necessary they are done in a scientific and a humane way;
   (vii) Ensure that while accepting such an assignment, a medical practitioner shall have the freedom to publish the results of the research in the greater interest of the society by inserting such a clause in the MoU or any other document/agreement for any such assignment.

(f) **Maintaining professional autonomy:** In dealing with pharmaceutical and allied healthcare industry a medical practitioner shall always ensure that there shall never be any compromise either with his/her own professional autonomy and/or with the autonomy and freedom of the medical institution.

(g) **Affiliation:** A medical practitioner may work for pharmaceutical and allied healthcare industries in advisory capacities, as consultants, as researchers, as treating doctors or in any other professional capacity. In doing so, a medical practitioner shall always:
   (i) Ensure that his professional integrity and freedom are maintained;
   (ii) Ensure that patients interest are not compromised in any way;
   (iii) Ensure that such affiliations are within the law;
   (iv) Ensure that such affiliations/employments are fully transparent and disclosed.

(h) **Endorsement:** A medical practitioner shall not endorse any drug or product of the industry publically. Any study conducted on the efficacy or otherwise of such products shall be presented to and/or through appropriate scientific bodies or published in appropriate scientific journals in a proper way.

CHAPTER 7: MISCONDUCT

The following acts of commission or omission on the part of a physician shall constitute professional misconduct rendering him/her liable for disciplinary action:

7.1 Violation of the regulations: If he/she commits any violation of these regulations.

7.2 If he/she does not maintain the medical records of his/her indoor patients for a period of 3 years as per regulation 1.3 and refuses to provide the same within 72 hours when the patient or his/her authorised representative makes a request for it as per the regulation 1.3.2.

7.3 If he/she does not display the registration number accorded to him/her by the State Medical Council or the Medical Council of India in his clinic, prescriptions and certificates, etc. issued by him or violates the provisions of regulation 1.4.2.

7.4 Adultery or improper conduct: Abuse of professional position by committing adultery or improper conduct with a patient or by maintaining an improper association with a patient will render a physician liable for disciplinary action as provided under the Indian Medical Council Act, 1956 or the concerned State Medical Council Act.

7.5 Conviction by Court of Law: Conviction by a Court of Law for offences involving moral turpitude/criminal acts.

7.6 Sex determination tests: On no account sex determination test shall be undertaken with the intent to terminate the life of a female foetus developing in her

mother's womb, unless there are other absolute indications for termination of pregnancy as specified in the Medical Termination of Pregnancy Act, 1971. Any act of termination of pregnancy of normal female foetus amounting to female foeticide shall be regarded as professional misconduct on the part of the physician leading to penal erasure besides rendering him liable to criminal proceeding as per the provisions of this Act.

7.7 Signing professional certificates, reports and other documents: Registered medical practitioners are in certain cases bound by law to give, or may from time to time be called upon or requested to give certificates, notification, reports and other documents of similar character signed by them in their professional capacity for subsequent use in the courts or for administrative purposes, etc. Any registered practitioner who is shown to have signed or given under his name and authority any such certificate, notification, report or document of a similar character that is untrue, misleading or improper is liable to have his name deleted from the Register.

7.8 A registered medical practitioner shall not contravene the provisions of the Drugs and Cosmetics Act and regulations made thereunder. Accordingly:

(a) Prescribing steroids/psychotropic drugs when there is no absolute medical indication;

(b) Selling schedule 'H' & 'L' drugs and poisons to the public except to his patient. Contravention of the above provisions shall constitute gross professional misconduct on the part of the physician.

7.9 Performing or enabling unqualified person to perform an abortion or any illegal operation for which there is no medical, surgical or psychological indication.

7.10 A registered medical practitioner shall not issue certificates of efficiency in modern medicine to unqualified or nonmedical persons.

(**Note:** The foregoing does not restrict the proper training and instruction of bona fide students, midwives, dispensers, surgical attendants, or skilled mechanical and technical assistants and therapy assistants under the personal supervision of physicians.)

7.11 A physician should not contribute to the lay press articles and give interviews regarding diseases and treatments that may have the effect of advertising himself or soliciting practices; but is open to write to the lay press under his own name on matters of public health, hygienic living or to deliver public lectures, give talks on the radio/TV/Internet chat for the same purpose and send announcement of the same to lay press.

7.12 An institution run by a physician for a particular purpose such as a maternity home, nursing home, private hospital, rehabilitation centre or any type of training institution, etc. may be advertised in the lay press, but such advertisements should not contain anything more than the name of the institution, type of patients admitted, type of training and other facilities offered and the fees.

7.13 It is improper for a physician to use an unusually large sign board and write on it anything other than his name, qualifications obtained from a University or a statutory body, titles and name of his specialty, registration number including the name of the State Medical Council under which registered. The same should be the contents of his prescription papers. It is improper to affix a sign board on a chemist's shop or in places where he does not reside or work.

7.14 The registered medical practitioner shall not disclose the secrets of a patient that have been learnt in the exercise of his/her profession except:

(i) in a court of law under orders of the Presiding judge;

(ii) in circumstances where there is a serious and identified risk to a specific person and/or community and

(iii) notifiable diseases.

In case of communicable/notifiable diseases, concerned public health authorities should be informed immediately.

7.15 The registered medical practitioner shall not refuse on religious grounds alone to give assistance in or conduct of sterility, birth control, circumcision and medical termination of pregnancy where there is medical indication, unless the medical practitioner feels himself/herself incompetent to do so.

7.16 Before performing an operation, the physician should obtain in writing the consent from the husband or wife, parent or guardian in the case of minor or the patient himself as the case may be. In an operation that may result in sterility, the consent of both husband and wife is needed.

7.17 A registered medical practitioner shall not publish photographs or case reports of his/her patients without their permission in any medical or other journal in a manner by which their identity could be made out. If the identity is not to be disclosed, the consent is not needed.

7.18 In the case of running of a nursing home by a physician and employing assistants to help him/her, the ultimate responsibility rests on the physician.

7.19 A physician shall not use touts or agents for procuring patients.

7.20 A physician shall not claim to be specialist unless he has a special qualification in that branch.

7.21 No act of in vitro fertilisation or artificial insemination shall be undertaken without the informed consent of the female patient and her spouse as well as the donor. Such consent shall be obtained in writing only after the patient is provided, at her own level of comprehension, with sufficient information about the purpose, methods, risks, inconveniences, disappointments of the procedure and possible risks and hazards.

7.22 **Research:** Clinical drug trials or other research involving patients or volunteers as per the guidelines of ICMR can be undertaken, provided ethical considerations are borne in mind. Violation of existing ICMR guidelines in this regard shall constitute misconduct. Consent taken from the patient for trial of drug or therapy which is not as per the guidelines shall also be construed as misconduct.

CHAPTER 8: PUNISHMENT AND DISCIPLINARY ACTION

8.1 It must be clearly understood that the instances of offences and of professional misconduct that are mentioned above do not constitute and are not intended to constitute a complete list of the infamous acts that calls for disciplinary action and that by issuing this notice the Medical Council of India and/or State Medical Councils are in no way precluded from considering and dealing with any other form of professional misconduct on the part of a registered practitioner. Circumstances may and do arise from time to time in relation to which there may occur questions of professional misconduct that do not come within any of these categories. Every care should be taken that the code is not violated in letter or spirit. In such instances as in all others, the Medical Council of India and/or State Medical Councils have to consider and decide upon the facts brought before the Medical Council of India and/or State Medical Councils.

8.2 It is made clear that any complaint with regard to professional misconduct can be brought before the appropriate Medical Council for disciplinary action. Upon receipt of any complaint of professional misconduct, the appropriate Medical Council will hold an inquiry and opportunity to the registered medical practitioner to be heard in person or by pleader. If the medical practitioner is found to be guilty of committing professional misconduct, the appropriate Medical Council may award such punishment as deemed necessary or may direct the removal altogether or for a specified period, from the register of the name of the delinquent registered practitioner. Deletion from the register shall be widely publicised in local press as well as in the publications of different Medical Associations/Societies/Bodies.

8.3 In case the punishment of removal from the register is for a limited period, the appropriate Council may also direct that the name so removed shall be restored in the register after the expiry of the period for which the name was ordered to be removed.

8.4 Decision on complaint against delinquent physician shall be taken within a time limit of 6 months.

8.5 During the pendency of the complaint, the appropriate Council may restrain the physician from performing the procedure or practice that is under scrutiny.

8.6 Professional incompetence shall be judged by 'peer group' as per guidelines prescribed by Medical Council of India.

8.7 Where either on a request or otherwise the Medical Council of India is informed that any complaint against a delinquent physician has not been decided by a State Medical Council within a period of 6 months from the date of receipt of complaint by it and further the MCI has reason to believe that there is no justified reason for not deciding the complaint within the said prescribed period, the Medical Council of India may:

(i) impress upon the concerned State Medical Council to conclude and decide the complaint within a time-bound schedule;

(ii) decide to withdraw the said complaint pending with the concerned State Medical Council straightaway or after the expiry of the period, which had been stipulated by the MCI in accordance with para (i) above, to itself and refer the same to the Ethical Committee of the Council for its expeditious disposal in a period of not more than 6 months from the receipt of the complaint in the office of the Medical Council of India.

8.8 Any person aggrieved by the decision of the State Medical Council on any complaint against a delinquent physician shall have the right to file an appeal to the MCI within a period of 60 days from the date of receipt of the order passed the said Medical Council. Provided that the MCI may, if it is satisfied that the appellant was prevented by sufficient cause from presenting the appeal within the aforesaid period of 60 days, allow it to be presented within a further period of 60 days.

Declaration

At the time of registration, each applicant shall be given a copy of the following declaration by the Registrar concerned and the applicant shall read and agree to abide by the same:

1. I solemnly pledge myself to devote my life to service of humanity.
2. Even under threat, I will not use my medical knowledge contrary to the laws of humanity.
3. I will maintain the utmost respect for human life from the time of conception.
4. I will not permit considerations of religion, nationality, race, party politics or social standing to intervene between my duty and my patient.
5. I will practice my profession with conscience and dignity.
6. The health of my patient will be my first consideration.
7. I will respect the secrets that are confided in me.
8. I will give to my teachers the respect and gratitude which is their due.
9. I will maintain by all means in my power, the honour and noble traditions of medical profession.
10. I will treat my colleagues with all respect and dignity.
11. I shall abide by the code of medical ethics as enunciated in the Indian Medical Council (Professional Conduct,

Etiquette and Ethics) Regulations 2002 including amendments effected from time to time.

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

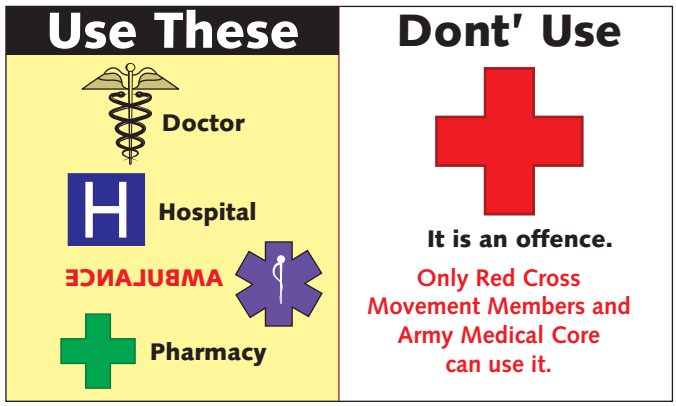
Signature :
Name :
Place :
Address :
Date :

Red Cross—Not For All

Geneva Convention (1949) regulates the use of the **Red Cross emblem**. India being signatory to the convention also adopted these emblems. The Indian Red Cross Society Act specifies that the Red Cross sign can only be used by Military Medical Services, their personal units, installations and means of transport and by International Committee of the Red Cross, etc. Abuse of emblem can lead to a fine of ₹ 500 besides the provision of forfeiture of the goods or vehicles on which the emblem has been used without authorisation.

Doctors need to use the symbol of a rod entwined by two snakes, decorated with wings on the top of the rod. **Hospitals** have a symbol with an 'H' written in white colour over a grey background. **Ambulances** have to use a symbol with a needle and thread inscribed in white over the central part of fanned out petals. **Medical stores** to use a symbol of 'green cross' over white background.

Red Cross Emblem Policy



Red Cross Emblem is used by those belonging to the Red Cross movement & Army medical services, who are involved in times of armed conflicts and natural disasters. It is not the emblem of Medical Profession and its use is regulated by International and National laws. After adoption at Geneva Convention 1949, the Red Cross Emblem has become a universally known & legally sanctified neutral sign to identify the people with protected status in the situation of armed conflicts.

23 CHAPTER

Medical Negligence

After going through this chapter, the reader will be able to describe: Negligence and its various components | Medical negligence—differentiation from negligence in other fields | Mistaken diagnosis/errors of clinical judgement whether amount to negligence | Defensive medicine | Proof of negligence with particular emphasis on the doctrine of 'res ipsa loquitur' | Contributory negligence | Vicarious liability | Medical product liability | Criminalisation of negligence | Failure to take X-ray—whether amounts to negligence | Consumer Protection Act and medical negligence

Negligence has two meanings in law of torts:

(1) Negligence as a mode of committing certain torts as, e.g., negligently or carelessly committing trespass, nuisance or defamation. In this context, it denotes the mental element.
(2) Negligence is considered as a separate tort. It means a conduct that creates a risk of causing damage, rather than a state of mind.

It is in the second sense that it has been discussed here. The term 'malpractice' refers to any professional misconduct that encompasses an unreasonable lack of skill in professional behaviour. The term 'medical negligence' is usually preferred to 'medical malpractice', which is charged with emotional baggage. **Negligence was defined by Baron Alderson in Blyth vs. Birmingham Waterworks Co** as 'the omission to do something that a reasonable man guided by those considerations that ordinarily regulate the conduct of human affairs, … would do, or doing something that a prudent and reasonable man would not do.'

It was suggested that medical negligence was an example of negligence at large, **but the more modern view is likely to be that expressed by Lord Denning in Hatcher vs. Black and others.** He declined to liken the case against a hospital to a motor car accident or an accident in a factory: 'On the road or in a factory there ought not to be any accidents if everyone used proper care. But in a hospital, when a person was ill and came in for treatment, no matter what care was used there was always a risk, and it would be wrong and bad law to say that simply because a mishap occurred the hospital and doctors were liable. Indeed it would be disastrous to the community. It would mean that a doctor examining a patient or a surgeon operating at the table, instead of getting on with his work, would be forever looking over his shoulder to see if someone was coming up with a dagger (i.e., an action for negligence). There are risks inherent in most forms of medical treatment. All that one can ask is that he should keep these risks to the minimum. If he has done this, no injury which occurs, however serious, is actionable.'

Elements of Negligence

All manner of interventions can be used in clinical work, some of which may be novel, strange or even bizarre, but none of them represents negligence unless the patient (plaintiff) can show the presence of following four elements by a preponderance of the evidence, i.e. they are more likely to be present than not. The four elements can be summarised as four Ds of negligence, namely: Duty, Dereliction, Direct causation and Damage.

DUTY

The concept of duty adapts itself to the changing circumstances even as the law itself, as stated by Lord Macmillan, "categories of negligence are never closed". It is, therefore, difficult to express it as a general proposition but, broadly, duty means a standard of behaviour imposing some restrictions on one's conduct. It implies a legal duty rather than a moral one. A duty is created where there is an offer to treat/care, i.e. **'therapeutic intent'** forms the key issue that is instrumental in establishing doctor–patient relationship. This relationship may be formed extremely easily and by no means depends upon any formal acceptance of a patient by a doctor. Even in an emergency, once a doctor approaches an ill or injured person with the intention of assisting him, then a valid duty of care is set up. This is notwithstanding the fact that the patient may be unconscious and

quite unaware of the doctor's presence. In the United States, some States brought in legislation to limit actions for negligence arising out of casual treatment at the scene of accidents, as the number of such actions had become so frequent that the doctors became extremely reluctant to render aid in emergencies.

It is important to appreciate that the negligent advice is just as actionable as negligent treatment. It must be something more than casual advice; there must be some evidence of assumption of responsibility, e.g. the payment of fee. Where a doctor examines a patient for some purpose other than providing advice and treatment, no doctor–patient relationship is established and thus no duty of care exists. A doctor conducting a medicolegal examination for any purpose (such as insurance, determination of disability, etc.) is not there in his capacity as a 'healer' and no duty of care arises. In such circumstances, there is obviously a duty not to inflict any damage upon the patient. If the doctor happens to inflict an injury, say, while attempting to draw a blood sample in such cases, the patient has a right of action. In such cases, a duty, however, exists between the doctor and the authority employing him to provide an accurate report and any incompetence, in this context, would be a breach of contract, not a tort.

Duty Depends Upon Reasonable Foreseeability of Injury

Whether the defendant owes a duty to the plaintiff or not depends upon reasonable foreseeability of injury to the plaintiff. To decide culpability, we have to determine what a reasonable man would have foreseen and thus form an idea of how he would have behaved under the circumstances. Explaining the standard of foresight of the reasonable man **Lord Macmillan observed**:

'The standard of foresight of the reasonable man is, in one sense, an impersonal test. It eliminates the personal equation and is independent of the idiosyncrasies of the particular person whose conduct is in question. Some persons are by nature unduly timorous and imagine every path beset with lions. Others, of more robust temperament, fail to foresee or nonchalantly disregard even the most obvious dangers. The reasonable man is presumed to be free both from over-apprehension and from over-confidence. But it is still left to the judge to decide what, in the circumstances of a particular case, the reasonable man would have had in contemplation, and what, accordingly, the party sought to be made liable, ought to have foreseen'. Here there is room for diversity of views. What to one judge may seem far fetched may seem to another both natural and probable.

In emergency situations (accidents, disasters, etc.) where healthcare professionals attend victims at the place of occurrence, they have to work in extremely adverse and hazardous conditions. Standard of care expected under such circumstances may not be that as expected otherwise. Further, under such situations, prioritisation of the victims may be done on the concept of 'necessity'. The position was pointed out by Mason and McCall Smith (1999), "A doctor working in an emergency, with inadequate facilities and under great pressure, will not be expected by the courts to achieve the same results as a doctor who is working in ideal conditions". This was alluded to by Mustill J in Wilsher, where he said, "If a person was forced by an emergency to do too many things at once, then the fact that he does one of them incorrectly 'should not be taken lightly as negligence'." **Further**, in such situations (and otherwise too, in the present scenario), the victim/patient is usually treated by a team of healthcare professionals to a varying degree. Though the need for inexperienced doctors (interns, house surgeons, etc.) to learn through training and experience is well-recognised, yet junior doctors can meet the standard of care by seeking help when they feel that things are becoming overly complicated. An action in negligence will obviously succeed if help is not sought and a deficient standard of care is provided.

DERELICTION

Negligence belongs to the part of civil law known as 'negligent torts' (a tort is a civil wrong) that may be styled as **'sins of omission'**, i.e. the plaintiff alleges that the doctor neglected to do something or left something out, which is ordinarily a part of the proper care. This points out to the central issue of 'standard of care' required to prove negligence. But how is that standard established and how does the court become aware of it? **The first question** is usually answered by the case laws (previous judgements of the courts) or the law on the books. Such a standard is usually set by the 'peer view', i.e. what a 'substantial minority' of respectable doctors skilled in the same speciality would have done under similar circumstances if they had been in the place of the defendant at that time. **The latter question**, i.e. how the judges (medical laypersons) are supposed to know what constitutes average practice, especially in terms of technical aspects of clinical work. This invites the role of 'expert witness' who is called by the court to help the jury to sort out the issue of liability based upon reasoning, background and scientific debate.

A much hyped Heart Care Hospital came under a sharp focus with the Consumer Disputes Redressal Forum holding the hospital guilty of unfair trade practices along with negligence. The forum observed, "There is no document to suggest whether the complainant or his family members were told by the hospital that angioplasty on other two vessels, which were blocked was not conducted. There are two consent letters but in none of these letters this fact is mentioned. It appears that the fact regarding the blockage of other two vessels of the patient was being concealed from him and his family. It must have been concealed so that the patient does not come to know that a part of the job has been completed and treatment of other two vessels is to be done for which the patient may come again after 4–6 months." The hospital was told to pay ₹ 2 lakh as compensation and was also directed to refund ₹ 2.27 lakh charged as fee.

MISTAKEN DIAGNOSIS

An erroneous diagnosis is negligent only if it implies an absence of due care or skill. Failure to adopt generally approved methods of diagnosis or treatment in the appropriate circumstances is a perilous course. Thus, the omission of X-ray examination in circumstances where a fracture or dislocation was suspected or ought to have been suspected may be sufficient to establish lack of reasonable care.

However, it should be noted that as the medical practice evolves, the criteria of 'accepted practice' changes. For example, it is no longer felt obligatory to go in for X-ray of the skull in cases of head injuries, both on the grounds of unnecessary expense, harmful radiation and doubtful efficacy. It is clinical and especially the neurological examination that is more important. Usually, the regimen of treatment would be the same whether or not the X-ray reveals a fracture. The practice of invariably X-raying the cases of head injuries has, probably, arisen from fears of medicolegal consequences rather than from clinical necessity. Notwithstanding all this, the radiological examination of a patient following any sort of traumatic injury has become so much of a routine that it is likely that there would be a danger of a finding of negligence if no radiological examination was sought, particularly when the history was suggestive of possibility of some fracture or dislocation or presence of a foreign body in a wound.

A doctor may find himself/herself negligent if a wrong solution has been injected by mistake or oversight. He may inject the fluid into an artery instead of vein or use solution intended to be used as intramuscular as intravenous. **In a case**, a nurse misread the instructions of the doctor and gave the child an intravenous injection of chloroquine instead of chloromycetine, which resulted in cardiac arrest. Delay in resuscitation left the child in a vegetative state and compensation to the tune of ₹ 12.5 lakhs was awarded to the minor child taking into account the cost of equipments and the recurring expenses for the rest of the life of the minor child and ₹ 5 lakhs to the parents of the minor child for the mental agony and the life-long care and attention that they have to bestow on the minor child [Harjot Ahluwalia (minor) through parents vs. M/s Spring Meadows Hospital and others].

DIRECT CAUSATION

The third element in liability requires that the damage, alleged to have resulted, should bear a direct 'cause and effect' relationship to the negligence of the doctor. A common concomitant of the causation factor is the legal concept of the 'but-for' test, i.e. 'But for that negligence, would the harm have occurred?' In that determination, causal contributions by the plaintiff's actions or failure to act, pre-existing or adventitious conditions and possible opportunities for follow-up remedial action, etc. may be explored. A typical example may be a case where there occurs an intestinal perforation resulting from abscess formation due to some retained instrument by a surgeon in the abdominal cavity. But for that instrument, such complication would not have occurred. In contrast, a doctor's delay in the diagnosis of a highly aggressive malignant neoplasm might not necessarily affect the patient's chance of survival.

DAMAGE SUFFERED BY THE PATIENT

This is an injury or disability suffered by the patient and must be distinguished from 'damages' which are the financial compensations awarded to the successful plaintiff. The purpose of awarding damages in a tort action is to ensure that a person who has suffered damage (injury/harm) is made 'whole' or is returned to the previous condition that existed before the injury. Because it is generally not possible to alleviate the effects of an injury resulting from medical negligence, public policy necessitates redressal through financial compensation to the plaintiff. Two types of compensatory damages have generally been recognised: **General damages** being awarded for non-economic losses like pain, sufferings, mental anguish, grief and other related complaints. **Special damages** include past and future medical, surgical, hospital and other related costs, past and future loss of income, expenses for the cremation in case of death and unusual consequences of the alleged injury such as where there is aggravation of a pre-existing state of disease. **Punitive or exemplary damages** may occasionally be awarded in addition to those that are intended to set an example for the future or to punish the egregious behaviour of the defendant.

Unfortunately, relatives/attendants of the deceased often equate the duty of care with that of 'recovery' or 'successful outcome' in the case (especially in surgery). In such like cases, conduction of autopsy by a board of doctors (including expert from the concerned specialty) **is an ideal procedure**. Photographic and videographic recording of the procedure may be carried out where felt necessary. Additional investigations including histopathological and toxicological studies need to be considered as a part of work-up. **Expressing opinion** invites caution and the same should be logical and appropriately worded. The autopsy surgeon carries ultimate responsibility to determine the cause of death and therefore, must not accept dogmatic or unsubstantiated findings/answers from the consultant(s). However, he/she must always remain open to healthy discussion in considering/discussing a range of possibilities. It needs be remembered that it is neither the job nor a correct practice to summarily comment upon the aspect of negligence in the postmortem report or in some other report because pronouncing the aspect of negligence is in the purview of the Honourable Court.

Proof of Negligence

The standard of proof in civil actions, which include virtually all cases of medical negligence, is the 'balance of probabilities'. This is unlike the burden of proof in criminal trials where the jury must be satisfied of the guilt, 'beyond reasonable doubt'.

Ordinarily, the task of proving negligence rests upon the plaintiff and it follows therefore that in medical cases it is for the patient or his relatives to establish his claim and not for the doctor to show that he acted with due care and skill. There are, however, certain cases, when the plaintiff need not prove that and the inference of presumption of negligence is drawn from the facts of the case, according to the maxim, **'res ipsa loquitur'**. An example of the application of maxim was in the case of Cassidy vs. Ministry of Health. The plaintiff was operated upon for a Dupuytren contracture of the third and fourth fingers of his left hand. After the operation the patient's left hand and forearm were bandaged to a splint, which was kept in place for 14 days.

During this period, he complained of pain in his hand but apart from the administration of sedatives no other action was taken. When the bandage was removed, it was discovered that all his four fingers of the hand were stiff and that the hand was to all intents and purposes useless. The Ministry denied negligence and liability for the surgeon under whose care the patient had been admitted. In the court of first instance, judgement was given for the Ministry on the ground that the patient had failed to establish negligence on the part of the surgeon or of any other member of the hospital staff. The patient appealed. The Court of Appeal held that the mere proof of the facts would cause a reasonable layman to draw the inference that the injury could have been caused only by want of care on the part of the hospital staff and that it was sufficient to call for an explanation from the defendant. The appeal was successful and the plaintiff was awarded damages.

Essentials of 'Res Ipsa Loquitur'

(i) Nature of injury or damage, suggested by common knowledge or inferred from expert evidence, that without negligence, it does not occur.
(ii) The defendant must be in exclusive control of the instrumentation or circumstances. The error in such cases is so obvious that the defendant has to establish his innocence.
(iii) The plaintiff must not contribute to his own injury or damage.

REBUTTAL OF PRESUMPTION OF NEGLIGENCE

The rule of *res ipsa loquitur* only shifts the burden of proof and instead of the plaintiff proving negligence on the part of the defendant, the defendant is required to disprove it. If the defendant is able to prove that what apparently seems to be negligence was due to some factors beyond the control of the defendant, he can escape liability. To rebut the presumption of negligence, it has also got to be proved that to ward off the evil consequences of such events, necessary preventive measures had been adopted.

The maxim of *res ipsa loquitur* applies when the only inference from the facts is that the damage could not have occurred but for the defendant's negligence. When the damage incurred is capable of two or more explanations, such a presumption may not be applicable.

SOME INSTANCES OF 'RES IPSA LOQUITUR'

Retention of Swabs, Packs, Instruments, Drains, etc.

Leaving a foreign body during an operation is the most deplorable accident. Many surgeons in conservation will reveal having removed foreign bodies, but seldom report the same. Such an accident of leaving a foreign body in the operation area may be seen in any area irrespective of specialty. Less than 10% have been reported to have been left to the upper abdomen where gallbladder surgery predominates and the gynaecologists appear to be the greatest offenders. The coils of intestines carry the notorious habit of crowding into the operative field thereby leading to certain unexpected situations. Under such circumstances, attention may be directed towards saving life while ignoring the less essential. Admittedly, the present day operation is an organised team effort, and the mishap of leaving a foreign body during surgery may have origin in any source, ultimately affecting the surgeon's reputation.

It is logical to think that surgeon and his assistant(s) are burdened with so much technical responsibility that it is not possible for them to give individual attention to sponges and instruments. This may be best left to the scrub nurse who may also be assisted by the floating member of the operating room staff. It is, however, essential that the surgeon and his assistant(s) must also confirm that nothing is left inside the abdomen by visually and manually examining the interior, particularly the recesses.

In cases, **where the loss of sponge or any other material/instrument is detected** while the patient is still in the operation theatre or in the adjoining recovery room, it may be safer to reopen under the same anaesthesia. It becomes simply an extension of the operative work necessitated by the complication. However, if the patient has come out of the anaesthesia, no further step may be taken without fresh consent and information to his relatives. Here, the troubled surgeon must act in the best interests of the patient thinking that it would decidedly be to his advantage.

Courts have stressed that the surgeon must himself determine that no sponge/instrument has been left behind. Frequently, the surgeon, because of the critical condition of the patient, is unable to make proper search without endangering the life of the patient. Under such situations the courts generally feel that the surgeon should take/show due care in retrieving all the sponges or instruments before he/she asks the nurse for the count. **Defences against the charge of negligence may include:**

- Difference in type of sponge found and the sponge used in the operation. It is for this reason that the law demands its preservation under seal and presentation to the court.
- Possibility of sponge having been left in another operation.
- Absence of causal relationship between leaving the sponge and the injury.
- In abortion cases, the patient may conceal the information of earlier attempts at abortion and may blame the surgeon

for a retained foreign body after attempt at abortion, if she happens to have had an abdominal operation before.

Operation on the Wrong Patient or on the Wrong Part of a Patient

The range of possible mistakes is wide, ranging from an operation on the wrong digit to an operation on the wrong patient, sometimes on the wrong side or on the wrong limb.

Administration of the Wrong Substance

The allegation that a wrong substance has been administered is normally unanswerable, but it is not always easy to determine the responsibility. A recurring error may be the administration of a substance of a right kind but in the wrong strength. Such cases also stress that it is not enough for the doctor to rely on the nursing staff. The doctor should preferably see the bottle or ampoule from which the solution has been taken and also verify the label. In the event that some dispensing error might have been there, it is imperative that the remaining solution and the stock bottle or ampoule are set aside for appropriate tests.

Contributory Negligence

If the plaintiff by his own want of care contributes to the damage caused by the negligent or wrongful act of the defendant, he is considered to be guilty of contributory negligence.

This is a defence in which the defendant has to prove that the plaintiff failed to take reasonable care of his own safety and that was a contributory factor to the harm ultimately suffered by the plaintiff. If A, going on the wrong side of the road, is hit by a vehicle coming from the opposite side and driven rashly by B, A can be met with the defence of contributory negligence on his part. In the medical practice, it may occur when the patient tampers with his dressings and induces infection or removes a plaster cast or bandage or more commonly ignores instructions to return for further treatment or follow-up, etc.

Vicarious Liability

Physicians usually employ or supervise other less qualified health team members. They, therefore, owe their patients the duty in assigning supervision to the nurses or subordinate staff properly. The duty may create vicarious liability, whereby one person may be liable for the wrongful acts or omissions of another. There are several legal doctrines in this context.

The simplest such doctrine is known as **'respondeat superior'** (let the master answer) and states that an employer is liable for the negligence of his or her employees. For example, if a doctor's office nurse injects a drug into a patient's sciatic nerve, causing injury, that patient may sue the physician for the nurse's negligence. Similarly, the interns, house surgeons/residents, etc. who work under the guidance of superiors will not be answerable to any negligent act committed by them during training. Further, their liability may also be vicariously imputed to the hospital as an employer. **In the words of Lord Denning,** "Whenever they accept a patient for treatment, they must use reasonable care and skill to cure him of his ailment. The hospital authorities cannot, of course, do it themselves; they have no ears to listen through a stethoscope and no hands to hold the surgeon's knife. They must do it through the staff, which they employ. And if their staff is negligent in giving treatment they are just as liable for that negligence as is anyone else who employs others to do his duties for him … once they undertake the task, they come under a duty to use care in the doing of it and that is so whether they do it for reward or not."

Doctors may also be vicariously liable under other legal doctrine for the negligence of hospital employees they supervise. For example, surgeons have been sued for errors and omissions by operating room personnel, under the **'captain of the ship'** doctrine. This doctrine holds a surgeon liable based on the legal action that he or she exercises absolute control much like the captain of a ship at sea who is responsible for all the wrong perpetrated by the crew. The captain of the ship doctrine has been largely replaced by the **'borrowed servant'** doctrine. Under this doctrine, a surgeon may be held responsible for the negligence of a nurse or any subordinate doctor, committed under direct control and supervision of the surgeon. Further, an employee may serve more than one employer. In such cases, the lending employer temporarily surrenders control over his worker and the borrowing employer temporarily takes over the control and thereby becomes responsible for all the acts committed under his or her direct supervision and control. Both the employer and the employee may be sued by the patient because the employee alone may be financially not so sound as to pay the entire damages. Usually, liability is fixed upon those who are actually at fault and those whose control over the negligent worker/employee is demonstrable.

Liability for Injury to the Third Parties

All doctors have a duty to warn patients about the aspects of their medical condition and/or treatment that could injure/harm others, e.g. the doctor of a epileptic patient may be liable for injury to the non-patient if the injury has indirectly been caused by the negligent treatment or failure to advise the patient of the risks of engaging in dangerous activities. Although there may not exist doctor–patient relationship with the third party victim, yet the doctor may be held liable for ordinary negligence under the concept of **'reasonable foreseeability'**, i.e. the injury to the non-patient was a foreseeable consequence of the patient's condition, which imposed on the doctor a duty to avoid injury to the foreseeable victims.

Medical Product Liability

Pharmaceuticals may be treated differently from other manufactured products. This different treatment may be partly due to the factor involving interaction that occurs between the body of the patient and the chemical compound of the drug. Frequently, the response of the drug depends upon the individual's physiology than on the product design.

The concept of strict liability eliminates the need to prove negligence for an injury caused by a defective product. Some commentators consider that the rules of strict liability should be applied to the drugs, while others think that a limited form of strict liability with less stringent rules be applied to drugs. Still others do not differentiate between drugs and other manufactured products. Multiple policy considerations govern the rules for applicability of strict liability in torts, namely: compensation or apportioning of the loss amongst all the consumers of the product, deterrence, encouraging useful conduct by both the parties to an action, protecting consumer expectations and improving the allocation of resources, etc. Injury or death of the patient may result from unexpected by-product of faulty, defective or negligently designed medical/surgical instrument or inadequate instructions. The manufacturer becomes responsible provided it is shown that he departed from the usual standards of care and skill with respect to design, assemblage, package, failure to test and inspect for defects or failure to warn. If the instrument functioned satisfactorily in previous operations or for several previous years in the hospital's possession, it is a proof that it was not defective at the time of supplying. Later, if the instrument develops a defect through ordinary and gradual wear and tear, or if the physician or the hospital misused the manufacturer's medical products, the hospital or physician owner is liable for the failure to inspect, test and get such defects repaired.

In the development of concept of defectiveness of a drug, the most appropriate approach seems to be the **'risk/utility' test**. This may be taken as a balancing test between the risk of danger associated with a product and the utility of the product to the consumer. The emphasis here is on the safety of the product rather than on the reasonable or unreasonable action of the manufacturer. The supplier of any product, including the manufacturer, is under a duty to use reasonable care **to warn adequately** about the risks associated with the use of its product(s). This duty extends to the risks about which the manufacturer knows and those about which, through reasonable care, should have known. However, a manufacturer is not responsible for unforeseeable or unknown dangers he is unable to discover with reasonable care. Drug manufacturer's duty to warn includes a warning to the physicians of the risks that are likely to ensue in the normal use. The doctor may be considered as the **'learned intermediate person'** and as such, the duty of the manufacturer to warn ends here in most of the cases and it is then the duty of the doctor to warn the patient. However, in cases where the manufacturer knows that the product will reach the public without individualised medical intervention, the drug manufacturer is duty-bound to warn the public at large. For example, in the case of immunisations where the individual is given the standardised dose of the vaccine, birth-control pills and certain drugs of common use with ample advertisements on the television. Adequacy of warning may also be material in determining the liability. **Adequacy of warning** is achieved, if it is obviously displayed giving fair appraisal of the extent of the danger and properly instructing about the manner of use of the product.

Medical/sales representatives occupy a position different from those of other salespersons. Their potential misrepresentation of the product may lead to cause harm to the ultimate consumer. These persons, acting as a link between the manufacturer and the doctor, are the most common transmitters of the information concerning the pharmaceuticals. They are frequently torn between a desire to increase the sale and a duty to inform the doctor about the possible side effects and contraindications of the drug. Even though the oral communications of these representatives are difficult to monitor, the manufacturer and the drug companies may be held liable for improper over-promotion of safety by representatives.

The manufacturer, the seller or anyone in the chain of sales may be sued by the buyer or any other ultimate user of the product who has suffered injury/harm/damage through the use of the product. If the injury/harm/damage was related in someway to the warning, evidence will be required as to the physical and chemical properties/qualities of the drug necessitating displaying of adequate warning.

Criminalisation of Negligence

Negligence in civil and criminal context carries some distinguishing features. There can be no civil action for negligence if the negligent act or omission has not been attended by an injury to any person, whereas bare negligence involving the risk of injury is punishable criminally though nobody is actually hurt by it. Further, in a civil action, the injured party has an option to sue specific person or those falling in the chain of events, whereas in an action for criminal liability, every person is responsible for his own act, there must be some personal act. And above all, in determining liability in civil cases, the outcome rests upon balance of probabilities, i.e. was it more likely than not that the condition was caused by negligence rather than some complication of disease, whereas in determining criminal liability, the standard of proof is 'beyond reasonable doubt' (Table 23.1). Thus, in Kay's Tutor vs. Ayrshire and Arran Health Board (1987), a young boy suspected to be suffering from meningitis was given a massive dose of penicillin and the boy was subsequently found to be profoundly deaf. His parents struggled for compensation through all levels up to the House of Lords. However, it was refused on the grounds that the child's deafness could also have been caused by meningitis

Table 23.1 Differentiating Features between Civil and Criminal Negligence

Civil negligence	Criminal negligence
Lack of reasonable care and skill in the professional behaviour.	Gross carelessness and scant regard for patient's welfare.
A dispute between two parties in their individual capacity.	A case between the State and the accused doctor.
The injured party has an option to sue specific person or those falling in the chain of events.	Every person is responsible for his own act. And, there must be some personal act.
There can be no civil action for negligence if the negligent act or omission has not been attended by an injury/harm/damage to the patient.	Bare negligence involving the risk of injury is punishable criminally, though nobody is actually hurt.
Contributory negligence can be cited as a defence.	Contributory negligence does not constitute defence.
The standard of proof rests upon the balance of probabilities, i.e. was it more likely than not that the condition/damage/harm was caused by negligence rather than by some complication.	The standard of proof requires establishment of guilt 'beyond reasonable doubt'.
Accused doctor is liable to pay damages.	Accused doctor is punishable with imprisonment or fine or both as per provisions of the IPC.

Note: Certain patterns of conduct may be more likely considered to equate a culpable state of mind with criminal negligence. Examples may include (*i*) disregarding past experience (i.e., the defendant doctor had sufficient knowledge of the problem based on his previous experience to have known that the problem would cause danger, but the doctor ignored the danger); (*ii*) failing to limit harm/damage in a timely manner (i.e., despite occurrence of initial negligence, exercising/not exercising due steps to limit the harm becomes the decisive issue) and (*iii*) appearance of improper motive (i.e., practicing in defiance of restrictions, or practicing in a manner that suggests more interest in financial gains than patient's well-being).

he was having and the parents failed to prove on the balance of probabilities that the overdose was the more likely cause.

Consumer Protection Act and Medical Negligence

Headlines proclaiming that a historic judgement of the Supreme Court permits doctors to be sued for medical negligence led to hysterical outbursts from doctors. The apprehensions that it will lead to huge increase in the medical expenditure on account of the insurance charges as well as tremendous increase in the defensive medicine and that there will be no safeguards against frivolous and vexatious complaints are largely unfounded. An analytical study of tort litigation in India during the period from 1975 to 1985 made by Professor Galanter reveals that a total number of 416 tort cases were decided by High Courts and Supreme Court, as reported in *All India Reporter*; out of which, 360 cases were related to 'Motor Vehicles Act', and cases related to medical practice were only three in number, because people in India are less litigation oriented as compared to those in England or USA.

The judgement has neither invented liability of doctors nor modified the notion. It has simply laid down that doctors provide a service and are accountable under the Act. It has provided a speedier and inexpensive mode of adjudication through the 'Consumer Dispute Redressal Fora' (Table 23.2). On balance, however, patients should be net gainers, as most people will happily pay a bit more for steps that may save their lives or limbs. The law, probably, does not want to permit the medical profession to play demi-God but wants to instill some fear in the minds of cavalier doctors.

The Apex Court in its latest judgement (Martin F D'Souza vs. Mohd Ishfaq; decided on 17.02.2009) has again emphasised that the guidelines conveyed in Jacob Mathew case be observed strictly and warned the police officials not to arrest or harass doctors unless there is a prima facie case of medical negligence. A couple of excerpts from the judgement conveying significant breather to the medical profession are being penned down:

- We, therefore, direct that whenever a complaint is received against a doctor or hospital by Consumer Fora (whether District, State or National) or by the Criminal Court then before issuing notice to the doctor or hospital against whom the complaint was made, the Consumer Forum or Criminal Court should first refer the matter to a competent doctor or committee of doctors, specialised in the field relating to which the medical negligence is attributed, and only after that doctor or committee reports that there is a prima facie case of medical negligence notice should be issued to the concerned doctor/hospital. This is necessary to avoid harassment to doctors who may not be ultimately found to be negligent.
- The Courts and Consumer Fora are not experts in medical science, and must not substitute their own views over that of specialists. It is true that the medical profession has, to an extent, become commercialised and there are many doctors who depart from their Hippocratic Oath for their selfish ends of making money. However, the entire medical fraternity cannot be blamed or branded as lacking in integrity or competence just because of some bad apples.

Table 23.2 Constitution and Jurisdiction of Consumer Dispute Redressal Fora

	District forum	State commission	National commission
President	A person who is or who has been or is qualified to be a District Judge to be appointed by the State Government	A person who is or has been a Judge of a High Court to be appointed by the State Government after consultation with the Chief Justice of the High Court	A person who is or has been a Judge of the Supreme Court to be appointed by Central Government after consultation with the Chief Justice of India
Members	Two other members (one of whom shall be a woman)	Not less than two and not more than such number of members as may be prescribed (one of whom shall be a woman)	Not less than four and not more than such number of members as may be prescribed (one of whom shall be a woman)
Duration	Every member of the District Forum shall hold office for a term of 5 years or up to the age of 65 years, whichever is earlier. However, a member shall also be eligible for re-appointment for another term of 5 years or up to the age of 65 years, whichever is earlier	Every member of the State Commission shall hold office for a term of 5 years or up to the age of 67 years, whichever is earlier. However, a member shall also be eligible for re-appointment for another term of 5 years or up to the age of 67 years, whichever is earlier	Every member of the National Commission shall hold office for a term of 5 years or up to the age of 70 years, whichever is earlier. However, a member shall also be eligible for re-appointment for another term of 5 years or up to the age of 70 years, whichever is earlier
Jurisdiction	To entertain complaints where the value of the goods or services and the compensation claimed, if any, **does not exceed ₹ 20 lakh**	To entertain complaints where the value of the goods or services and the compensation claimed, if any, exceeds ₹ 20 lakh but **does not exceed ₹ 1 crore**. Appeals against the orders of the District Forum	To entertain complaints where the value of the goods or services and the compensation claimed, if any, **exceeds ₹ 1 crore**. Appeals against the orders of any State Commission
General qualifications for the members	Not to be less than 35 years of age. Possession of bachelor's degree from a recognised university. They should be persons of ability, integrity and standing, and have adequate knowledge plus experience of at least 10 years in dealing with problems relating to economics, law, commerce, accountancy, industry, public affairs or administration.		
Penalties	Any trader, or a person complained against, or the complainant who fails or omits to comply with any order made by the District Forum or State/National Commission shall be punishable with imprisonment for a term that shall not be less than 1 month but may extend to 3 years, or with fine that shall not be less than ₹ 2000 but that may extend to ₹ 10,000, or with both.		
Limitation period under the Act	Section 24A of the Act prescribes a period of 2 years within which the complaint is to be filed. The period is counted from the date on which the cause of action has arisen. The same Section further provides that where the complainant shows sufficient cause for not filing the complaint within such period, the Commission/Forum can allow relaxation in filing the complaint after recording reasons for condoning such delay.		

However, in V Krishan Rao vs. Nikhil Superspeciality Hospital and Another (decided on 08.03.2010), the Apex Court clarifying its general directions communicated in the Martin F D'Souza vs. Mohd. Ishfaq judgment observed as such: "before forming an opinion that expert evidence is necessary, the Fora under the Act must come to a conclusion that the case is complicated enough to require the opinion of an expert or that the facts of the case are such that these cannot be resolved by the members of the Fora without the assistance of expert opinion. This Court makes it clear that in these matters no mechanical approach can be followed by these Fora. Each case has to be judged on its own facts. If a decision is taken that in all cases medical negligence has to be proved on the basis of expert evidence, in that event the efficacy of the remedy provided under this Act will be unnecessarily burdened and in many cases such remedy would be illusory."

Failure to Take X-ray—Whether Amounts to Negligence

Although radiologists are not in the same high-risk category of medical negligence and malpractice as surgeons, anaesthetists and physicians; they are by no means immune from such allegations, whether ill founded or otherwise. It is understandable that the

radiologist is rarely the primary medical attendant and therefore, being joined with the clinicians as co-defendant, the so-called due to his/her vicarious role. In some nonmedicolegal situations like impaction of foreign body in children, mentally defectives or individuals suffering from perversions, though the essential radiological aspects are concerned with the treatment and prognosis, they simultaneously become of considerable evidential value in matters of compensation and sometimes, even proceeding to civil litigation for damages for negligence against the employer/manager (in case the happening occurred during the course of an occupation/employment or during the hospital stay, etc.).

The first alleged malpractice suit for failure to take a radiograph in the evaluation of trauma followed hard upon the heels of Roentgen's original discoveries of X-rays. It occurred in Denver in April, 1896, only 4 months after the discovery of X-rays. The case was against Dr. WW Grant, a prominent Denver physician, for clinically missing a fracture of the hip of Mr. Smith. Smith's attorneys approached HH Buckwalter, a photojournalist, who helped in taking X-rays of Smith's hip. It took 4 days for preparing and developing radiographs and same were admitted to the court after a lengthy argument by the defendant's attorney (who was asserting that the fact seen by the direct witness, i.e. absence of fracture as observed through clinical assessment by Dr. Grant, was direct testimony and therefore, carried better evidentiary value). Two trials were held, and both ended in a **hung jury**. Finally, with the financial aid of some of Dr. Grant's friends, a settlement was made and the litigation ended.

In a case, the author appeared as an expert witness before the 'State Commission for the Redress of Consumer Affairs' for clarifying certain issues regarding the cause of death. The case related to the death of the complainant's son, aged about 34 years, who met with a scooter accident on the night intervening 22nd/23rd October, 1998. Apart from some laceration on the head, the victim received comminuted fracture of the left distal humerus for which he was operated under general anaesthesia on 27th October at about 8.00 a.m. However, the patient died around 11.00 a.m. during the early postoperative period. The board of doctors conducting the postmortem had opined as to the cause of death as, "The cause of death in the case is asphyxia, which is due to respiratory arrest". Obviously, this provides inadequate information because the term 'asphyxia' in the medicolegal field denotes 'mode of death' and not the 'cause of death'. It needs to be qualified by the condition/finding responsible for its production (the expression 'respiratory arrest' may be acceptable in the clinical setting/situation but not in the 'autopsy diagnosis'). Two major issues surfacing in the complaint were:

- Accusation against the surgeon (opposite party number 2) for not evaluating head injury through X-ray or CT scan before operating the victim.
- Accusation against the doctor administering anaesthesia (opposite party number 3) that he was only MBBS and therefore, was not qualified for administering anaesthesia and was not an expert in that branch.

As far as the first issue is concerned, i.e. in relation to presumption of negligence where no radiograph has been taken following an injury, the Master of the Rolls, Lord Denning said, "In some of the earlier cases, the doctor had been criticised for not having taken X-rays with the result that they had sometimes been taken unnecessarily. This case showed that the courts did not always find that there had been negligence because a patient had not had an X-ray; it depends upon the circumstances of each case." The case in question was Braisher vs. Harefiled and Northwood Hospital Management Committee (CA July 13, 1966). In general, it has been advocated that doctors need not feel that for their own safety, patients/victims be exposed to potential harmful effects that a full 'malpractice check-up' could entail. Such a practice of 'defensive medicine' could lead to over utilisation of healthcare facilities including unnecessary hospital stays and finally, telling upon nation's health bills.

The second issue related to the competency of the anaesthetist on the grounds that he had no special degree/education in anaesthesiology and therefore was not an expert in the field. The doctor, on the other hand, pleaded that he had undergone one year's housemanship in anaesthesiology and had been practicing anaesthesia for about 20 years without any complaint from any quarter. The issue may be considered in the light of following provisions:

- Section 45 of the IEA makes the opinion of persons specially skilled in some science, art, foreign law, identity of handwriting and finger impressions, relevant. Such persons are called experts. It is clear from the Section that to be designated as an expert, the person requires 'special skill' in that field/branch/trade/profession/art/science/foreign law, etc. The word 'skill' essentially conveys the concept of practical experience rather than any specific degree/certificate, etc. Black's Law Dictionary gives the meaning of this word as: "Ability; proficiency, especially the practical and familiar knowledge of the principles and processes of an art, science, or trade, combined with the ability to apply them appropriately, with readiness and dexterity." The concept is amply conveyed in the words enshrined in the case Devi Prasad vs. State, AIR 1967 A11 64: 1967 Cr LJ 134—"The value of the expert does not depend on his qualification rather it depends upon the soundness of the reasoning advanced by him."
- As per Medical Council of India notification dated 11th March, 2002, concerning "Professional conduct, etiquettes and ethics for Registered Medical Practitioners", the Regulation 7.20 reads as "A physician shall not claim to be specialist unless he has a special qualification in that branch". Black's Law Dictionary gives the meaning of the word 'qualification' as "the possession of qualities or properties inherently or legally necessary to make one eligible for a position or office, or to perform a public duty or function". Hence, the MCI regulation carrying the words 'special qualification' may be construed to convey 'special qualities/properties or special experience/training, etc.' There is no expression like 'special educational qualification'.

24 CHAPTER

Consent to and Refusal of Treatment

After going through this chapter, the reader will be able to describe: Types of consent | Scope of consent | Doctrine of informed consent and its components | Hospital's role | Ability to consent | Exceptions to material disclosure | Evidentiary proof of adequate disclosure | Decision-making for the patient without capacity | Sections 53, 53A and 164A CrPC in relation to consent | Doctor-assisted suicide and euthanasia

"Every human being of adult years and sound mind has a right to determine what shall be done with his own body, and a surgeon who performs an operation without patient's consent commits an assault for which he is liable in damages". With these words Judge Cardozo in 1914 expressed a patient's right to autonomy in medical decision making. One who consents to being 'touched' cannot later complain that he or she has been battered, even though the touching may have caused actual harm.

Consent plays an important role in the criminal law in the sense that it has the effect of exonerating or extenuating a criminal act. The concept is primitive one and is based upon the Roman maxim 'volenti non-fit injuria', i.e. he who consents cannot complain of it. The concept was also to be seen in the ruling of the Judge Cardozo in context with the famous amusement ride case: "One who takes part in such sport accepts the dangers so far that they are obvious and necessary just as a fencer accepts the risks of a thrust by his antagonist or a spectator at a ball game the chance of contact with the ball" [Murphy vs. Steeplechase Amusement Co; 250, NY; 479, 482(1929)].

Consent may be defined as the concurrence of 'wills', and its chief essential constituent is the consciousness or knowledge of the act consented to ('will' implies the faculty by which a rational mind makes choice of its ends of action, and directs the energies in carrying out its determinations). **Section 90** of IPC instead of giving positive definition of the word 'consent' defines it in the negative terms. This Section explains that if the consent is obtained by coercion (under fear of injury), undue influence, fraud, misrepresentation or misconception of fact, the consent gets vitiated. Further, the Section goes on to say that the consent given by a person who by reason of unsoundness of mind, intoxication, or immaturity of age (i.e., a child under 12 years of age) is incapable of understanding the nature and consequences of the act to which he consents is not valid in the eyes of the law. **Sections 87 and 88** of IPC speak of exemption of liability when the harm is caused by an act done in good faith and for the benefit of the consenting individual. **Section 89** of IPC deals with cases where the act is done in good faith and for the benefit of the child or insane person, by or by consent of guardian or person empowered to give consent for that purpose on behalf of the child or the insane. **Section 92** deals with cases of emergency. Under this Section, consent may be absolutely dispensed with when the circumstances are such as to render consent impossible or when, in case of person incapable of assenting, there is not one at hand whose consent can be substituted.

Types of Consent

The consent may either be implied or express. **An implied consent** is a consent which is not written, i.e., its existence is not expressly asserted, but nonetheless, it is legally effective. It is provided by the demeanour of the patient and is by far the most common variety of consent in both general and hospital practice. It implies consent to medical examination in a general sense but not to procedures more complex than inspection, palpation, and auscultation. Some medical procedures in which implied consent is readily apparent include an emergency, a comatose patient requiring immediate treatment, a mentally incompetent patient requiring treatment when a legal guardian is unavailable, an intoxicated patient lacking capacity to reasoning and any patient allowing treatment to proceed without objection. Typical situation may be cited when the patient proffers the arm for the venepuncture. Legal problems may not normally arise from a simple venepuncture. However, the issue may get complicated when the repeated attempts are being made, the initial being unsuccessful. Admitted that there is never any intention to harm, it is nevertheless possible that such unskilled attempts may be regarded by the courts amounting to 'recklessness' denoting want of care and breach of duty imposed by law. **An express consent** is one, the terms of which are stated in distinct and explicit language. It may be oral or written. For the

majority of relatively minor examinations or therapeutic procedures, oral consent is employed but this should preferably be obtained in the presence of a disinterested party and not a person closely associated with the patient whose later testimony may be biased. Disinterested witness in a hospital practice could be any literate paramedical staff, e.g. a nurse, a pharmacist, etc. Oral consent, where properly witnessed, is as valid as written consent, but the latter has the advantage of easy proof and permanent form.

Doctrine of Extension and Proportionality

When a patient consents to medical therapy or for the performance of a procedure or a surgical operation, the scope of the consent is limited to whatever parameters were expressed before the medical intervention. Nevertheless, in appropriate circumstances an **extension of the scope of the consent** would be permissible to save the life of the patient. The **doctrine of proportionality** advocates that artificial life support (in the form of respirator, intravenous fluids, or nasogastric feeding, etc.) needs to be maintained as long as it constitutes proportionate treatment, i.e. the treatment that has at least a reasonable chance of providing benefit to the patient that outweigh the burden attendant upon the treatment.

A classical example of the application of this doctrine may be seen in the case of Samira Kohli vs. Dr. Prabha Manchanda & Another (decided by the Apex Court on 16.01.08). Facts, in brief, were as follows:

On 09.05.1995, the appellant, an unmarried woman aged about 44 years, visited the clinic of the respondents for her complaint of prolonged menstrual bleeding for 9 days. After examination and ultrasound report, the respondent had a discussion with the appellant and advised her to come on the next day for making an affirmative diagnosis.

On 10.05.1995, the appellant was admitted for diagnostic and operative laparoscopy. The 'consent form' for surgery filled by Dr. Lata Rangan (respondent's assistant) described the procedure to be undergone by the appellant as "Diagnostic and operative laparoscopy. Laparotomy may be needed".

On 10.05.1995, the appellant was put under general anaesthesia and subjected to laparoscopic examination. Finding her to be suffering from severe endometriosis (Grade IV), the respondent asked her assistant (Dr. Lata Rangan) to obtain the consent of appellant's mother (who was waiting outside) for performing abdominal hysterectomy, i.e. removal of uterus and bilateral salpingo-oophorectomy (i.e., removal of ovaries and fallopian tubes). (The respondent was of the view that had these organs not been removed, there was likelihood of the lesion extending to the intestines and bladder and damaging them. Hence, thought it better to remove uterus, ovaries, and fallopian rubes in the interest of the patient.)

The appellant left the respondent's clinic on 15.05.1995 without settling the bill. Consequently, the respondent lodged a complaint with the police alleging nonclearance of bill by the appellant. However, appellant also lodged a complaint against the respondent on 31.05.1995 alleging negligence and unauthorised removal of her reproductive organs.

The litigation was dragged through various strata of Consumer Courts and finally, National Consumer Disputes Redressal Commission decided in favour of the doctor. However, the Apex Court setting aside 'the order' of the Commission decided the appeal in favour of the patient on the basis of inadequate consent for surgery. Some relevant contents from the judgement are being reproduced:

- Consent given only for a diagnostic procedure cannot be considered as consent for therapeutic treatment. Consent given for a specific treatment procedure will not be valid for conducting some other treatment procedure. The fact that the unauthorised additional surgery is beneficial to the patient, or that it would save considerable time and expense to the patient, or would relieve the patient from pain and suffering in future, are not grounds of defence in an action in tort for negligence or assault and battery. The only exception to this rule is where the additional procedure though unauthorised is necessary in order to save the life or preserve the health of the patient, and it would be unreasonable to delay such unauthorised procedure until patient regains consciousness and takes a decision.
- There can be a common consent for diagnostic and operative procedures where they are contemplated. There can also be a common consent for a particular surgical procedure and an additional or further procedure in the event of doctor/surgeon having anticipated the possible need for the same.
- Highlighting the components of **informed consent**, the Apex Court observed that it should include (*i*) disclosing nature, purpose and procedure of treatment with its benefits and effects; (*ii*) alternatives (if any available); (*iii*) an outline of the substantial risks; and (*iv*) adverse consequences of refusing treatment. "But there is no need to explain remote or theoretical risks involved in the process or in its refusal, so as to frighten a patient either into refusing the necessary treatment or undergoing a fanciful or unnecessary treatment", the court stressed (also see below).

Doctrine of Informed Consent

The term 'informed consent' was first used in 1957 by a California Appellate Court in Salgo vs. Lenand Stanford, Jr., University Board of Trustees case. In this case, the patient consented to an aortogram without being advised, allegedly, of the risk posed by the use of contrast medium. The patient suffered damage and filed a suit against the doctor. The court asserted, "A physician violates his duty to the patient and subjects himself to liability

if he withholds any fact that is necessary to form the basis of an intelligent consent by the patient to the proposed treatment." Since that pronouncement, the doctrine of 'informed consent' has evolved largely through case law and the following elements are generally considered to meet the standard of an **adequate dissemination of information**:

- A doctor should explain to his patient the nature of the procedure, treatment or disease.
- The patient should be informed about the expectation of the recommended treatment and the likelihood of success.
- The patient should know what reasonable alternatives are available and what the probable outcome would be in the absence of treatment.
- The patient should be informed about the particular known inherent risks that are material to the informed decision.
- Of considerable importance, however, is the necessity to convey to the patient the doctor's readiness to listen and to discuss anything the patient may fear as a risk, a side effect, or a concern about the proposed treatment. **Informed consent is an ongoing process**, a two-person conversation extending over time, rather than a form signed once and for all, never again to be discussed.

Who Discloses?

Who is responsible for obtaining the patient's informed consent? The duty falls upon the patient's attending doctor at the time in question, as it is obvious that a doctor is in the best position to decide what information should be disclosed for the patient to make an informed choice, notwithstanding the fact that courts do not provide practical standards of disclosure. The nurse or the other provider may only supplement or complement the doctor's specific information with general information regarding the patient's condition. A substitute doctor covering for the patient's original doctor has an independent obligation to inform the patient of the risks, benefits and alternatives to the part of the treatment that he/she is to administer.

Hospital's Role

A question that may creep, particularly for those practising in a hospital setting, is—Does the hospital has a responsibility to ensure that the patient received adequate disclosure? Under the theory of 'respondeat superior', an employer-hospital could be held jointly liable with an employee-doctor whose failure to obtain informed consent can be shown to have caused injury and damage to a patient. A hospital policy must govern the procedure by which consents are obtained and any deviation from such a policy may be admissible evidence. Hospital liability can arise when the hospital knew or should have known that the doctor did not obtain the patient's informed consent or when the hospital failed to prevent surgery or another treatment from proceeding without the informed consent of the patient.

Ability to Consent

A patient giving consent must be of adult years and capable of comprehending the information provided by the doctor during the dialogue and making a decision concerning the course of treatment. The patients lacking mental capacity to give informed consent require a surrogate, usually a close family member or guardian to give substitute consent. The substitute consent must be obtained through the same process of dialogue that the doctor would have had with patient if he or she were competent. In some situations, where there exists difference of opinion among the family members regarding a patient's care or if family members are distant emotionally or geographically, formal legal proceedings may be advisable to determine who can give consent for the incompetent patient.

Exceptions to Material Disclosure

There may be some exceptions to doctor's duty to make prior disclosure of material risks:

Firstly, the relationship of doctor and patient is considered as of fiduciary nature requiring utmost trust, confidence, fidelity and honesty. However, the concept of 'therapeutic privilege/professional discretion' allows the doctor some privilege under certain circumstances for withholding information from the patient on the basis of the opinion that the information might seriously hurt/harm the patient or make him/her to resort to rash action. Though the concept still seems to have some currency in the sphere of medical profession, yet it is not to be based upon the view that the doctor may remain silent because divulgence might prompt the patient to forego needed therapy. Depending upon the circumstances, the doctor may make revelations to the next of the kin.

Secondly, a competent patient may specifically ask not to be informed, i.e. a patient may waive his or her right to make an informed consent. The patient may reject disclosure out of a desire to remain ignorant or the patient may have already had a similar medical experience.

Thirdly, a doctor is privileged not to advise the patient of the matters that are of common knowledge or of the matters of which the patient has actual knowledge, particularly on the basis of past experience.

Fourthly, no duty to inform arises in an emergency in which the patient is unconscious or otherwise incapable of giving valid consent and harm from failure to treat is imminent. However, an emergency does not give the doctor a license to do whatever

he deems advisable for the patient; it supports only limited measures to save patient's life and/or preserve function.

Evidentiary Proof of Adequate Disclosure

"Document it. If you haven't documented it, you didn't do it." I agree with Dr. Mark E Battista's premise that failure to document usually reflects negatively on the part of the doctor. Moreover, fighting in the patient's medical record is often a key factor that a plaintiff's attorney looks for before undertaking the case. Written documentation of the informed consent is of prime importance for both the parties should litigation later arises. The weight to be accorded to such documentary evidence versus a mere oral consent is a question for the trier of the fact. However, a written consent form signed by the patient often provides strong documentary evidence, which usually forms a rebuttable presumption that valid consent was obtained. It may be necessary to establish the time, location, the persons present and the content of the document, etc.

The imbalance of authority and specific knowledge between the doctor and the patient dictates that society must expect the doctor to pay the highest duty of fidelity and honesty to his patient. In the legal parlance, it has been termed as 'fiduciary relationship' (i.e., relationship of trust and confidence between the doctor and patient). Doctor's over-riding consideration should remain towards his patient, while also remaining aware of his duty towards colleagues and the community at large. Failure of empathy and communication often act as precipitating factors for negligence suits. When something has gone wrong, the doctor should show readiness to assure every step to readdress patient's medical and related problems. Falsification of records to hush up the things, 'scratching out' the records to mend the same or inserting addenda between other notes is readily identifiable. Such modifications/alterations raise suspicion rather than lending support.

Sections 53, 53A and 164A of CrPC in Relation to Consent

SECTION 53: EXAMINATION OF ACCUSED BY MEDICAL PRACTITIONER AT THE REQUEST OF POLICE OFFICER

- When a person is arrested on charge of committing an offence of such a nature and alleged to have been committed under such circumstances that there are reasonable grounds for believing that an examination of his person will afford evidence as to the commission of an offence, it shall be lawful for a registered medical practitioner, acting at the request of a police officer not below the rank of sub-inspector, and for any person acting in good faith in his aid and under his direction, to make such an examination of the person arrested as is reasonably necessary in order to ascertain the facts that may afford such evidence, and to use such force as is reasonably necessary for that purpose.
- Whenever the person of a female is to be examined under this Section, the examination shall be made only by or under the supervision of a female registered medical practitioner.

[**Explanation:** In this Section and in Sections 53A and 54, 'examination' shall include the examination of blood, blood stains, semen, swabs in case of sexual offences, sputum and sweat, hair samples and fingernail clippings by the use of modern and scientific techniques including DNA profiling and such other tests that the registered medical practitioner thinks necessary in a particular case; 'registered medical practitioner' means a medical practitioner who possess any medical qualification as defined in clause (h) of Section 2 of the Indian Medical Council Act, 1956 (102 of 1956) and whose name has been entered in a State Medical Register.]

SECTION 53A: MEDICAL EXAMINATION OF ACCUSED OF RAPE

- When a person is arrested on charge of committing rape or an attempt to commit rape and there are reasonable grounds for believing that an examination of his person will afford evidence as to the commission of such offence, it shall be lawful for a registered medical practitioner employed in a hospital run by the Government or by a local authority and in the absence of such a practitioner within the radius of 16 km from the place where the offence has been committed by any other registered medical practitioner, acting at the request of a police officer not below the rank of a sub-inspector, and for any person acting in good faith in his aid and under his direction, to make such an examination of the arrested person and to use such force as is reasonably necessary for that purpose.
- The registered medical practitioner conducting such examination shall, without delay, examine such person and prepare a report of his examination giving the following particulars, namely: the name and address of the accused and of the person by whom he was brought; the age of the accused; the marks of injury, if any, on the person of the accused; the description of material taken from the person of the accused for DNA profiling; and other material particulars in reasonable detail.
- The report shall state precisely the reasons for each conclusion arrived at.
- The exact time of commencement and completion of the examination shall also be noted in the report.
- The registered medical practitioner shall, without delay, forward the report of the investigating officer, who shall forward it to the Magistrate referred to in Section 173 as part 1 of the documents referred to in clause (a) of sub-section (5) of that Section.

SECTION 164A: MEDICAL EXAMINATION OF THE VICTIM OF RAPE

- Where during the stage when an offence of committing rape or attempt to commit rape is under investigation, it is proposed to get the person of the woman with whom rape is alleged or attempted to have been committed or attempted examined by a medical expert; such examination shall be conducted by a registered medical practitioner employed in a hospital run by the Government or a local authority and in the absence of such a practitioner, by any other registered medical practitioner, with the consent of such woman or of a person competent to give such consent on her behalf and such woman shall be sent to such registered medical practitioner within 24 hours from the time of receiving the information relating to the commission of such offence.
- The registered medical practitioner to whom such woman is sent shall, without delay, examine her and prepare a report of his examination giving the following particulars:
  - the name and address of the woman and of the person by whom she was brought;
  - the age of the woman;
  - the description of material taken from the person of the woman for DNA profiling;
  - marks of injury, if any, on the person of the woman;
  - general mental condition of the woman; and
  - other material particulars in reasonable detail.
- The report shall state precisely the reasons for each conclusion arrived at.
- The report shall specifically record that the consent of the woman or of the person competent to give such consent on her behalf of such examination had been obtained.
- The exact time of commencement and completion of the examination shall also be noted in the report.
- The registered medical practitioner shall, without delay, forward the report to the investigation officer who shall forward it to the Magistrate referred to in Section 173 as part of the documents referred to in clause (a) of sub-section (5) of that Section.
- Nothing in this Section shall be construed as rendering any examination lawful without the consent of the woman or of any person competent to give such consent on her behalf.

Euthanasia

The word **'euthanasia'** is a Greek word, which means easy (or gentle) death. It is a practice of granting a painless death to persons suffering from painful and incurable illness or from incapacitating physical disorder. The various forms may be as shown in Table 24.1.

Table 24.1 Considerations for Euthanasia

Term	Essential features
Voluntary active euthanasia	Intentionally administering medications or other interventions to cause the patient's death at the patient's explicit request and with full informed consent
Involuntary active euthanasia	Intentionally administering medications or other interventions to cause the patient's death when patient was competent but without the patient's explicit request and/or full informed consent; e.g. patient may not have been asked
Nonvoluntary active euthanasia	Intentionally administering medications or other interventions to cause the patient's death when patient was incompetent and mentally incapable of explicitly requesting it; e.g., patient might have been in coma
Terminating life-sustaining treatment (passive euthanasia)	Withholding or withdrawing life-sustaining medical treatments from the patient to let him or her die
Indirect euthanasia	Administering narcotics or other medications to relieve pain with incidental consequence of causing sufficient respiratory depression to result in patient's death
Physician-assisted suicide	A physician providing medications or other interventions to a patient with understanding that the patient intends to use them to commit suicide

AID IN DYING, DOCTOR-ASSISTED SUICIDE AND EUTHANASIA

A doctor's compassionate considerations to pass orders for turning off the respirator in a respiratory dependent patient or for withholding needed medication or not administering hydration and nutrition in patients who suffer excruciating, agonising, slow and very painful deaths may be regarded as some examples of **'letting the patient die'** or **'aid in dying'**. Here, the doctor is playing his role, though on compassionate grounds but with the approval of the patient or the immediate next family member, to the course of treatment or nontreatment. Another example of 'aid in dying' with the doctor's involvement is the 'terminal sedation'. This method of 'doctor-aided dying' includes the administration of large doses of morphine and similar medication that has a dual effect of relieving pain and hastening death of terminally ill patient. Such forms of aid in are not to kill or hasten death but rather to relieve the intractable pain and suffering of the terminally ill patient. Consent, as written earlier, is provided by the patient or the surrogate decision maker (where the patient is incapable of expressing consent). Even the

'advanced medical directives' or the so-called 'living wills' (laws to say in advance that one does not want to be kept alive by artificial means, when there is no hope) have also been recognised. On the other hand, if the patient requests the very same treatment with its known dual effect and the doctor knowingly provides the medication so that the patient can end his/her life, it is considered as **doctor-assisted suicide**. Here, the intent of the patient to end life prematurely by unnatural means is communicated to the doctor, which introduces a criminal element and, therefore, not recommended/advocated. In 'doctor-assisted suicide', the doctor provides the patient with medical know-how (i.e. discussing painless and effective medical means of committing suicide) enabling the patient to end his/her life. Finally, where a doctor or any healthcare provider provides a medical treatment intended to cause death of a terminally ill patient without that patient's or a family member's consent may be charged with **murder or culpable homicide**. The doctor will be committing voluntary active euthanasia wherein the doctor causes the death of a competent patient. Criminal law exists to protect the public interest as opposed to the private interests.

This becomes all the more relevant while looking at the judgement of the Supreme Court, declaring: 'Right to die' not included in the **'Right to life'**. The operative part of the judgement is produced below:

"When a man commits suicide, he has to undertake certain positive overt acts and the genesis of those acts cannot be traced to, or be included within the protection of the 'right to life' under Article 21. The significant aspect of 'sanctity of life' is also not to be overlooked. Article 21 is a provision guaranteeing protection of life and personal liberty and by no stretch of imagination can imply 'extinction of life'. Whatever may be the philosophy of permitting a person to extinguish his life by committing suicide, we find difficult to construe Article 21 to include within it the 'right to die' as a part of the fundamental right guaranteed therein. 'Right to life' is a natural right embodied in Article 21, but suicide is an unnatural termination or extinction of life and therefore incompatible and inconsistent with the concept of 'right to life'. With respect and in all humility, we find no similarity in the nature of the other rights, such as the right to 'freedom of speech', etc., to provide a comparable basis to hold that the 'right to life' also includes the 'right to die'.

25
CHAPTER

Acquired Immunodeficiency Syndrome: Medical, Social, Ethical and Legal Implications

After going through this chapter, the reader will be able to describe: Healthcare worker(s) with HIV infection | Criminalisation of HIV transmission | AIDS and autopsies | Universal blood and body fluid precautions

Introduction

Much of the controversy about AIDS and its victims involves the legal rights of the public whose health should be protected (and 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 such as schooling and so on; 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 him or her but not to the general public. Those in the healthcare profession are acutely concerned because they have a greater chance for personal contact with patients with the disease.

In context with the issues relating to disclosure, some basic information about VCT and ICTC is warranted. The Government of India initiated action for prevention and raising awareness under National AIDS Control Programme, viz., NACP-I (1992–1999) and NACP-II (1999–2000). Based on lessons learnt and achievements made, NACP-III (2007–2012) has been launched. The primary goal is to halt and reverse the epidemic over the next 5 years by integrating programmes for prevention, care, support and treatment. Accordingly, NACO has supported the establishment of voluntary counselling and testing (**VCT**) centres all over India. It is a non-coercive, confidential, and cost-effective approach that provides information, education and communication to motivate behaviour change in HIV-positive individuals. Commencing with 62 VCT centres in 1997, there were 875 centres till 2006. Integrated counselling and testing centres (**ICTCs**) have been established to extend access to counselling and testing facilities and launch comprehensive programmes including prevention, care, support and treatment. The number of ICTCs has increased from 982 in 2004 to 4132 in August 2007. The number of persons tested in these centres has increased from 17.5 lakhs in 2004 to 40.3 lakhs in 2006. Reportedly, there has been an epidemic shift from the highest risk group (commercial sex workers, homosexual men, drug users) to bridge population (clients of sex workers, STD patients, migrant population and partners of drug users), and then to general population.

VCT and ICTC services enable the 'client' to confidentially explore and understand his or her risk of HIV infection, and to learn strategies for preventing HIV and reducing the risk of acquiring or transmitting HIV infection. **Client** means a person seeking healthcare services including VCT (client is considered as 'consumer' who makes a choice whether or not to avail of a certain service). **Voluntary clients** (direct walk-in clients) are those, who present themselves at the VCT centre of their own volition, and free will. **Referred clients** are those, who are referred to the VCT centre for the purpose of HIV testing mainly from within the hospital set-up. **Confidentiality** in context with HIV/AIDS is defined as the state of being 'private'—maintaining the client's privacy by restricting access to personal and confidential information, especially in respect of HIV test results. **Informed consent** for HIV testing implies that the client gives deliberate and autonomous permission to the healthcare provider to proceed with the proposed HIV test procedure. This permission is based upon an adequate understanding of the advantages, risks, potential consequences and implications of an HIV test result. **Counselling** in context with the HIV/AIDS implies a confidential dialogue between a person and a counsellor aimed at enabling the person to explore the risk of HIV infection, to cope with stress and make personal decisions related to HIV/AIDS. **Disclosure** in context with HIV/AIDS

refers to the act of informing any individual or organisation (such as health authority, an employer or a school/institution) of the HIV 'sero-status' of an infected person, with or without consent. Except in circumstances when disclosure to another person is required by law or ethical considerations, the person with HIV has the right to privacy, and the right to exercise informed consent in all decisions about disclosure of his/her HIV status. **Non-voluntary disclosure** of an individual's confidential medical information including HIV status can be made by the counsellor under circumstances like (*i*) to a healthcare worker involved in the treatment, care or support of the people living with HIV/AIDS (PHLAs), where disclosure is medically beneficial for the treatment and to avoid a threat to the life of the infected person; (*ii*) to the spouse/sexual partner or injecting drug partner sharing the same needles when there is a significant risk of HIV transmission, etc. [As per judgement of Apex Court, if the HIV positive partner refuses to disclose the HIV status to the spouse or partner, it is the obligation of the treating physician or counsellor to disclose the result to the spouse/partner of the HIV positive person.]

Healthcare Workers with HIV Infection

A timely and sensitive issue is whether an employer, particularly a healthcare employer, may screen employees for HIV infection and refuse to employ, terminate employment or limit employment of people who are sero-positive. Centre for Disease Control (CDC) estimates that 5.5% of all HIV-positive people are employed in the healthcare field, making healthcare services one of the largest single industry group affected by this issue.

Although there has been no documented case of a healthcare worker infecting a patient with the virus or of an HIV-positive coworker infecting another coworker, the risk is ever present. Healthcare workers who are known to have antibodies to the virus might be advised to refrain from participating in certain traditionally haemorrhagic surgical procedures. Not only would the healthcare provider risk personal liability by continuing to operate in such circumstances, the hospital may be found liable for permitting a healthcare provider with antibodies to the virus to continue to operate.

When a healthcare provider is determined to be sero-positive, or develops AIDS, the hospital should review that person's staff privileges and determine whether or not the medical condition interferes with the person's ability to perform on the job and whether the condition creates a health risk to patients. The healthcare provider's performance must be continuously monitored and evaluated with the goal of protecting the patient. The CDC has recognised that certain direct patient care areas such as surgery may create an increased risk of transmission of HIV from doctor to patient. Although it does not recommend that HIV-positive individuals be routinely restricted from performing surgery, it does recommend that the restrictions be determined on a case by case basis. The healthcare provider could be given other duties in the hospital that involve lesser degrees of direct patient care, or could be required to use extra safety precautions while working with patients.

There is no generally accepted medical evidence that HIV can be transmitted through normal day-to-day contact in typical private workplace settings. The CDC has issued guidelines that recognise that, with the exception of healthcare workers and personal service workers who use instruments that pierce the skin, no testing or restriction is indicated for workers known to be infected with HIV but otherwise able to perform their jobs. Since present medical evidence indicates that the HIV-infected individuals pose virtually no threat of infection to fellow workers, HIV-positive persons in most settings may be permitted to continue their employment as long as they are able to perform their job.

An American study tested 1200 health workers handling HIV patients, 860 of whom knew they had come in contact with blood while administering injections or intravenous drips. It found positivity only in 0.44% of the whole sample. While many doctors feel that this is an occupational hazard, the others who are not prepared to undergo even as low a percentage of risk could be reassured by some government policy. In order to protect healthcare workers from the risk of accidental injury and HIV infection by needles used in the treatment of patients, legislations have been framed in developed countries requiring the use of retractable needles. CDC also advocates the use of retractable needles by medical employers to reduce the number of needle related injuries.

Criminalisation of HIV Transmission

The success of public health is determined by law and politics as much as by medical science. Prevention of 'sexually transmitted diseases' (STDs) is the most legally and politically complex public health problem, as STDs involve the most intimate human behaviours and are intertwined with deeply held religious and moral beliefs. Further, the possibility of HIV transmission during the sexual assault has added to the panic and post-traumatic stress related to sexual victimisation. **Sections 269 and 270 of IPC** are intended to avert the danger to society from the spread of infection. The spread of infection from human intercourse and/or intimate genital contact cannot, of course, be altogether prevented by the measures enacted under these laws. However, the provisions do lay down the limit within which a certain course of action is compulsory, and which is consistent with the underlying principle of criminal law that no one shall be punished for what he could not avoid. Offences described in these Sections are the same, the only difference being that the offensive act under Section 269 is done 'negligently' and under Section 270, 'malignantly'. The presence of malice then aggravates the crime for which higher penalty has been prescribed (indeed, if it could be unmistakably traced, the case would then be one of homicide, i.e. the Section 270 is

intended to deal with those intermediate cases in which the malignant act is not proximate cause of death, but at the same time, it is ultimately traceable to it).

There may be a number of ways in which one might conceptualise **knowledge** as far as the risk of transmission is concerned. This may be exemplified through a couple of situations, viz.:

(i) those who had actual knowledge (because they had tested +ve) and therefore, obviously, criminally liable for transmitting the disease;
(ii) those who were merely carrying awareness that they might be HIV +ve (for their previously engaged sexual activities) and therefore, carried the risk of transmission.

AIDS and Autopsies

Human immunodeficiency virus (HIV) has been isolated in concentration high enough to become hazardous for transmitting infection from blood, semen, vaginal secretions, saliva, tears, breast milk, cerebrospinal fluid, amniotic fluid and urine. However, epidemiological evidence has implicated only blood, semen, vaginal secretions and possibly breast milk in transmission. The morbid anxiety about occupationally acquired HIV infection in the forensic practice has made mortuary workers unduly overcautious. In contrast, a false sense of safety can pose a health hazard and carries public health implications. The serophobia related to the mortuary procedures is without much justification, as there has been hardly any evidence that HIV infection can readily be acquired through participation in a necropsy. The period for which the corpse remains potentially contagious with human immunodeficiency virus is disputable. Reportedly, infectious virus has been recovered from the liquid blood held at room temperature for 3 weeks. Further, culture of blood and effusions from the refrigerated bodies have reportedly shown viable virus at nearly 17 days postmortem. Such information conveys that there was no well-defined safe period at which the virus ceases to be an infective risk.

RISK/BENEFIT DOCTRINE

One school maintains that all autopsies should be carried out with total precautions against infective risk. However, this is almost impracticable to achieve in the present set-up. The other school advocates pre-autopsy testing for HIV and other infective agents by obtaining blood sample from the corpse through a needle puncture. The decision may then rest upon the outcome of the results, depending upon the risk–benefit ratio. However, a blood test before autopsy tends to be governed by ethical considerations, mortuary ethos, laboratory facilities and budgetary constraints. As with all diagnostic testing, the sensitivity and specificity of the test needs to be considered. False positive results may be a source of emotional harm to the family members. False negative results may pose a false sense of security to those with whom the deceased had been in intimacy. Such like situations may compound the issue and when viewed generally, may outweigh any potential benefits. Further, the period between infection and detectable antibody development (the so called 'silent/window period'—about 4 weeks) increases potential for false negative results. Unfortunately, sophisticated tests such as immunocellular culture, genomic RNA or proviral DNA with polymerase chain reaction or direct p 24-core antigen capture immunoassay to detect HIV immunomarkers much earlier are too costly for routine use and therefore beyond practicality.

The precautions to be observed while conducting autopsies may be discussed under the following heads.

AUTOPSY PREPARATION

No unauthorised person should be admitted to the autopsy and body preparation rooms to minimise exposure. The body should be transported to the mortuary by duly plugging all the natural orifices and sites of intravenous drip. It should be wrapped in a plastic bag and closed tightly. Before removing the body from the bag to the table and handling the case, the autopsy surgeon must be properly clothed in the 'AIDS suit' comprising a disposable type of scrub suit, a plastic apron, double rubber gloves, cap, shoe covering, face mask and goggles.

PRECAUTIONS IN THE AUTOPSY ROOM AND LABORATORY

It is almost impossible to know the status of each and every deceased person due to practical, financial, ethical and legal reasons. Therefore, it is preferable to change our work culture and follow universal work precautions. Thus, there may be need to consider all bodies as potentially infected with HIV and adhere to the 'Universal Work Precautions' for minimising the risk of exposure to blood and body fluids. These precautions are based upon the CDC recommendations.

Universal Blood and Body Fluid Precautions

- Entry to the laboratory/work area should be restricted only to persons who are trained to handle infectious material.
- Laboratory door should be closed and should have a 'Biohazard No admission' sign to prevent unauthorised entry.
- Proper protective clothing: Staff should wear a fully covered laboratory coat instead of simple surgical gowns along with heavy autopsy gloves or double rubber gloves, caps, masks, protective eye wear or goggles, shoe covers. Gloves should be worn for all manipulations of infectious material or where there is a possibility of exposure to blood or body fluids. Gloves should be changed at the least suspicion of damage. Hands and other skin surfaces should be washed immediately and thoroughly if contaminated with blood or other body fluids.
- Handling sharp instruments: All workers should take special precautions in handling needles, scalpels and other sharp instruments used during procedure and prevent accidental pricks and cuts.

- Disposal of used instruments: After use, disposable needles and syringes, scalpel blades and other sharp items should be placed in puncture-resistant container, which should be located as close as practical to the used area. To prevent needle-tip injuries, needles should be recapped, purposely bent or broken and removed from the disposable syringe, or otherwise manipulated.
- Workers who have exudative lesions or weeping dermatitis should refrain from work in those areas until their condition resolves.
- Clean-up procedure: Wear intact gloves. Work surfaces should be cleaned and disinfected when procedure is completed at the end of each working day. Small spatters and spills of blood and other body fluids can be wiped up with disposable tissues or towels, which are discarded in a special biohazard bag and properly disposed.

All specimens intended for laboratory examination must be packaged and labelled in such a way that they present no risk to the staff who transport them, or to the staff who subsequently examine them (a clear indication of risk must appear on the specimen container, e.g. 'Danger of infection'). In general, it must be ensured that specimen containers are robust and leak-proof, and that the exterior is not contaminated by blood or other body fluids. The container must be enclosed within a sealable plastic bag. Most retained samples must be fixed in an adequate excess of formalin for a period sufficient to preserve the tissue (potential generation of contaminated aerosols is a reported hazard while slicing and processing the lung tissue, which may lead to contracting tuberculosis).

AFTER COMPLETION OF AUTOPSY

Disinfect the sink. After removing the body, the table surface should also be thoroughly washed and the tabletop should be disinfected. The blood should be mopped with a disinfectant. Although soiled linen has been identified as a source of a large number of certain pathogenic micro-organisms, the risk of actual disease transmission is negligible. Soiled laundry should be handled as little as possible and with minimum agitation to prevent gross microbial contamination of the air and workers handling the laundry. Put the soiled linen into a double plastic bag. At the completion of autopsy, the personnel involved should carefully remove their AIDS suits and put them into a double plastic bag, containing the gloves and sheets used during transportation and tied properly. Label the bag 'Infectious Risk' and send it immediately for incineration. If the contents cannot be immediately incinerated, it should be treated with disinfectant and tightly closed. The healthcare workers should then wash thoroughly with soap and water before dressing.

Disinfectants

Phenolic disinfectants are generally active against many bacterial pathogens (including *Mycobacterium tuberculosis*). They are not generally inactivated by organic matter and do not corrode metal. **Hypochlorites** have the advantage of being active against viruses. However, they corrode metal and get significantly inactivated by organic matter. Where indicated, they are normally used at concentrations of 1000 or 10,000 ppm available chlorine depending upon the degree of contamination. In autopsy room, their main use is in the decontamination of nonmetallic surfaces known or thought to have been contaminated by hepatitis viruses or HIV. **Glutaraldehyde** (2%) is effective against viruses and does not corrode metal. It is, therefore, useful for decontamination of instruments where blood-borne virus contamination is suspected. The instruments need be dipped in the solution for 30 minutes, washed with soap and finally autoclaved. **Embalming** of bodies known or suspected to have been infected with blood-borne viruses is usually not recommended. It is prudent to cremate the body and send the ashes in an urn for later rites. However, in compelling cases where the bodies need be transported outside the place of death, it may be embalmed using formalin 50%, alcohol 45%, phenol 2.5% and water 2.5%.

The major danger to all workers should be any action that produces an aerosol of biologic material, such as that produced by the saw during autopsy. To minimise aerosol splatter, cranium may be opened with an electrical oscillating saw attached to a vacuum dust exhaust and filter or with a hand saw under a transparent anti-slash cover. The most common method of exposure includes being pricked with a used needle or other contaminated material. In preventing this, body sewing needles and staples may be avoided and the mortician's stitches be replaced by suture-free closure using tapes. In case of any accidental prick, it must be reported to the medical superintendent and the immediate proper measures instituted. The individual should be advised that he/she could possibly have been infected by the needle prick and counselled appropriately. HIV testing should be done there and then, after an interval of 3 and 6 months to trace the possible introduction of the virus to the related event so as to evaluate the ensuing claims, if need be.

Reconstruction of the cadaver needs great care as the stitching carries high risk of needle stick injury. Wounds and incisions must be covered to prevent leakage. The body must be enclosed in a robust plastic bag bearing label reading as "danger of infection" so as to caution funeral attendants. Relatives need be discouraged from touching, embracing or kissing the body, because the risk of transmitting infection is greater after the body has been opened for autopsy. Section 297 IPC, in general, deals with the punishment for committing trespass in any place of worship, or any place of sepulchre (burial), or any place set apart for the performance of funeral rites, or for offering any indignity to human corpse, etc. Cases have been reported in the literature showing the approach of the courts in negligent actions for infliction of emotional distress and the negligent mishandling of the corpse (Mackey vs. US, 1993).

26 CHAPTER

Abortion and Delivery

After going through this chapter, the reader will be able to describe: Abortion and its classification | Grounds for justifiable abortion | Rules of the MTP Act | Methods of inducing abortion under the MTP Act | Methods used in criminal abortion | Unskilled, semi-skilled and skilled interference for inducing abortion | Abortion stick and its hazards | Enema syringe and its hazards | Complications of criminal abortion | Examination of a woman who has allegedly aborted | Differentiation between nulliparous and parous uterus | Penal provisions relating to criminal abortion

Abortion

Miscarriage or abortion implies spontaneous or induced termination of pregnancy before the foetus is independently viable (i.e., before 28th week of pregnancy). Legally, miscarriage, abortion and premature labour are considered as synonymous terms indicating termination of pregnancy at any stage before confinement. After conception, it takes about 7–10 days before implantation of the developing ovum takes place when it is called **embryo**. This embryo goes on developing up to the end of 8th week and from then onwards up to birth, it is termed as **foetus**.

Abortion is classified as natural (spontaneous) and artificial (induced). Artificial or induced abortion can further be of two types—legal (or justifiable) and criminal. Table 26.1 lists some differentiating features between natural and criminal abortions.

MEDICAL TERMINATION OF PREGNANCY ACT (1971)

This Act liberalised termination of pregnancy on various socio-medical grounds. It was brought into force on 1st April, 1972 and was aimed at eliminating abortion by untrained persons and in unhygienic conditions and saving lives of women who resorted to criminal abortion out of desperation, thereby succumbing to its complications. (According to Family Planning Association of India, an estimated four million unsafe abortions are carried out every year in the country, and about 20,000 women die due to such abortions. Abortions carried out for the purpose of female foeticide are generally 'mid-trimester' and involve greater risk.) The Act provides the following grounds for termination of pregnancy:

- Where the continuance of the pregnancy would involve a risk to the life of the pregnant woman or of grave injury to her physical or mental health (**therapeutic ground**); or
- Where there is a substantial risk that if the child is born, it would suffer from such physical or mental abnormalities so as to be seriously handicapped (**eugenic ground**).

What may constitute grave injury to pregnant woman's mental health has been exemplified in the explanations attached to the Section, viz.

Explanation 1: Where any pregnancy is alleged by the pregnant woman to have been caused by rape, the anguish caused by such pregnancy shall be presumed to constitute a grave injury to the mental health of the pregnant woman (**humanitarian ground**).

(Though the words used are 'shall be presumed', but such presumption can be rebutted on the facts of the case. There may be no question of anguish caused by pregnancy in the pregnant woman particularly when the girl was keen on continuing the pregnancy and bearing the child.)

Explanation 2: Where any pregnancy occurs as a result of failure of any device or method used by any married woman or her husband for the purpose of limiting the number of children, the anguish caused by such unwanted pregnancy may be presumed to constitute a grave injury to the mental health of the pregnant woman (**socio-economic ground**).

(The Act further clarifies that in determining whether the continuance of a pregnancy would involve such risk of injury to the health, account may be taken of the pregnant woman's actual or reasonable foreseeable environment.)

MTP AND DURATION OF PREGNANCY

For a pregnancy of less than 12 weeks' duration, one medical officer can take the decision of performing abortion. If the pregnancy is beyond 12 weeks but less than 20 weeks, opinion of two medical officers is required to terminate the pregnancy.

Table 26.1 Some Differentiating Features between Natural and Criminal Abortions

Feature	Natural (spontaneous) abortion	Artificial (criminal) abortion
Cause	May reside in the general or local disease of the mother. Disease of the reproductive organs/tissues, etc. Sudden shock/fear/sorrow, etc. Predisposition in some women	Pregnancy in unmarried woman or widow. Occasionally, married woman contracting pregnancy due to some illicit relations may resort to such procedure
Toxic effects of drugs/chemicals	Absent	Erosions and inflammation of vagina and cervix due to local application of irritant and/or caustic substances may be present. GI or urinary tract may show signs of irritation
Genital organs	Injuries usually absent (it has been forwarded that in a miscarriage of 2–3 months' pregnancy, the foetus may be expelled without lacerating the cervical tissue)	Injuries such as abrasions, contusions, and/or lacerations, etc. may be seen in the vagina, cervix, or uterus and its contents. Their site, extent and appearance vis-à-vis the physical development of the foetus need to be taken into consideration to evaluate that whether the same are due to criminal interference or traumatism by the passage of the foetus during spontaneous miscarriage
Infection	Rare (when proper aseptic precautions are not taken in its after treatment)	Frequent (highly suggestive of criminal interference)
Foreign bodies	Not present in the genital tract	May be present in the genital tract
Foetal injuries	Usually absent	May be present

However, in case of an emergency situation, a pregnancy exceeding 20 weeks can be terminated even if only one doctor is available and the opinion to terminate pregnancy is formed in good faith. Further, under such a situation, the specific experience or training in gynaecology and obstetrics, i.e. holding of postgraduate degree or diploma, shall not be applicable.

PLACE FOR MTP

The Act lays down certain requirements in respect of place where the MTP can be performed: government and quasi-government hospitals, MTP centres approved by the government, etc. 'Certificate of approval' by the government must be conspicuously displayed at such approved centres.

EXPERIENCE AND TRAINING OF A DOCTOR

The Act describes 'registered medical practitioner' as one who possesses any recognised medical qualification as defined in clause (h) of Section 2 of the Indian Medical Council Act, 1956, whose name has been entered in a State Medical Register and who has such experience or training in gynaecology and obstetrics as may be prescribed by rules made under this Act. The Rules provide for the following experience and training.

Up to 12 Weeks of Gestation

If he has assisted a registered medical practitioner in performing 25 cases of medical termination of pregnancy, of which at least five have been performed independently, in a hospital established or maintained or a training institute approved for this purpose by the Government.

Up to 20 Weeks of Gestations

The medical practitioner who was registered in a State Medical Register immediately before the commencement of the Act, having experience in the practice of gynaecology and obstetrics for a period of not less than 3 years.

In other cases, if he has completed 6 months of house surgency in gynaecology and obstetrics or if he had experience at any hospital for a period of not less than 1 year in the practice of obstetrics and gynaecology.

In case of a medical practitioner who holds a postgraduate degree or diploma in gynaecology and obstetrics, the experience or training gained during the course of such degree or diploma.

ROLE OF CONSENT

The entire scheme of the Act shows that the provisions thereof can be invoked only by the pregnant woman. An adult woman (18 years and above) can give a valid consent. However, in case of a minor (below 18 years) or a mentally ill woman, consent of a guardian must be obtained. (It is the doctor's responsibility to ensure the age of the patient and to take her guardian's consent if she is a minor. A doctor got into trouble where a minor girl claimed herself to be a major and signed the consent form). Her spouse's consent is not required. However, it is wise to get either husband or any other adult person's signature as a witness to the woman's consent.

ETHICAL AND MEDICOLEGAL CONSIDERATIONS

Let me write the words of Dr. Alec Bourne (1938) before marching to discuss such considerations, "The trauma of pregnancy resulting from rape is not merely physical. We must consider sympathetically the damage it can wreak on a sensitive mind". Liberalisation of rules relating to performance of abortions in the western world owes much to Dr. Alec Bourne. He conducted termination of pregnancy arising out of rape in a 14-year-old girl after obtaining due consent of the parents and successfully defended his action in the Court on the grounds of mental agony likely to be suffered by the victim. This became a benchmark case, serving as a forerunner for the subsequent liberalisation of medical termination of pregnancy.

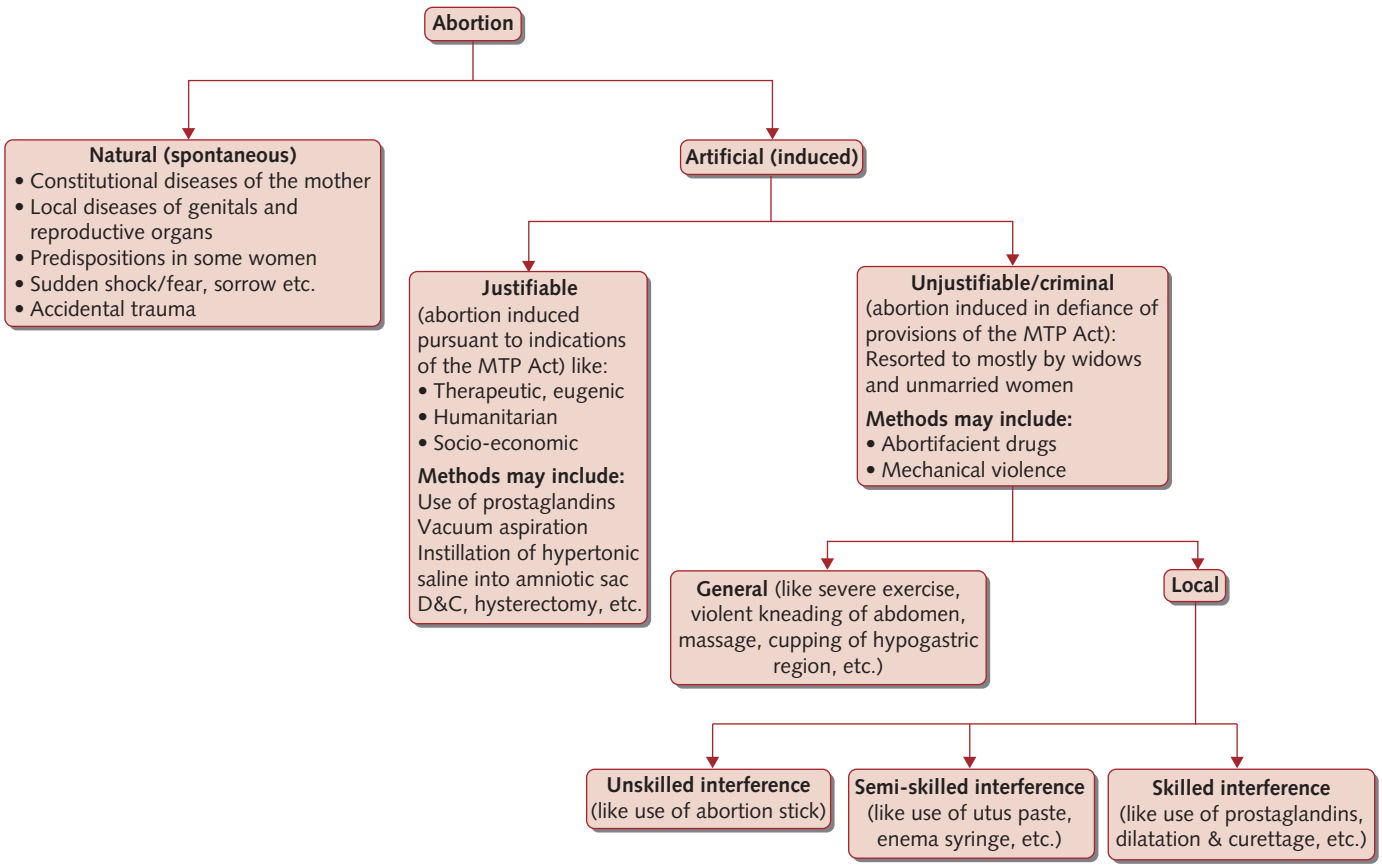
The judge, in summing up, said, "no line can be drawn between danger to life and danger to health; that no doctor knows whether life is in danger, until the patient is dead; and that if on reasonable grounds based on adequate knowledge after consultation with colleagues, a doctor forms an opinion that the probable consequences of the continuance of pregnancy would make the woman a physical wreck, then he is not only entitled, but it is his duty, to perform abortion. To preserve a woman's life is not merely to save her from death; it is also to save her from illness that would destroy so much of her life that it would hardly be worth living."

METHODS OF INDUCING ABORTION UNDER THE MTP ACT (Flowchart 26.1)

During the First Trimester

Medical methods presently include the use of the following drugs:

- **Prostaglandins:** PGE_1 and PGE_{2alpha} are quite effective that bring about uterine contraction and expel the foetus.
- **Antiprogesterones** are the compounds that inhibit the action of progesterone at the receptor site. Mifepristone, also known as RU-486, is the only antiprogesterone available for use in the fertility control. Alone, it is not so effective but when followed by prostaglandins (PGs), it is very useful. For induction of abortion, RU-486 followed after 36–48 hours by PGs has been found to be the most effective medical method (WHO, 1994). RU-486 is a synthetic steroidal compound, having chemical configuration comparable to progesterone; because of this, it competes and binds to the progesterone receptor sites and inhibits the normal biological effects of progesterone on the uterus. Other effects include the following points:
  - It causes ripening and softening of the cervix and produces increased contractility of the myometrium, helping expulsion of the products of conception.



Flowchart 26.1 Classification of abortion and methods used in causing justifiable and unjustifiable abortion.

- It acts on pituitary producing remarkable decrease of LH secretion leading to luteolysis and shedding off endometrium and bleeding in the luteal phase of the cycle.
- It causes marked increase in sensitivity of the uterus to exogenous prostaglandins. When used with PGs, much lower doses of PGs are needed.

Combipack in the form of MTP kit is available presently. Each kit contains one tablet (200 mg) of mifepristone and four tablets of misoprostol (each of 200 μg). During pregnancy, mifepristone sensitises the myometrium to the contraction-inducing activity of prostaglandins. Misoprostol is a synthetic prostaglandin E1. It causes myometrial contractions by interacting with specific receptors on myometrial cells. This interaction results in a change in calcium concentration, thereby initiating muscle contraction. By interacting with prostaglandin receptors, it also causes softening of cervix and thereby resulting in the expulsion of the uterine contents. The dosage schedule constitutes mifepristone 200 mg orally followed 1–3 days later by 800 μg (4 tablets, each of 200 μg) of misoprostol for women at 49–63 days of gestation.

Surgical methods include two types of surgical procedures, namely:

- Vacuum aspiration
- Dilatation and evacuation (also called dilatation and curettage)

During the Second Trimester

Medical methods presently being used these days are one or the other form and in various combinations of **intra-uterine instillation of hypertonic saline (20% NaCl) or urea or rivanol and prostaglandins by various routes**. Hypertonic saline is used either by intra-amniotic instillation into the amniotic sac of the foetus or by extra-amniotic instillation into the extra-ovular space. Even during this trimester, PGs have been found relatively more safe than hypertonic saline in inducing abortion. The mechanism of action of hypertonic saline is not definitely known. Several factors seem to be involved:

- Liberation of prostaglandin from degenerated decidua and foetal tissues;
- Suppression of progesterone synthesis from placenta, thus releasing progesterone block;
- Acute salt poisoning of the products of conception (hypertonicity and dehydration of the foetal-placental unit resulting in foetal death);
- Overdistension of the uterus owing to drawing of fluid by the hypertonic solution; and
- Change in electrolyte balance of the amniotic fluid.

Surgical methods include the following:

- Dilatation and evacuation (D&E)
- Hysterotomy (abdominal hysterotomy is performed when sterilisation is wanted along with termination of mid-term pregnancy or in some cases of failure in the induction of labour. In India, hysterotomy is gaining relative popularity and is preferred for termination of pregnancy in second trimester).
- Hysterectomy (preferred in elderly patients with fibroids and other pelvic pathology).

CRIMINAL ABORTION

Nearly all criminal abortions take place at about 2nd or 3rd month of pregnancy when the woman is certain about her condition. The term **'unsafe abortion'**, proposed by the WHO lately, has been accepted by most other international health institutions. Unsafe abortion means 'abortion not provided through approved facilities and/or persons'. It may be legal or illegal depending upon the abortion laws of the country.

Methods for Inducing Criminal Abortion (Flowchart 26.1)

- Abortifacient drugs
- Mechanical violence, which may be of two types:
  - General violence
  - Local violence

Abortifacient Drugs

Use of abortifacient drugs depends, to some extent, upon local fashions, customs, availability and the faith engendered by the advice from some amateur abortionist. Most of them have no effect on the uterus or the foetus unless given in toxic doses. Drugs commonly employed may be given as follows.

Ecbolics: These are drugs that contract the pregnant uterus, for example:

(a) Ergot preparations
(b) Synthetic oestrogens
(c) Pituitary extract
(d) Strychnine
(e) Quinine

These are not always successful in low doses, and in high doses they may cause danger.

Emmenagogues: These are drugs that initiate or increase menstrual flow, e.g., oestrogen, borax, sanguinarine, savin, etc.

Genitourinary irritants: The drugs that irritate genitourinary tract may sometimes be employed, e.g., cantharides, oil of turpentine, oil of tansy.

Reflex uterine stimulants: Some drugs primarily irritate gastrointestinal tract and reflexly stimulate uterine contraction, e.g., drastic purgatives like julep, castor oil, croton oil, calomel, senna, rhubarb, saline purgatives like magnesium sulphate.

Drugs that are primarily toxic to other systems: These may include inorganic irritants like lead, arsenic, phosphorous, copper, mercury; organic irritants like *Abrus precatorius, Calotropis, Plumbago*, unripe fruit of papaya, etc.

Preparations comprising of seeds and unripe fruit of *Carica papaya*, the seeds of carrot (*Daucus carota*), the unripe fruit of pineapple, the milky juice of madar (*Calotropis gigentea*), the bark of lal chitra (*Plumbago rosea*), etc. are usually used in India.

Mechanical Violence

It may be general or local as mentioned above. General violence acts directly on the uterus or indirectly by promoting congestion of the pelvic organs or haemorrhage between the uterus and the membranes. Local violence acts by (*i*) causing local irritation followed by septic abortion or (*ii*) by dilatation of cervix with or without rupture of membranes.

General Violence This method is usually used during the first month. It is generally believed that abortion is readily precipitated by violence, and the pregnant woman must not be exposed to undue exercise. However, many reports from the literature indicate that general violence is more likely to cause injury than abortion. Such violence may take the form of severe exercise, cycling, riding, jumping from heights and the use of alternate hot and cold baths. Sometimes, some injury is caused directly to the abdomen by tight lacing or violent kneading of the abdomen but it mostly fails because the tenacity of the ovum in a healthy subject is indeed remarkable. Massage of the uterus through the abdominal wall may occasionally succeed and so also cupping of the hypogastric region in later months of pregnancy. (A flaming light is placed on abdominal wall that is covered by a pot or bowl. Air expands due to heat and escapes. On cooling, the air contracts and the pot sets tightly on the abdominal wall. When the pot is pulled, the abdominal wall with anterior uterine wall is stretched forward, which sometimes causes separation of placenta and the consequent abortion.)

Local Violence This method is usually employed in the third or fourth month when the other methods have failed. The method employed depends largely upon the skill and knowledge of the anatomy of the pelvic parts and, accordingly, **the interference may be unskilled, semi-skilled and skilled**. However, it may be laid down as a general proposition that the criminal abortion is always fraught with danger. The need of secrecy and haste and the criminal nature of the procedure are bound to create an emotional disturbance in the mind of the victim that predisposes to shock. Some skilled operators may conduct their procedures, which closely conform to the surgical standards but many of their victims may be subsequently compelled to seek treatment, usually in the hospital, for one reason or the other.

- **Unskilled interference:** It is commonly restricted to self-instrumentation. A woman who is driven in desperation may use anything at hand that is capable of penetration such as knitting needle, pencil, hair pin, etc. (Fig. 26.1). She may even resort to self-syringing or take the assistance of some other person. In such cases, attempts towards asepsis are usually wanting and, therefore, there exists great danger of sepsis being introduced.

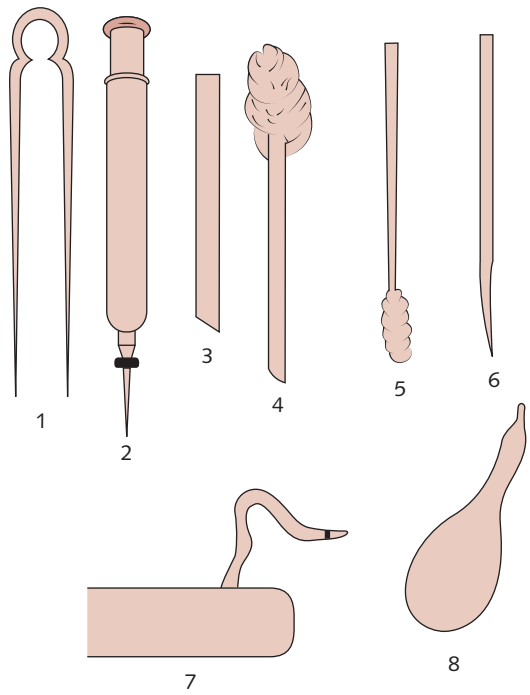


Fig. 26.1 Materials used for causing criminal abortion: (1) hair pin, (2) syringe, (3) nail, (4) and (5) abortion sticks, (6) root of abortifacient plant, (7) douche and (8) part of Higginson syringe.

It is true that women can and do procure their own abortions by local violence. The practicability of such self-abortion, however, depends upon the circumstances. The multipara, having some knowledge of anatomy of pelvic parts, can succeed even without inflicting any injury. The primipara, ignorant of both anatomy and procedure, will have great difficulty and is almost certain to injure herself in and around the genital parts.

Use of abortion stick: A peculiar method of abortion is being practised in India by ***dais***, the so-called midwives/abortionists. The method consists in the use of specially prepared object, known as abortion stick. This object may be a thin, wooden or bamboo stick about 15–20 cm long or a twig of similar length from some irritant plant such as madar (*Calotropis gigantea*), chitra (*Plumbago zeylanica*), lal chitra (*Plumbago rosea*) or kaner (*Nerium odorum*). At one end, the stick is equipped with cotton wool or a piece of rag soaked in an irritant substance such as juice of marking nut or a paste prepared from white arsenic, red lead or asafoetida. The abortion stick is introduced into the os of the uterus. While abortion with or without rupture of membranes may ensue, sepsis is likely to be followed. Excoriation, bruising and perforation in the upper part of vagina or uterus can occur due to irritant action of the substances used and the violence perpetrated during the insertion of the stick.

- **Semi-skilled interference:** It usually comprises **'instrumentation'**. Here, the abortionist usually tries to achieve asepsis either by sterilisation of the instruments or by the use of antibiotic preparations to sterile the genital passage. Large number of metallic/other objects and instruments including hair pins, hat pins, bobby pins, knitting needles, umbrella spokes as well as surgical bougies has been used. Nonmetallic compounds of plastic or celluloid material including urethral catheters have also been employed. **Slippery elm bark** (*Ulmus fulva*) derived from a tree in Central America has been frequently used in the past. When brought into contact with moisture, mucilage is formed around it. This property makes the bark a self-lubricating instrument producing dilatation of the cervix when kept in genital passage. The disadvantage is that when sharply pointed, it may force its way through vagina or uterine wall. Intrauterine injection of **abortion pastes** has also been described. **Utus paste** (a semi-solid soap mixed with potassium iodide and astringents) has been instilled in the extra-ovular space for induction of abortion, mostly in Great Britain, but has been abandoned owing to high rates of failure and infection.

  The enema syringe has been used frequently as an instrument for abortion. The standard Higginson syringe may be used or its nozzle be exchanged for one which is long and slender, to facilitate entry into the uterus. In most cases, plain water or a solution of ordinary soap and water is used, because the method essentially depends upon the mechanical separation of membranes and the placenta by a fluid wedge driven between them and the uterine wall. The soapy solution also acts as an irritant. Some favour the use of solution of Lysol or Dettol or carbolic acid, presumably to disinfect the parts.

  This method carries two major risks: (*i*) sudden death from vagal inhibition through rough insertion of syringe into the cervix or rapid injection of cold or hot fluid and (*ii*) air embolism.

  Air embolism, probably, is a common cause of sudden death in these circumstances. Mixture of air and fluid may enter the circulation through the opened venous spaces resulting from separation of placental membranes from the uterine wall. This leads to entry of soap and air into the large veins, vena cava and eventually the pulmonary arterial circulation and then through the alveolar capillaries into the coronary and cerebral circulation. The risk of embolism increases as the pregnancy advances. In early pregnancy, the antiseptic fluid (soap and water) may enter the peritoneal cavity via the fallopian tubes causing shock and chemical peritonitis. Death usually occurs immediately (within minutes) under such circumstances. However, delayed deaths following air embolism have also been reported. This is possible if the victim is at rest and the air is temporarily locked in the uterus. As soon as the woman gains movement, the placental separation opens the vessels to allow air to enter into the circulation and transference of air into the heart takes place. Gormsen (1960) had reported four cases of abortion with a symptom-free interval of several hours between syringing and death. It is reported through animal experiments that a volume of 480 ml or one as little as 10 ml are the extreme limits. Actual amount that kills the victim may be modified by the general condition of the victim and more so by the rate at which the air is introduced.
- **Skilled interference:** It usually comprises methods likely to ensure immediate and complete evacuation of the uterine contents, and the method approximates to the therapeutic procedure. The well-informed and skilled can succeed in procuring own abortion without causing any injury. **During the first trimester,** skilled interference takes the form of vacuum aspiration or dilatation of cervix under anaesthesia and immediate evacuation of the uterus with the flushing curette (D&C). The preliminary use of laminaria tent (6–12 hours before) is often of considerable help as an aid to the dilatation of cervix. (Laminaria tents are seaweeds available in the Scandinavian countries and Japanese coasts and are supplied to the whole world from these two regions. They are available in different sizes and are sterilised by keeping in absolute alcohol for at least 24 hours. They draw fluid and swell and help in dilating the cervix. They should be used in cases of more than 8 weeks' gestation age, in nulliparous women and in cases with rigid cervix.) **During the second trimester,** PGs can be used to terminate pregnancy either via amniocentesis or through vaginal route.

  In some advanced countries, passage of **electric current** is being used to induce abortion. An electric current of 110 volts via negative pole applied to posterior vaginal cul-de-sac and positive pole to lumbosacral region leads to contraction of uterus and expulsion of the contents. Unless there is a burn or mechanical injury, such crime is difficult to detect.

Causes of Death and Dangers of Criminal Abortion (Fig. 26.2)

Immediate Complications

It is well-recognised that **vagal inhibition or reflex shock** may result in cardiac arrest if the cervix or uterus is manipulated, particularly in an unanaesthetised state and if the patient is in a state of apprehension. As mentioned earlier, the need of secrecy and haste and the criminal nature of the procedure are bound to create an emotional imbalance in the mind of the victim, which predisposes to shock. Sudden death from vagal inhibition can also occur due to rough insertion of syringe into the cervix or rapid introduction of hot or cold fluid.

Air embolism is a common complication following the use of enema syringe for injecting soap solution into the uterus. About 100 ml of air is considered sufficient to cause air-lock and death. Though death is often rapid, delayed deaths have also been reported. It is, therefore, unwise to be too dogmatic as to the time interval between the criminal interference and

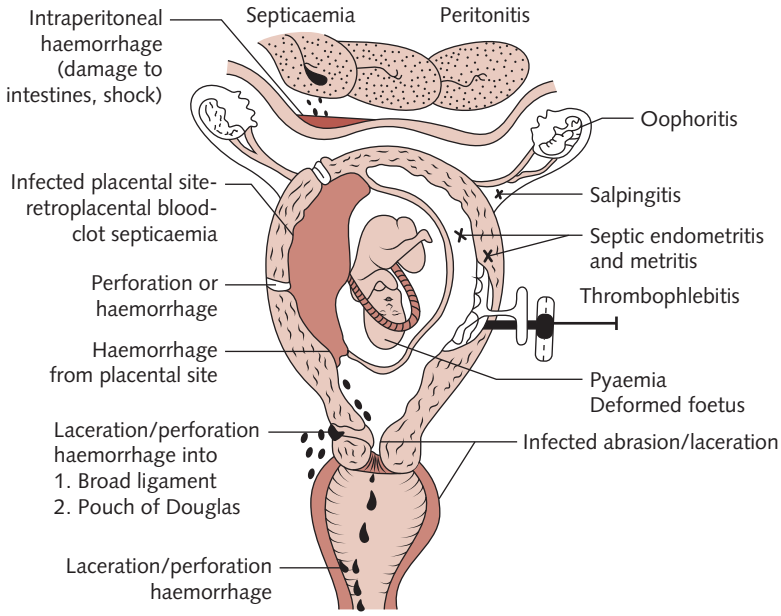


Fig. 26.2 Diagram illustrating the complications of criminal abortion.

death in case of air embolism. Sufficient time may elapse for the woman to take a few paces or to replace the syringe or to throw it somewhere else.

Severe haemorrhage may occur following vaginal or uterine laceration/perforation from instrumentation and responsible for death. Use of abortion stick may sometimes be accompanied by lacerations and perforation leading to rapid death.

Amniotic fluid embolism: Of all the causes of sudden disaster in labour, amniotic fluid embolism ranks high. This is an unpredictable and unpreventable cause of maternal mortality. During labour and in the immediate postpartum period, the contents of amniotic fluid may enter the uterine veins and reach right side of the heart resulting in fatal complications. This fluid contains foetal squames, lanugo, material from the vernix, cells from the chorion and amnion, meconium, and other cellular detritus. The mechanism by which these amniotic fluid components gain entry into the maternal circulation is not clear. Possibly, the contents gain entry either through tears in the myometrium and endocervix, or the amniotic fluid is forced into uterine sinusoids by vigorous uterine contractions. The cause of death may not be obvious, but death can occur as a result of any of the following mechanisms:

- Anaphylactoid reaction to amniotic fluid components.
- Mechanical blockage of the pulmonary circulation in extensive embolism.
- Disseminated intravascular coagulation (DIC) due to liberation of thromboplastin by amniotic fluid.
- Haemorrhagic manifestations due to thrombocytopenia and afibrinogenaemia.

Diagnosis is approached through histology by seeing squames detached from the foetal epidermis. These are much better seen under special stains. Immunohistochemical techniques have also been used in the lung section to demonstrate meconium and amniotic fluid-derived mucin as well as isolated trophoblastic cells in deaths due to amniotic fluid embolism syndrome. However, necropsy findings may get disappeared because of autolysis if there is much delay in carrying out the examination as is often the case owing to lengthy inquest procedures.

Delayed Complications

The main delayed causes of death in criminal abortion are the **sepsis and hepatorenal failure**. Infection can easily be conveyed from the instrument or perineum into the uterine cavity. Any injury to the uterine wall or the neighbouring area or the presence of irritant products with necrosis of the tissue will favour such infection. Infection may occur immediately or days/weeks later. Bacterial shock resulting from endotoxins may be associated with enterobacillary septicaemia. Septicaemia may originate from organisms varying from *E. coli* to *Staphylococci* and nonhaemolytic Streptococci. It occurs more rapidly from uterine invasion by anaerobic organisms like *C. welchii*. Renal failure following acute tubular necrosis used to be somewhat common cause of death prior to methods of dialysis.

- Tetanus is also a hazard. Infection by *Clostridium tetani* usually develops after 3 days to 3 weeks, while infection by *C. welchii* develops usually within 3 days.
- When drugs have been used to procure abortion, death may result from their toxic effects, as most of the reputed abortifacients are irritant poisons. Death may occur during any period depending upon the nature of the drug and the quantity administered.

Penal Provisions Relating to Criminal Abortion

Sections 312–316 of the Indian Penal Code have made criminal abortion punishable. Speaking in legal terms, criminal abortion/causing miscarriage consists of causing the expulsion of products of conception at any time during pregnancy before the term of gestation is completed. The term 'miscarriage' popularly refers to 'spontaneous abortion'. And, **causing miscarriage** in defiance of provisions of the law constitutes an offense and stands for criminal abortion.

Section 312: It makes voluntarily causing miscarriage an offense under two situations, i.e. when a woman is **with child** and **quick with child**. As per judicial interpretations, a woman is considered to be in the former situation as soon as gestation begins, and in the latter situation, when the motion/movements of foetus is/are felt by the mother (obviously referring to the advanced stage of pregnancy). Explanation appended to the Section makes it clear that this Section applies equally to both, to the woman miscarrying and to the abortionist who causes her miscarriage. The offence is committed by the latter **with the consent of the woman** and they, therefore, are both *particeps criminis*. The person who aids and facilitates a miscarriage is liable for the abetment of the offence of miscarriage under Section 312 read with Section 109 of the IPC, even though the abortion did not take place. A person is also liable for attempt to commit a criminal abortion under Section 312 read with Section 511 IPC, even if he fails in his endeavour. For instance, in Queen Empress vs. Aruna Bewa [1873, 19WR (CR) 230], where the term of pregnancy was almost complete and attempted abortion resulted in the birth of child, a conviction under this Section was set aside and one under Section 511 IPC for attempt to bring about miscarriage was maintained.

Section 313: It penalises causing miscarriage of a woman with child (whether she has attained the stage of quickening or not) **without her consent**, i.e. it relates to the commission of an offense of causing miscarriage of a woman when the woman, who primarily interested in the result, is not a consenting party to the act. Here, therefore, only the person procuring the abortion is liable to punishment, which is imprisonment of either description extendable to 10 years and liability to fine. Where the accused woman kicked a pregnant woman in her abdomen resulting in miscarriage, her conviction under this Section was sustained (Tulsi Devi vs. State of UP 1996 CrLJ 940 AII).

Section 314: It prescribes punishment in the situation where **death of a woman has occurred in causing miscarriage**. In order to render the accused liable, all that is necessary is that act should be done with intent to cause miscarriage. It is immaterial whether the act done was or was not intended or known to cause her death (the act might have been done by either administering some abortive, or performing an operation). A person named C was alleged to have had illicit relations with the deceased woman. He took her to the house of appellant doctor for the purpose of aborting her of pregnancy. However, in that process, death of the woman occurred due to rupture of blood vessels occasioned from the use of crude abortion stick. Neither the doctor was qualified for that purpose, nor his clinic was approved by the government under the MTP Act. Further, he did not dare inform the police. Under such circumstances, it was held that there was direct nexus between the death of the deceased with the act done by the appellant. The doctor was convicted under this Section [Telenga Munda vs. State of Bihar 2001 CrLJ 3094 (Pat)].

Section 315: It is aimed at 'foeticide' while in the womb, **after the foetus has developed sufficiently** to assume the human form so as to be designated as 'child'. The Section makes any act done with the intent to prevent a child from being born alive or to cause it to die after birth punishable. The words 'preventing the child from being born alive', obviously, refer to the advanced stage of development of the foetus in the womb so as to be capable of leading an extra-uterine existence in event of its birth. Observations of the court, though pertain to a case decided in 1966 (Jabbar vs. State, 1966 AII), deserve mention here, "where the postmortem report shows that the child had developed sufficiently to have an identity of its own as child, it would be enough to satisfy the definition of the term 'person' as used in the Section 304A of the Code".

Section 316: It finds its application wherein death of a child is caused **when the pregnancy had advanced beyond the stage of 'quickening'. It differs from the preceding Section** in the intention of the offender. Under the last Section, the primary intention of the offender was to cause abortion, failing which, to prevent the birth of a living child or to ensure its death after birth. Under this Section, the intention of the offender is to commit culpable homicide against the mother. And, if in that process, mother however survives but the child with which she is pregnant gets killed, this Section gets attracted. The offence described here may be called as a modified form of culpable homicide as applied to an unborn child through the principle of 'transfer of malice' envisaged under Section 301 IPC. Illustration appended to the Section is quite illuminating. A, knowing that he is likely to cause the death of a pregnant woman, does an act that, if it caused the death of the woman, would amount to culpable homicide. The woman is injured, but does not die; but the death of an unborn quick child with which she is pregnant is thereby caused. A is guilty of the offense defined under this Section.

Examination of the Woman Who has Allegedly Aborted

The doctor may have to examine a living subject, or sometimes a dead body of a woman may be sent to him for the postmortem examination for alleged abortion.

IN THE LIVING

It may include the following steps:

- Requisition from the concerned authority
- Identification of the female
- Written informed consent of the female
- Better to keep some female witness (nurse) nearby
- Short history must be obtained as to date, time, place of abortion, means adopted to procure abortion and material expelled, etc.
- Clothing must be examined meticulously, especially the undergarments for some stains of blood and, occasionally, of some liquid abortifacient agent used locally. These must be retained for contact traces of any foreign substances.

The findings are more or less akin to those found in the recent delivery and will depend upon the period of gestation, the mode of abortion practised and the time elapsed between abortion and the examination. As in case of delivery, so in case of abortion too, some of the findings are related to pregnancy and some to the expulsion of products of conception. Therefore, the doctor should examine breasts, pigmentation of different places and the abdominal wall. Changes will be appreciable depending upon the period of gestation at which the abortion was carried out.

Since most of the reputed abortifacients are irritants, the women may show signs of ill-health, gastrointestinal disturbances and nervous exhaustion. In case of localised sepsis, temperature may be raised and the woman remains indisposed for some days. The serum and urine of woman gives positive result for the tests for hCG up to about 7–10 days.

- Appearance of perineum, external genitals and vagina should be noticed. See for presence/absence of injuries in the form of abrasions, lacerations, contusions, etc.
- Condition of os needs to be examined. It remains dilated for a few days. The extent of dilatation and period of its stay depends upon the size of the foetus expelled. It may also show abrasions/tears/contusions, etc. due to instrumentation.
- Presence of recent tears, the marks of forceps or other instruments in and around genitals should be observed.
- Character and amount of discharge need to be observed.

In abortion during the earlier months of gestation, the signs will be ill-defined and return to normal in a few days. Signs persist for a longer time if sepsis has taken place or if abortion has been carried out in later months of pregnancy.

IN THE DEAD

There have been relatively few deaths from criminal abortion, but it must be borne in mind in the investigation of any unexpected death in a female of child-bearing age. Prompt action on the part of police is also desirable to prevent disposal of the evidence.

External Examination

It will include formal identification of the deceased and meticulous examination of the clothing as detailed earlier also. General external findings upon the breasts and abdominal wall should be noticed. Presence of scars, injuries (general as well as local injuries) needs detailed description. Expression of fluid from breast may be of indirect value in the diagnosis of recent abortion. Areas of distribution of hypostasis carry significance. In many cases, death being due to haemorrhage, the body may look extremely pale with less prominent hypostasis.

Labia majora, minora, vagina and/or cervix, etc. may show injuries and may be congested. These may be stained with locally used abortifacient agent, when such agent has been used. If the agent was injected parenterally, then the injection mark(s) will be detectable over the usual sites of intramuscular injections.

Where air embolism is suspected, pre-autopsy radiology of the chest and abdomen must be performed for visualising air bubbles in the heart, great veins in the thorax, peritoneal cavity, and pelvic veins. Availability of MRI or computed tomography may greatly increase the chance of detecting air in the vessels. Further, where radiology is not possible, the air needs to be sought in the cardiovascular system through dissection. The usual method is to open the heart chambers in situ under water poured into the pericardial sac. Escaping bubbles are considered to be indicative of air embolism. Delay in conduction of postmortem allows absorption of air and therefore, at instances, one may not be able to demonstrate air in the heart on dissection, while it was demonstrable on radiology. Conversely, the possibility of pseudo-embolism due to formation of gases of decomposition may be kept in mind in cases where decomposition has set in (see also the Chapter 'Complications of Trauma').

Internal Examination

Initially, a small suprapubic incision should be given and any crepitation due to gas formation in the uterine cavity and venous channels, if suspected, should be assessed. The uterus, ovaries and the adnexal tissues should be removed en masse following severance of the symphysis pubis and including vagina, vulva and rectum with adjacent skin, taking care to collect any foreign fluid/material.

The abdominal cavity may be seen to contain liquid and clotted blood, if there is perforation of uterus due to instrumentation. In such cases, injuries to the neighbouring organs like intestine or bladder should also be noticed.

Findings in the Uterus

The size of the uterus deserves observation. It may be enlarged, soft and congested. Wall may show thickening on longitudinal sectioning. Cavity may show presence of products of conception in full or in parts. It is advisable to weigh the uterus and measure its size. Nonpregnant uterus weighs about 40 gm; it is 7.0 cm long, 5.0 cm broad and 2.0 cm thick. The length is

approximately 10 cm at the end of third month, 12.5 cm at the end of fourth month, 16 cm at the end of sixth month, 20 cm at the end of eighth month and 27 cm at the end of ninth month.

It may show perforation. Endometrium may show evidence of scooping, if evacuation was done by curetting. If the evacuation was incomplete, the placenta may still be there. Otherwise, the site of placental attachment may be seen as a raw and slightly depressed area. In case of use of any chemical, the inner surface of uterine wall may be stained and/or damaged. If soap water was used, froth may be present in the cavity. In some cases, there may be presence of hair pin or root of a plant if any of these elements was used. Swabs of the uterine wall should be taken for microbiology. Tissues may be preserved in 10% formalin for histology.

Ovaries

Either of the ovary should be examined for the presence of corpus luteum. They may be congested. In some cases where there is suspicion that a poisonous substance has been used locally to induce abortion, then the specimens of vagina, uterus and appendages need to be sent for toxicological examination.

Heart

After opening the chest cavity, right ventricle is opened to elucidate the presence of frothy blood, which is often seen in air embolism. Samples of blood from the inferior vena cava and both the ventricles should be collected.

Examination of Aborted Material

Sometimes the substance alleged to have been expelled from the uterus as a product of conception is brought to the doctor for his opinion as to the nature of the substance/material. When available in pieces, it is suggestive of instrumental abortion. All material passed should be examined microscopically. In the early months of pregnancy, if the embryo is not found, the presence of chorionic villi found under the low power of microscope will decide the fact of miscarriage. It is always advisable to remove a piece of tissue from what is thought to be the placental site and examine it microscopically for the presence of chorionic villi. It is a sure sign of pregnancy. While examining such an aborted foetus, it is important to ascertain its age, as it has a bearing under the legal provisions. This may be determined from the Hasse's rule.

HASSE'S RULE

For the first 5 months of the foetus	Square root of the length of the foetus in cm gives its age in lunar months
After 5 months	1/5th of the length of the foetus in cm gives the age in lunar months

Thus, when the foetus measures 16 cm, its age is $\sqrt{16} = 4$ lunar months. When the foetus measures 35 cm, its age is $35/5 = 7$ lunar months (also see the Chapter 'Infanticide and Foeticide' for determination of age of the foetus).

IMPORTANT FOETAL AGES RELATED TO ABORTION

12th week: Length of the foetus is 9 cm, weight is about 30 gm. Pupillary membrane is present in the eye. Scalp hair and lanugo hair are absent. Sex is not distinguishable. Meconium is absent. Ossification centres for clavicle, mandible, ribs and vertebrae are present.

20th week: Length of the foetus is 25 cm; weight is about 400 gm. Scalp hair and lanugo hair have appeared. Sex can be differentiated. Gallbladder is present. Meconium present at the beginning of ascending colon. Ossification centres for manubrium, 1st segment of sternum and calcaneum may appear.

28th week (stillborn above this age). Length is about 35 cm, weight around 1 kg. Scalp hair—usually more than 1 cm long. Nails are thick and proximal to tips of fingers. Left testicle is at the external ring. Gallbladder contains bile. Meconium is present up to the distal end of large intestine. Pupillary membranes vanish, eyelids can be opened. Ossification centre for talus usually has appeared.

30th week or 210 days (usually accepted as the age of viability). Length about 37 cm, weight around 1500 gm. Scalp hair about 2.5 cm. Nails almost reach the tip of the fingers. Left testicle in the scrotum, right near the external ring. Ossification centres for all sacral vertebrae present.

Delivery

Delivery means expulsion or extraction of a foetus (that has attained viability) and other products of conception from the genital passages into the environment. The process may be spontaneous or assisted. When delivery occurs after full period of gestation, i.e. 280 days, it is called 'full-term delivery'. If it occurs earlier or later than this, it is said to be 'preterm/premature' and 'post-term/postmature', respectively.

Sometimes, a doctor is asked to examine a woman for signs of delivery and, if there is evidence of such delivery, to state the probable time since her delivery. The signs of delivery are better marked where it is recent and the child is more mature. Therefore, it is important to make the examination at the earliest possible opportunity. This is an obstetrical exercise and where the issues are important, a specialist obstetrician and gynaecologist should be consulted on the living person. However, where an autopsy is to be conducted, the issues may be straightforward.

The legal issues linked with delivery may include the following:

- **Feigned delivery:** When a woman may pretend to have been pregnant for some time and later produces a child,

alleging that it is her to claim property of a deceased husband. Such a child is called a 'suppositious child'.

- **Affiliation cases:** Where a woman claims that her child has been fathered by a person who is not her lawfully wedded spouse, or by a husband who has subsequently divorced her, and who must therefore adopt the child as his own and pay for its upkeep.
- **Legitimacy:** When as part of the evidence that a child was born of a particular woman and her husband, it must be proven that woman did indeed deliver a child at the time claimed by her.
- **Disputed chastity:** When an unmarried woman, a widow or a wife living apart from her husband is defamed or libelled to have delivered a child, she may like to vindicate herself.
- **Abortion and infanticide:** When a woman is alleged to have aborted, or delivered and killed the child.
- **Concealment of birth:** If a child is born to an unmarried woman or widow, or out of lawful wedlock, the woman might wish to conceal the child out of shame.
- **Blackmail:** A woman may produce a suppositious child and blackmail someone as the father to extort money.
- **Nullity of marriage:** when delivery occurs shortly after marriage, the issue may be dragged to the court for the nullity of marriage.
- **Divorce:** When delivery occurs, with the wife during calculated period of conception being apart from the husband.
- **Criminal breach of trust:** In case of prior promise of marriage.
- **Inheritance of property:** When the delivery is followed by death of the husband, the widow may claim greater share in the ancestral property of the husband.
- **Homicide and suicide:** Delivery of a child by an unmarried or a widow may be the cause for killing of that woman. Sometimes, she may even commit suicide due to sheer frustration.
- **Higher compensation claim:** In case of death of husband on duty.
- **Additional leave facility:** For working women.
- **Delivery:** After being pregnant as a result of rape, kidnapping, seduction, etc.
- **Execution of judicial death sentence:** May be delayed up to 6 months after delivery.
- **Deferring trial of a case:** In a Court of Law as in case of pregnancy.

In such cases, it is obvious that the doctor will have to examine the woman to determine if she was pregnant and delivered a child. As in the case of examination for pregnancy, informed consent of the woman is necessary.

SIGNS OF RECENT DELIVERY IN THE LIVING

When the delivery has been recent, the following findings may be helpful in determining the state of affairs.

General disposition: For the first 2 or 3 days after delivery, the woman wears a languished look with sunken eyes, and having a dusky pigmentation about the lower eyelids and often shows a slight increase in the pulse and temperature. These signs may be absent in strong women, or may be found in any other illness or at the time of the monthly course. The intermittent contractions of the uterus are usually present for the first 4 or 5 days. These are termed 'after pains' when they are vigorous and painful.

Breasts: There is breast enlargement, and the breasts are full, firm, knotty and tense with darkening of the areolae and prominent Montgomery's tubercles (these tubercles are enlarged sebaceous glands and appear as raised spots in the areolae). The surface veins are prominent, and there is more or less dark coloured pigmentation round the nipples. Striae are usually seen on the breasts; on pressure, colostrum can be squeezed out from the breasts. (**Colostrum** is the secretion of the breasts during the latter part of pregnancy and for the 2–4 days after delivery. It has a deep lemon-yellow colour, its reaction is alkaline, and its specific gravity is 1.040–1.060, in contrast to the average specific gravity of 1.030 for mature breast milk. It contains several times the protein of mature breast milk and more minerals but less carbohydrates and fat. It also contains some unique immunologic factors. After the first few days of lactation, colostrum is replaced by secretion of a transitional form of milk that gradually assumes the characteristics of mature breast milk by the 3rd or 4th week.)

Abdomen: In multiparae, the skin of the abdominal wall is frequently flaccid, and may be wrinkled. In primiparae, the condition of flaccidity may not be so marked, due in large measure to the better tone of the abdominal structures. There is usually evidence of **striae gravidarum** (slightly pink in colour), due to the stretching of the skin of the abdomen and resultant formation of scar tissue in the deeper layer of the cutis. As time elapses, the striae become silvery-white in colour as a result of diminishing vascularity of the scar tissue, and are termed **lineae albicantes**. Some degree of pigmentation, chiefly in the neighbourhood of the umbilicus and median abdominal line **(linea nigra)**, is frequently present. On palpation of the abdomen, the fundus of the uterus is usually palpable as a flabby mass extending almost to the umbilicus a few hours after delivery. It then reduces in size progressively at approximately half an inch per day. By the tenth day, it is felt on a level with the brim of the pelvis. In 2–3 weeks, the fundus sinks below the level of the pubis into the pelvic cavity. It reaches its normal size in about 6 weeks.

External genitalia: The **labia** are tender, swollen, gaping and bruised or lacerated. The **fourchette** is usually ruptured and perineum is sometimes lacerated. The **cervix** is soft and patulous and its edges torn and lacerated transversely. The internal os begins to close within first 24 hours. The external os is soft and patent admitting two fingers for the first few days and one finger with difficulty at the end of a week. It remains open for a number of days, taking between first and second week to

close. There is a **vaginal discharge**, which in many cases presents a peculiar sour disagreeable odour. This discharge is known as **lochia** and is a part of the healing process of the uterus after delivery. It contains red cells, leucocytes, decidual debris, vaginal epithelium, etc. During the first 4–5 days, it is bright red in colour and therefore known as **lochia rubra** consisting of blood mixed with large clots, and is strongly suggestive of recent delivery. During the next 4 days, it becomes paler and serous and therefore known as **lochia serosa**. At about the ninth day, it becomes yellowish-grey or slightly greenish, when it is known as **lochia alba** or **green water**. It then gradually diminishes in quantity, till it disappears in about 2 weeks.

From the above signs taken collectively, it will scarcely be difficult to diagnose a case of recent delivery for about first 2 weeks after parturition. These signs are more characteristic of a full-term delivery than of a premature one. They are likely to disappear within a week or ten days or even at an earlier date in a strong and vigorous woman, especially if she happens to be a multiparous. **The biological tests** may be of value in certain cases. They remain positive for about a week or so after delivery. Usually, after 2 or 3 weeks, it is impossible to fix the date of delivery with any degree of certainty.

SIGNS OF RECENT DELIVERY IN THE DEAD

The diagnosis of recent delivery in the dead hardly presents any difficulty. The external and genital signs of recent delivery as are available in the living subjects will also be present in the dead subjects. In addition to these, **additional internal findings on dissection will be demonstrable in uterus and its appendages**. The uterus is soft, flabby and enlarged in size, which gradually becomes firm and returns to a permanent reduced size. The measurements of uterus at different times after delivery are given in Table 26.2.

The placental site can be identified shortly after full-term delivery by its dark colour and coarsely granular appearance. It is about 15 cm in diameter and covered with clotted blood, lymph and portions of decidua. The site measures 3–5 cm at the end of second week.

The opening of its vessels are well-marked and recognisable up to 8 or 9 weeks after delivery. **The ovaries and fallopian tubes** are usually congested but may become normal in a few days. In case of doubt, histological examination needs to be resorted to, wherein endometrium may show the presence of trophoblastic epithelium and chorionic villi. A large corpus luteum is usually detectable in one or other ovaries.

Table 26.2 Approximate Weights and Dimensions of Involuting Uterus

Duration after delivery	Weight (gm)	Dimension (L × B × Th) (cm)
Immediately after delivery	900	20 × 15 × 5
2–3 days after delivery	700	17 × 10 × (4–5)
End of the 1st week	500	14 × 8 × 4
End of the 2nd week	300	11 × 7 × 3
5–6 weeks after delivery	80–100	10 × 6 × 2.5

SIGNS OF REMOTE DELIVERY IN THE LIVING

After the initial post delivery phase is over, there are certain changes in the parturient female that persist virtually indefinitely. The extent and character of the signs found will depend upon whether the woman is primiparous or multiparous. The diagnosis of past pregnancy may be considered justifiable if all or a majority of the following signs are demonstrable:

Breasts: It may remain large and perhaps pendulous, due to loss of their previous nulliparous elasticity. Nipples are usually prominent and retain their hyperpigmentation. Montgomery's tubercles are usually present. In some multipara, there may be stria present on the surface of the breasts.

Abdomen: The abdominal wall may never regain its former elasticity and usually remains lax. It shows the presence of white silvery streaks, called the **lineae albicantes**, the result of hyperdistension of the skin and resultant scar formation in the cutis due to rupture of deeper layers of the skin. There is commonly a deeply pigmented line **(linea nigra)** from the pubis to the umbilicus.

External genitalia: The appearances of **vulva** are variable, as sometimes the post-delivery vulva may return virtually to normal, but commonly there is laxity of the labia that do not come into close apposition as previously. The **vaginal rugae** are usually lost. The **fourchette** is almost always lost. The **hymen** is virtually completely destroyed and may be almost undetectable, though usually some nodules are left on the vaginal wall called the 'carunculae myrtiformes'. There may be a scar of an old perineal tear or an episiotomy. Another important finding is the appearance of the os uteri. **In a parous woman**, the internal os is not well-defined while the external os is transverse, irregular, fissured, and may admit the tip of the finger. **In a nulliparous woman**, the internal os is well-defined, while the external os is rounded with a dimple in the centre and the orifice closed (Fig. 26.3).

Most of the above signs may possibly be simulated by the passage of a large fibroid tumour per vaginum. Again, most of these signs may disappear in a woman who has had only one delivery short of the full-term several years ago, and it is possible for the vagina and uterus to regain normal appearances as observed in a nulliparous woman. The examining doctor, must therefore, be prepared in exceptional instances to meet with cases where a woman who has borne one or more children may not exhibit any of the above signs, or little evidence of them.

SIGNS OF REMOTE DELIVERY IN THE DEAD

In a dead subject, in addition to the signs available in living subject for remote delivery, examination of uterus and its

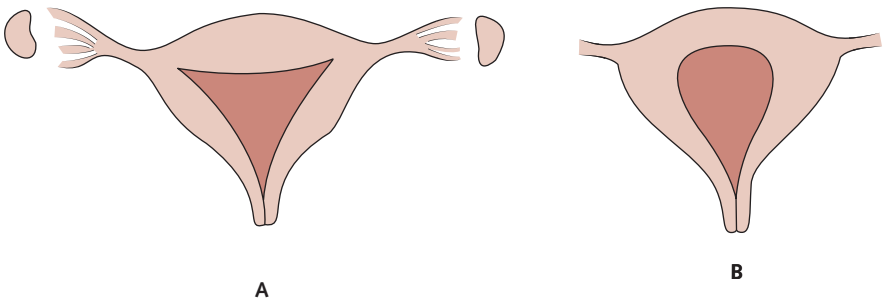


Fig. 26.3 **(A)** Shape of uterine cavity in case of a nulliparous woman. **(B)** Shape of the cavity in case of a parous and gravid woman.

Table 26.3 Differences between Nulliparous and Parous Uterus

	Nulliparous uterus	Parous uterus
Size	Smaller ($7 \times 5 \times 2\,cm^3$)	Larger ($10 \times 6 \times 2.5\,cm^3$)
Weight	40–50 gm	80–100 gm
Ratio between body and cervix	Length of body and cervix almost equal	Body length is almost double the length of cervix
Upper surface of fundus	Less convex and is almost in the same line as broad ligament	More convex and is at a higher level than the line of broad ligament
Uterine cavity	The inner walls are convex, and that makes a triangular cavity with less space (Fig. 26.3A)	The inner walls are concave, and that makes a comparatively large and spacious cavity (Fig. 26.3B)
Scar for placental attachment	No such thing	It is present. May be very faint in very old pregnancies
External os	Small roundish dimple-like depressed opening	Transverse slit-like opening
Internal os	Circular, well-defined	Ill-defined, with wrinkled margin

appendages will be of additional assistance. **The uterus** usually does not involute to such an extent that it resembles the nulliparous womb. It remains larger and thicker and tends to have a concave interior, rather than the convex inwardly bulging walls of the uterus, which has never carried a pregnancy. **The fundus** tends to be higher than the line of fallopian tubes, which is not so in the nulliparous uterus. **The length of the body** is twice the length of the cervical segment in multiparous; whereas in the nulliparous uterus, the length of the body is about the same as that of the cervix (Table 26.3).

The **cervix** is irregular in form and shortened, and its edges show cicatrices on account of previous tears and lacerations caused during delivery. The **external os** is enlarged, irregular and patulous so as to admit the tip of the finger, and the **internal os** is not so well-defined as in the virgin or nulliparous woman. (**'Arbor vitae'** is the name given to the mucosal folds in the canal of the uterine cervix, which extends from internal os to external os. The canal is spindle shaped and has two transverse mucosal folds, one on anterior and another on posterior edge of the canal. Numerous oblique mucosal folds run from these transverse folds, giving the appearance of a tree and hence the name 'arbor vitae'. These folds disappear in a parous woman.) It must be remembered that uterus undergoes atrophy in old age and makes such differentiation very difficult. After about 2 months, it is not possible to give the exact period of delivery.

27
CHAPTER

Impotence, Sterility, Sterilisation and Artificial Insemination

After going through this chapter, the reader will be able to describe: Impotence and sterility | Examination of a case of impotency and sterility and expressing opinion | Causes of impotence and sterility in the male and female | Medicolegal aspects of sterilisation | Types, procedures, guiding principles and legal status of artificial insemination | Test tube baby | Concept of 'wrongful pregnancy', 'wrongful birth' and 'wrongful life' cases

The question of impotence in the male arises frequently in nullity of marriage cases, and less frequently in disputed paternity cases, and in cases of gender-related offences. **Impotence** is the inability to perform, or permit the performance of, the act of sexual intercourse, whereas **sterility** is the inability to impregnate/get impregnated. Incapacity leading to impotence can be of absolute or relative type. **In the absolute type**, there is inability of intromission or introduction of the male organ into the vagina, e.g. absence of penis or even the partial amputation of penis making the sexual act impossible. In case of females, absent or rudimentary vagina likewise makes the act impossible because of configuration of the female organ (the main factor governing potency in the females is the patent vaginal canal). The question of **relative impotency** is hard to decide and is mainly influenced by psychic background.

It must be remembered that 'impotency' and 'sterility' denote lack of two different powers. A person may be lacking both the powers or only one of them. Excision or involvement of both testes as a result of injury or surgery most often leads to impotence and sterility. However, in relation to impotence, the concept carries some reservations. For example, if the eunuch is castrated before puberty, the castration is followed by impotence. This castration, if effected after puberty, does not effect the possibility of erection and hence, of coitus. According to Reyden, sexual intercourse or consummation is referred as 'vera coupla' consisting of erection and intromission, i.e. erection and penetration by the male of the female. Baxter vs. Baxter (1947) 1 A11 ER 387 was the first case where House of Lords held that the procreation of children was not the sole purpose of marriage. The question of curability of impotence is not a relevant consideration for the purposes of the law (Samar vs. Snigdha, AIR 1977 Cal 413).

The question of impotence and sterility may arise in civil as well as criminal matters as given below:

In civil cases, this question may arise in (*i*) nullity of marriage and divorce (where marriage cannot be consummated on account of impotency), (*ii*) in cases of contested paternity and suits for adoption (where the alleged father pleads impotency and/or sterility as his defence), and (*iii*) claim for damages where loss of sexual function is claimed as the result of assault or accident.

In criminal cases, this question may arise in (*i*) accusations of adultery, rape and un-natural sexual offences (where the alleged condition of impotence is put forward as a defence) and (*ii*) in cases where a sterile woman puts forward a supposititious child to claim property.

EXAMINATION OF A CASE OF IMPOTENCY

The examination should be undertaken only when asked by the court or the police. Before examining, care should be taken to obtain informed consent. Complete history of the previous illness, especially with reference to nervous and mental condition and sexual history, should be obtained. A general physical examination should then be made followed by systemic examination. Special attention should be directed to the nervous system and assessment of the mental condition.

The private parts must be examined for injuries or malformations with a view to ascertaining any impediment to intercourse, and whether the same is permanent or irremediable. The condition of testes, epididymis, cord and penis should be noted and private parts tested for sensations. The length of the penis is measured from mons to the tip of glans, and circumference about middle of the shaft. The penis varies greatly in size. The size of penis has less constant relation to general physical development

than that of any other organ of the body. The axis of the erect penis averages 26° to the horizontal ranging from 16° to 36°.

It should also be ascertained if the impotence existed prior to marriage, as the impotence occurring subsequent to marriage does not constitute a ground for divorce.

Opinion

If the doctor finds that the person is normal in all respects, i.e. physically well-developed with normal genitals, well-developed secondary sex characters and is not carrying any obvious cause of impotence, he is justified in certifying that there is nothing to suggest that the person is impotent. In all such cases, **opinion should be given in negative form** stating that 'from the examination of the individual there is nothing to suggest that the person is incapable of performing sexual intercourse'. Proof of potency or impotency is largely inferential, as is obvious from **Casper's statement**, "The possession of virility and procreative power neither requires to be, nor can be, proved to exist by any physician, but is rather, like every other normal function, to be supposed to exist within the usual limits of age".

EXAMINATION OF A CASE OF STERILITY

In case of male, semen may be obtained either through the act of masturbation or pressing the vesicles through the rectum. It should be examined for spermatozoa. It is desirable for the individual to be examined to abstain from sexual intercourse for about a week or so before examination. The sample of semen should be examined as early as possible. Fertility in the male is reduced clinically when the total sperm count falls below 60 million (average about 100 million per ml) or the motility of sperms in a fresh specimen of semen is impaired. **In case of female**, attention is directed to the development of ovaries, uterus and patency of the fallopian tubes.

Causes of Impotence and Sterility in Males

Causes of impotence and sterility in males may be described under the following heads:

AGE

Impotence is generally observed at the extremes of age. Boys are considered to be sexually potent at the age of puberty, which usually occurs at the 15th or 16th year of life. Sexual intercourse, however, may be possible at about 13th or 14th year as the power of coitus commences earlier and ceases later than the power of procreation. The changes that usually occur in a boy at puberty are the development of genital organs, the ability to secrete semen, the growth of hair on the pubes, axillae and chin, and the increase in size of the larynx leading to the deepening of the pitch of the voice.

As the age advances, the power of sexual intercourse and procreation diminishes, but no limit can be assigned at which this power ceases, as men of 80 years and over have been known to have begotten children. It may be supplemented through physiological approach. With advanced age, whatever, sterility is because of obliteration of canal of epididymis or vas deferens. This is a part of general obliteration process with the advancing age. This would prevent the collection and passage of seminal fluid but not its production. Thus, the situation could be styled as excretory rather than secretory.

MALFORMATIONS

Absence of male organ constitutes absolute impotence, as the act of sexual intercourse is physically impossible; in cases of partial amputation of penis, performance of sexual intercourse may be rendered impossible. Such a person, however, is not sterile if semen can be deposited into the vagina with the partially developed penis.

HYPOSPADIAS

It is characterised by the urethral orifice being situated on the undersurface, does not as a rule produce incapacity for sexual intercourse, unless it is associated with a marked deformity of the penis, which may interfere with its intromission into vagina. Spermatozoa, if deposited within the labia, can travel upwards into the vaginal canal owing to their motility.

EPISPADIAS

A deformity in which the urethra opens on the dorsum of the penis is extremely rare and is often associated with the rudimentary and stunted penis and extroversion of the bladder rendering sexual intercourse impossible.

The congenital **absence of testicles** produces sterility and impotence, but it is possible for a man to impregnate a woman after vasectomy if semen had already been present in the seminal vesicles before the operation; the person becoming permanently sterile after this stock of semen has been exhausted. **Monorchids** (i.e., those who have one testicle only) are physiologically quite potent, whereas **cryptorchids** (i.e., those who have undescended testicles) are usually, but not invariably, impotent and sterile.

LOCAL DISEASES OR INJURIES

A large hydrocele or scrotal hernia, elephantiasis, phymosis, paraphymosis and adherent prepuce may cause temporary impotence by mechanical obstruction to coitus, as these conditions can be remedied by proper surgical treatment. Marked diseases of penis or of the testicles, such as orchitis following mumps, syphilis, tuberculosis, etc. may lead to impotence or sterility or both. Inflammatory conditions of the testicles, epididymis, prostatic gland and seminal vesicles are the frequent causes of impotence and sterility. The ejaculatory ducts may be obliterated by chronic gonorrhoea, so that the seminal discharge may be prevented from flowing into the urethra.

Accidental injury to genitals or any other parts of the body resulting in impotence may lead to civil as well as criminal cases. Blows on the head or spine (cauda equina) may result in impotence, which may be temporary or permanent. A condition of temporary azoospermia (absence of spermatozoa in the semen) unattended with any loss of sexual power is observed in the individuals, who work in the X-ray department without proper protection.

GENERAL DISEASES

- Endocrine disturbances may produce sexual infantilism, rendering an individual impotent.
- Certain general diseases like diabetes, tuberculosis, chronic nephritis, prolonged priapism may produce temporary or permanent impotence, though the genital organs are apparently quite normal.
- Neurological diseases like tabes dorsalis, paraplegia, hemiplegia, syringomyelia, GPI, etc. may lead to impotence and consequential sterility. (Increased sexual activity may occasionally be noticed in early stages of these diseases.)
- Chronic indulgence to alcohol or certain drugs like morphine, heroin, cannabis, cocaine, tobacco or hormonal preparations may cause impotence and sometimes sterility.
- Chronic exposure to certain poisons like lead, arsenic or prolonged use of so-called aphrodisiac agents may also lead to both impotence and/or sterility.

PSYCHOGENIC CAUSES

These causes are most frequent, though mostly transient in nature. A temporary absence of desire for sexual intercourse may result from fear, anxiety, guilt sense, timidity, aversion, hypochondriasis, excessive passion and sexual over-indulgence. Persons with homosexual tendencies may be impotent. Sometimes, an individual may be impotent with one particular woman, but not with another. Dislike for the partner may preclude an erection and coitus, and this is of special significance in cases of nullity of marriage and divorce where the potency is to be ascertained in relation to the married partner only. As mentioned above, psychological causes outnumber all other causes, but the effects are mostly temporary and are overcome with adjustment and time.

The practice of coitus interruptus in sexual intercourse or the use of contraceptives is not an accepted reason for invalidating the act of consummation. It is also established that the conception is possible without penetration of the vagina (fecundation ab extra) and hence it does not establish consummation of marriage. Thus, the birth of child is not conclusive evidence that the marriage has been consummated.

CASE: PRESENCE/ABSENCE OF VIRILITY—LARGELY INFERENTIAL WITHIN USUAL LIMITS OF AGE

An alleged accused was brought to the GMCH on 10th February, 2001 for the medicolegal examination. The FIR was of the incidence of sexual assault committed by the accused to have taken place on 14th July, 2000 (i.e., alleged accused being put to examination after a lapse of about 7 months). The examining doctor gave the opinion as is usually given in such cases, i.e. "From the general physical and clinical examination, there is nothing to suggest that the individual is incapable of performing sexual intercourse". During cross-examination in the court, the defence counsel was assertive in pointing out to the honourable court that the opinion of the doctor pertained to 'the date' on which he examined the alleged accused (i.e., 7 months after the incidence) and therefore, the doctor was not in a position to opine as to the potency/impotency of the accused on the date of the incidence. Here, one is reminded of the Casper's statement, viz., "the possession of virility and procreative power neither requires to be, nor can be, proved to exist by any physician, but is rather, like every other normal function, to be supposed to exist within usual limits of age". Volunteering of this statement, probably, was the need of the hour.

Puberty is generally held to be attained at the age of 14 years, and by the term 'puberty' ought to be meant the attainment of 'virility' (not merely the power of coitus) because the latter power commences earlier than puberty or virility. And further, it continues for some period after procreative power has ceased. Cases of **virile power in elderly men** are well-known. The point was raised in the famous Banbury peerage case, where the putative father was 80 years of age at the date of the birth of the claimant. However, the judge ruled that there was no legal limit to the age when procreative power ceased. Spermatozoa have been reportedly found in a man of 96 years. Following removal of testes, procreative power is progressively lost. Physical causes of impotence afford the safest basis upon which to form an opinion. **Relative impotence** is sometimes present and may be due, among other causes, to neurasthenia, frigidity, or sexual perversion. **Impotence quoad hance** with a particular individual owing to invincible repugnance to the sexual act has been recognised (Venkateshwar Rao vs. Negamani AIR 1962 AP151; 54 Bom LR 725).

Causes of Impotence and Sterility in Females

The causes that prevent sexual intercourse and conception in females may be described under the similar heads as in the case of males, viz.:

AGE

As women are passive partners in sexual intercourse, so far impotence is concerned, theoretically no age in a woman's life is a bar from sexual intercourse. A woman is, of course, sterile in the extremes of her age (before attainment of puberty and after menopause). Puberty in the female usually commences at 13th or 14th year of life in India. A state of functional sterility exists in most young girls because of the initial menstrual cycles being

anovulatory. The age of onset of menstruation is quite variable, and factors like race, heredity, general health, environment, climate, diet and hygiene play a part. The average age of menarche in India is 13–14 years. The signs of puberty in a girl are the development of external and internal genitals, including a healthy ovulating ovary, the appearance of menstruation, growth of hair on the pubes and axillae and the development of breasts.

The possibility of pregnancy must primarily depend upon the function of ovulation. Generally, it can be assumed that in a woman, procreation power comes with the start of menstruation and stops with the stoppage of menstruation. However, it must be born in mind that menstruation and ovulation, though closely inter-related, yet do not occur at the same periods. It is because of this anomaly that there is possibility of conception for a limited time after cessation of menstruation. Likewise, it is also understandable that conception can occur before external manifestations of menstruation are apparent. Cases have been reported either side. Glaister reports a case of a woman, aged 47, who had ceased to menstruate for over a year and still became pregnant. Two other unusual cases have been reported. In one, there was cessation of menstruation at the age of 23, and in the other, the menstrual flow continued until the age of 75 years.

MALFORMATIONS

Congenital malformations such as the total occlusion of vagina, adhesion of the labia and the tough imperforate hymen are barriers to coitus and consequently lead to impotence. (These malformations are such as can be remedied by surgical interference.) Congenital defects of uterus (septate, infantile) and fallopian tubes (lack of patency) and ovary (cystic or fibrous with absence of primordial follicles) are the causes of sterility.

LOCAL DISEASES OR INJURIES

Local diseases of the genital organs may not ordinarily prevent sexual intercourse provided the vagina is normal. Inflammatory afflictions involving the cervix, uterus, ovaries and fallopian tubes may produce sterility. Painful and spasmodic contraction of the constrictor muscle of the vagina (vaginismus) may lead to temporary impotence. Rectovaginal fistula, ruptured perineum, disorders of menstruation, etc. may contribute towards sterility.

GENERAL DISEASES

General infective, metabolic and hormonal conditions may lead to sterility but not impotence. Occupational exposure to lead, X-rays without proper protection may lead to temporary or permanent sterility.

PSYCHOGENIC CAUSES

Hatred, fear, passion, neurotic temperament, etc. may produce a hysterical fit on an attempt at copulation and may thus render a woman temporarily impotent. Such women through reflex spasm of the levator ani, the perineal and the adductor muscles of the thigh develop distressing painful **vaginismus** whenever sexual intercourse is attempted. It may be considered as a psychosomatic illness. The spastic contraction of the vaginal outlet is completely involuntary reflex, stimulated by imagined, anticipated or real attempts at vaginal penetration. The aetiological factors may include (*i*) specific incidents of prior sexual trauma, (*ii*) stimulus derived from attempted heterosexual function by a woman with prior homosexual practice, (*iii*) secondary to dyspareunia and (*iv*) rarely, personal dislike or a general feeling of disgust at the idea of coitus. Psychotherapy is beneficial. As already mentioned, it is possible for a woman to be sterile or impotent with a particular man and quite the opposite with another.

Sterilisation

Sterilisation is a procedure to make a male or female person sterile, without any interference with potency.

TYPES

It may be of following types.

Voluntary

It is performed on married persons with the consent of both the husband and wife. It may be performed for the following purposes:

- **Therapeutic sterilisation:** This is performed to prevent danger to the health or life of the woman due to future pregnancy.
- **Eugenic sterilisation:** When the sterilisation is performed to prevent the conception of children who are likely to be physically or mentally defective, it is called eugenic sterilisation. The object is to improve the race by preventing the transmission of disease and hereditable defects.
- **Contraceptive sterilisation:** It is performed to limit the size of the family, i.e. for the purposes of family planning.

Compulsory

It is performed on a person compulsorily by order of the State. It may be carried out on mental defectives, sexual perverts, epileptics, and recidivist criminals. Some States in the USA have allowed this only under limited situations involving those mentally retarded persons who would be unable to appreciate the consequences of their acts or care for their children and who might pass on a hereditary form of retardation.

METHODS

Permanent

Vasectomy in male and tubectomy in female and exposure to deep X-rays in both sexes.

Temporary

- Observation of safe periods: Intercourse is avoided around the period of expected ovulation.
- Coitus interruptus: Ejaculation is done after withdrawing the organ from the vagina.
- Prevention of spermatozoa entering the uterus by use of diaphragm in female (outside the os) and use of condom by males.
- Use of spermicidal jellies or foam tablets.
- Use of intrauterine contraceptive devices (loop), etc.
- Oral hormonal pills.

GUIDING PRINCIPLES

To avoid legal complications, following precautions are required:

- Written concept of both wife and husband should be obtained for contraceptive sterilisation (it is advisable to have separate consent form for sterilisation. This form should, among other things, emphasise (*i*) that the procedure intends a permanent condition and (*ii*) that there is a small but real chance of failure, which may result in an unintended pregnancy).
- It is not unlawful if performed on therapeutic or eugenic grounds after obtaining due consent.
- The pills containing hormonal substances may rarely be harmful, and so necessary precautions have to be taken to avoid any complications.
- It is preferable to have a check-up after the vasectomy. The person should be advised to abstain from sexual intercourse for about 3 months or until the seminal examination shows absence of spermatozoa on two successive occasions. (The proper procedure would be to require two negative ejaculate specimens, 2 weeks apart, taken 2 months after the vasectomy followed by a third negative specimen 1 month later before the subject is declared sterile.)

MEDICOLEGAL ASPECTS

- Failure of contraceptive measure adopted by males may be a cause to suspect the wife to have sexual relationship with any other man, which may initiate litigations like divorce, legitimacy, disputed paternity, etc.
- Failure of sterilisation procedures is the most common basis for the birth-related actions called **'wrongful conception'** or **'wrongful pregnancy'** cases, viz., actions by parents against doctors for negligent contribution to unplanned pregnancies. The injury in these malpractice cases is the unplanned pregnancy, usually followed by the birth of a normal child. The plaintiffs generally attempt to prove that the unplanned pregnancy is due to the negligence of the doctor. **There are primarily four situations that result in wrongful pregnancy cases:**
  - A failed sterilisation or failure to ascertain the success of a sterilisation operation.
  - The ineffective prescription of contraceptives or counselling on contraception.
  - The failure to diagnose pregnancy in time for an elective abortion.
  - An unsuccessful abortion.

In a recent case [State of Punjab vs. Shiv Ram 2005 (6)], **the Apex Court over-ruling its earlier judgements held,** "Merely because a woman having undergone sterilisation operation became pregnant and delivered a child, operating surgeon or his employer could not be held liable for compensation on account of unwanted pregnancy or unwanted child. The cause of action for claiming compensation in cases of failed sterilisation operation arises only on account of the negligence of the surgeon and not on account of the birth of the child. Having gathered the knowledge of conception in spite of having undergone sterilisation operation if the couple opts for bearing the child, it ceases to be unwanted child, the compensation for maintenance and upbringing of such child cannot be claimed".

Artificial Insemination

Artificial insemination (AI) may be considered as the deposition of semen into the vagina, cervix or uterus by instruments to bring about pregnancy, which is not attained or is unattainable by sexual intercourse. It has been recommended by some who regard it as the ideal method of preserving the stability of marriages, which are childless and are thereby threatened. However, it is fraught with certain legal implications, as there is no specific provision for or against artificial insemination in the Indian Law.

TYPES

It may of following types:

Artificial Insemination Homologous (AIH)

When the husband's semen is biologically normal but he either cannot pour the same into the wife's genital tract by way of intercourse or due to some defect with the cervical opening of the wife or some defect in the vagina, living sperms cannot enter inside the uterus. Semen from the husband is collected by way of masturbation and deposited into the uterus by instrumentation.

Artificial Insemination Donor (AID)

When the defect is in the seminal fluid of the husband, semen from some other healthy suitable male is used and introduced inside the vagina or the uterus of the wife.

Artificial Insemination Homologous Donor (AIHD)

A mixture of husband's semen as well as that of a donor (pooled semen) is used in cases where the motile spermatozoa count in the husband's semen is poor though present in the

semen. The advantage of this method is that both the husband and the wife may believe that the husband's spermatozoa could have brought about the fertilisation and the child thus might have been the product of the husband.

PROCEDURE

The usual practice is to deposit 1 ml of semen above the internal os by means of a sterile syringe, at or about the time of ovulation, i.e. 14th day after menstruation. The semen should be collected by masturbation, preferably after a week's abstinence, and used within about 2 hours. The timing of insemination is important as the lifespan of the spermatozoa in the female reproductive tract is short. Because of the problem of timing, insemination on several successive days in the month increases the chances of pregnancy. The use of frozen semen for AID is becoming increasingly common. This is done by addition of glycerol, slow cooling and rapid deep freezing, the so-called cryopreservation.

INDICATIONS

- When the husband is impotent but fertile.
- When the husband is sterile.
- Where there is Rh incompatibility between husband and wife.
- When the husband is suffering from hereditary disease.

GUIDING PRINCIPLES

As has already been mentioned, there is no specific law in India regarding artificial insemination; legal problems may arise on the allied aspects of the procedure adopted for the purpose. To avoid the possible problems, certain procedural precautions are recommended:

- Knowledge and full informed consent of both spouses are essential. The consent must be in writing.
- The identity of the donor and recipient must not be revealed to each other nor should the donor know the result of insemination.
- The donor must be below the age of 40, not related to either spouse, and should have children of his own.
- The donor must be in robust health both physically and mentally. He should not be suffering from any hereditary or familial disease. The medical examination should exclude such diseases as tuberculosis, diabetes, epilepsy, endocrine dysfunction and Rh incompatibility, etc. In the present scenario, he must be screened for AIDS, initially at the time of donation of semen and be retested after 3 months. The semen should be frozen and not used until the result of the second test is known.
- There should be parity of race, religion and as much as possible the morphological appearance between the donor and the husband of the recipient woman.
- The donor should give a written declaration that he will not prefer parenthood claim for any child on the ground of donation of semen.
- The physician who administers the artificial insemination should avoid delivering the child.
- The wife of the donor must agree for donating the semen for the purpose of insemination, and the semen should be obtained from an act of masturbation.
- A nurse/female attendant should be present when the insemination procedure is being carried out.

LEGAL PROBLEMS OF ARTIFICIAL INSEMINATION

Artificial insemination with the semen of the husband is justifiable and unobjectionable, since the child is actually the biologic product of both husband and wife. However, it does not constitute evidence of proper consummation of marriage.

When for any reason, AIH is not possible and artificial insemination is done with the semen of an unknown donor (AID), the position is quite different from the above. The legal issues arising therefrom may be as follows:

- **Danger of litigation:** There exists a danger of litigation against the doctor following the birth of a defective child. To avoid this, the donor must be screened for chromosomal studies for possible genetic defects.
- **Adultery:** The recipient cannot be held guilty of adultery because there is no physical union in the form of coitus (Section 497 IPC requires sexual intercourse as necessary ingredient of adultery). For the same reason, the donor is not guilty of adultery.
- **Legitimacy:** The husband is not the actual father of the child and, therefore, the child is illegitimate and cannot inherit property. Any attempt to conceal this fact by registering the husband as the father amounts to perjury.
- **Nullity of marriage and divorce:** Mere AI is not a ground for nullity of marriage or divorce because sterility is not a ground for it. However, if AI is due to impotence of the husband, it is a ground. When AI is done due to impotence of the husband, the wife may ask for nullity or divorce even if a child is born out of AI. The maintenance and custody of the child in the event of divorce would raise complex issues.
- **Natural birth:** If a child is born in the natural course sometime after the birth of child through AI, the status of the child born through AI remains illegitimate and the status of the naturally born child remains legitimate.
- **Incest:** There is risk of incestuous relationship between the offspring and children of the donor.
- **Posthumous child:** The human semen can be preserved by freezing. The possibility that human semen can be preserved does not create additional problems where the semen used is that of the donor, but some complex legal problems may arise if the semen used was that of her husband. It may

be argued (where the mother has not remarried after death of her husband) that such a 'posthumous child' is legitimate because the child, although not conceived during the continuance of marriage, may be regarded as a child of the marriage. The practice of inseminating a woman with her husband's semen after his death is not yet common place. Such developments may have to be faced in the near future.

(The majority view today is to consider the child born through AID to be legitimate, based on public policy considerations and the desire not to stigmatise innocent children.)

In Vitro Fertilisation (Test Tube Baby)

In vitro fertilisation (IVF) is now an accepted alternative method of conception. The sperm and ovum are allowed to incubate outside the human body (i.e., in the laboratory and therefore popularly called Test Tube Baby) and the resulting embryo is then implanted into the uterus. Legally, the process is comparable to AIH. New techniques allow drugs to cause multiple ovulations and the laparoscopic removal of a number of ova, which can then be fertilised in the laboratory (in vitro fertilisation) and implanted into the uterus for the remainder of gestation.

INDICATIONS

- Absent or nonpatent fallopian tubes.
- Inadequate motile sperm count.
- Hostile cervical mucus.
- Refractory endometriosis.
- Unexplained infertility.

This allows the following permutations:

- The woman's own ova to be fertilised by her husband's sperm and re-introduced into her uterus.
- The woman's own ova to be fertilised by a donor's sperm and returned to her own uterus.
- The woman's own ova to be fertilised by her husband's sperm and returned to another woman's uterus ('surrogate motherhood').
- The woman's own ova to be fertilised by a donor's sperm and returned to a surrogate woman's uterus.
- An infertile woman may have another woman's ova implanted in her, fertilised either by her husband or by a donor.

CRYOPRESERVATION OF EMBRYOS AND GAMETES

The preservation of embryos and gametes by deep freezing is an integral part of IVF. The first successful births after freezing, thawing, and implantation of human embryo were in 1984 by groups in Australia and the Netherlands. Cryopreservation has now become routine for preserving multiple embryos for use in subsequent cycles in foreign countries. (Prior use of 'fertility drugs' allows more ova to be harvested than are needed for implantation. When fertilised in vitro, this produces **spare embryos,** which can be deep frozen for an indefinite period before use. 'Spare embryos' are also available for medical research into congenital and familial diseases.) Some special issues in cryopreservation have been focused in the court cases in foreign countries. For example:

- A wealthy couple died in an airplane crash, leaving millions of dollars and two frozen embryos in Australia (Australians Reject Bid to Destroy Two Embryos, *New York Times*, 24th October, 1984).
- A Tennessee couple who had cryopreserved seven embryos separated, and the husband filed a court action seeking to enjoin the wife from having embryos implanted against his will. The Tennessee Supreme Court ruled that the biological father of the cryopreserved embryos had an absolute right not to become a birth father against his will (Davis vs. WL Tenn Cir; 1989).

'Wrongful Birth' and 'Wrongful Life' Cases

The time is not far, when the claims of liability may be made for negligence resulting in a defective child being born when that child is unwanted because of the defect. Such a claim may be referred to as **'wrongful birth'** claim. A doctor may be liable when the defect is foreseeable or discoverable, and he fails to foresee or test for the defect. The parents may argue that they would have prevented the birth of the child through the use of contraception or abortion, had they been properly informed and advised.

In the **'wrongful life'** claim, unlike a wrongful birth suit (in which the plaintiffs are the parents of the disabled child), a suit claiming 'wrongful life' is brought by or on behalf of the disabled child. Essentially, the child or his representative argues that he or she should never have been born that nonexistence is preferable to the life of an individual so handicapped by a congenital disability.

28 CHAPTER

Nullity of Marriage, Divorce and Legitimacy

After going through this chapter, the reader will be able to describe: Circumstances for void and voidable marriage | Legitimacy of children of void and voidable marriages | Grounds for divorce | Alternate relief in divorce proceedings | Medicolegal issues relating to legitimacy | Some important considerations

Void and Voidable Marriages

Sections 11 and 12 of the Hindu Marriage Act (HMA) 1955 deal with void and voidable marriages, respectively. A **void marriage** is a nullity from the inception, i.e. the marriage is void ab initio and the decree passed under Section 11 does not add anything to it except declaration is made as to the nullity of already void marriage. The words "be so declared by a decree of nullity" amply throw light on this aspect. A **voidable marriage** is one that remains valid until annulled by the Court, and Section 12 lays down grounds for this purpose.

CIRCUMSTANCES FOR A VOID MARRIAGE

Any marriage solemnised after the commencement of the Act shall be null and void and be so declared by a decree of nullity if it contravenes any one of the conditions enumerated as under:

- Marriage with another without dissolution of earlier marriage, i.e. another marriage during the lifetime of one's spouse **(bigamy)** is void ab initio and is punishable under Section 494 and 495 of IPC. The second wife, whose marriage is void, is not entitled to claim even the maintenance under Section 125 of CrPC.
- Marriage within **the degrees of prohibited relationship** unless the custom or usage governing each of them permits a marriage between the two. For this purpose, certain terms have been described in the Act, viz:
  - Full blood, i.e. where two persons share both parents or grandparents;
  - Half blood, i.e. where two persons share only one male parent or grandparent; and
  - Uterine blood, i.e. where two persons share only one female parent or grandparent. In Hindi, word '*sagaa*' is used for many of the 'full blood' and '*sautela*' for 'half blood' relations. The marriage between full blood, half blood or uterine blood relatives is prohibited under the Act.
- Marriage within **sapinda relationship**, i.e. the relationship extending as far as the third generation (inclusive) in the line of ascent through the mother, and the fifth (inclusive) in the line of ascent through the father, the line being traced upwards in each case from the person concerned who is to be counted as the first generation. Many of the relations that fall within sapinda relationship are also covered under degrees of prohibited relationship.

CIRCUMSTANCES FOR VOIDABLE MARRIAGE

Nonconsummation of Marriage Owing to Impotence of the Respondent

The 'concept of marriage', in a broad sense, implies a legally and socially sanctioned union of a man and woman that accords status to them as husband and wife and legitimacy to their offsprings. It involves certain rights and duties between the parties entering into wedlock and presupposes on the part of each the lawful use of the body, or in other words, capability of the fulfillment of the act of physical union leading to **consummation of marriage**. Erection and penetration (without emission) although not constituting sexual intercourse in the full sense of the word, but were considered sufficient to consummate marriage [R vs. R (1952) 1 A11 ER 1194]. A couple of cases amply highlight the point: (*i*) in order to consummate marriage, a normal sexual intercourse must take place but the degree of sexual satisfaction obtained by the parties from each other is not relevant (Rajinder Kaur vs. Manmohan Singh AIR 1972 P&H 142); (*ii*) where complete coitus is established, the discharge of semen in wife's body is not necessary condition of consummation (Moina Khosla vs. Amardeep Khosla AIR 1986 Delhi 399).

Unsoundness of Mind

In this context, law provides that the marriage is voidable if at the time of marriage, either party

- is incapable of giving a valid consent in consequence of unsoundness of mind; or
- though capable of giving valid consent, has been suffering from mental disorder of such a kind or to such an extent as to be unfit for marriage and procreation of children; or
- has been subject to recurrent attacks of insanity.

Consent by Force, Fraud or Misrepresentation

The words 'force' and 'fraud' have not been defined in the Act. For this purpose, 'force' implies threat of violence, physical juxtaposition or other kind of injury (including mental) to the party (giving consent) or his near one. And to constitute 'fraud', there must be deliberate concealment of material fact or some projection or getting projected for the purposes of obtaining consent. Examples may include the following:

- Concealment of factum of annulment of first marriage
- Impersonation at the time of selection for marriage, i.e. person shown was different than the person actually married to the petitioner
- Misrepresenting oneself as graduate in a matrimonial advertisement but actually not being so, etc. However, condonation after disclosure of fraud disentitles the party to the relief of getting marriage annulled

Pregnancy of the Respondent at the Time of Marriage

The conditions necessary for this ground include the following:

- The pregnancy was due to some person other than the petitioner
- The petitioner was ignorant of the fact at the time of marriage
- Petition is brought within 1 year of marriage
- Marital intercourse with consent of the petitioner did not take place after the discovery of such pregnancy. The burden of proof as to pre-nuptial pregnancy lies on the husband and it should be proved beyond reasonable doubt (Nand Kishore vs. Munni Bai AIR 1979 MP 45).

LEGITIMACY OF CHILDREN OF VOID AND VOIDABLE MARRIAGES

Where the marriage is declared void pursuant to decree arising out of petition presented under Section 11 or 12 of the HMA, 1955 (i.e. Section dealing with void and voidable marriages, respectively), the children of such marriages would be legitimate, whether begotten or conceived before the decree is made. Further, they would also be entitled to inherit property of their parents. This has been enacted by the legislature to put an end to a great social evil.

Furthermore, the law even extends protection to children resulting from live-in relationships. The Protection of Women from Domestic Violence Act (2005) extends equal benefit to women in live-in relationship and married women. The Apex Court, in a couple of judgements observed, "The children born out of such a relationship will no more be called illegitimate. The law inclines in the interest of legitimacy and thumbs down 'whoreson' or 'fruit of adultery'" [Section 2 of the Act, amongst other definitions, explains 'respondent' as any adult male person who is, or has been, in a domestic relationship with the aggrieved person and against whom the aggrieved person has sought any relief under the Act provided that an aggrieved wife or a female living in relationship in the nature of a marriage may also file a complaint against a relative of the husband or the male partner]. Table 28.1 lists the grounds for void and voidable marriages.

Divorce

Before commencement of the Hindu Marriage Act (HMA) 1955, divorce was unknown for Hindus as they believed marriage to be a sacrament, a holy union forever. However, due to influence of Islam and Christianity, and also with the change of social and educational standards, need for divorce was felt under the Hindu Law also. Section 13 of HMA, 1955 provides for the grounds on which divorce can be sought. Even in these cases too, a duty is cast on the court under Section 23 (2) to make every endeavour to bring about reconciliation between the parties.

The word 'divorce' has been derived from the word 'diverse'. Divorce is another name for **dissolution of marriage**, i.e. parties cease to be husband and wife after the decree of divorce is passed by the Court. For Hindus, it is achieved only as a consequence of proceeding under the Law, whereas for Muslims, it may be achieved as a consequence of proceedings at Law or may result from the acts of parties. Various grounds for divorce as mentioned under Section 13 of HMA, 1955 appear as follows:

ADULTERY

The word 'adultery' has not been used under the law for divorce and instead the expression 'voluntary sexual intercourse with any person other than his or her spouse' finds place therein. The expression means 'an act of consensual sexual intercourse between a married person and another person of opposite sex who is not his or her spouse during the subsistence of former's marriage'. Either party to the marriage may present petition of divorce on the ground of adultery of the respondent. General evidence of ill reputation of the husband or that of wife or that he or she was seen with a doubtful person of opposite sex, does neither prove nor indicate probability of adultery (D Handerson vs. D Henderson, AIR 1970 Mad 104). In a case for

Table 28.1 Grounds for Void and Voidable Marriage

Void marriage	Voidable marriage
Here, the marriage is void *ab initio*, i.e. it is null from the time of its inception in the eyes of law • Either party has a spouse living at the time of the marriage (**bigamy**), i.e. marriage with another without dissolution of earlier marriage is void from the very inception and is punishable under Section 494 and 495 of IPC. • The parties are within **prohibited degree of relationship** (consanguinity) to each other unless custom or usage governing each of them permits a marriage between the parties. • The parties are within **sapinda relationship** to each other, i.e. the relationship extending as to the third generation (inclusive) in the line of ascent through the mother and as to the fifth (inclusive) in the line of ascent through the father. (Many of the relations that fall within sapinda relationship are also covered under degrees of prohibited relationship).	Here, the marriage remains valid until a decree of nullity is passed by the court • **Nonconsummation** of marriage owing to the impotence of the respondent • **Unsoundness of mind** because of which either party was incapable of giving valid consent at the time of marriage **or** the mental disorder was of such a kind and to such an extent as to make the party unfit for marriage and procreation of children **or** either party has been suffering from recurrent attacks of insanity. • **Consent** of the either party to the marriage was obtained through force, fraud, or misconception of facts. • **Pregnancy** of the respondent at the time of the marriage due to some person other than the petitioner, and the petitioner was ignorant of the fact at the time of marriage.

Note: Age for marriage as prescribed under the law is that the bridegroom should have completed the age of 21 years and the bride, the age of 18 years at the time of marriage. Any marriage solemnised in contravention of this condition attracts punishment under the Prohibition of Child Marriage Act 2006. The marriage is **voidable** at the option of the contracting party to the marriage who was a 'child' at the time of marriage. **Additionally,** in case of female, if the marriage had been solemnised before she attained the age of 15 years and she repudiates the marriage after attaining that age but before attaining the age of 18 years, a decree of **divorce** can be granted at the instance of the bride.

divorce based on adultery, the adulterer is a necessary party and ought to be made second respondent.

CRUELTY

The word 'cruelty' under this provision has been used in the context of human conduct and behaviour in relation to matrimonial duties and obligations. It may be explained as a willful and unjustifiable conduct of such a nature as to cause danger to life, limb or health of another and also includes mental torture. Whether an act constitutes cruelty or not is a matter of fact. Cumulative conduct should be taken into consideration in explaining the cruelty. Sentiments, ambitions and aspirations are also relevant factors in this regard (Rajani vs. Subramanium, AIR 1990 Ker 1: 1989 Mat LR 510).

DESERTION

The expression 'desertion' in context of matrimonial law represents a legal conception and is very difficult to define. The essence of desertion is the forsaking and abandonment of one spouse by the other without reasonable cause and without consent or against the wish of the other. One of the essential ingredients is the factum of separation and the intention to bring cohabitation permanently to an end (animus deserendi). The relief under this ground is available only if desertion has been for a continuous period of not less than 2 years immediately preceding the presentation of the petition. Where the cause of desertion is the misconduct of the petitioner, it is called as 'constructive desertion' and in such a case, the party is not permitted to take advantage of his/her own fault. Actually, it is he/she who has constructively deserted the partner (Ashok Kumar vs. Shabnam, AIR 1989 Delhi 121). Similarly, if there is a strong evidence of demand of dowry compelling the wife to stay with her parents, there is no desertion from the side of the wife. Desertion also includes willful neglect of the petitioner by the other party to the marriage.

APOSTASY

Change of religion has been provided as a ground for divorce as also a ground for judicial separation under the HMA, 1955. However, change of religion does not automatically dissolve the marriage performed between the two parties and does not bring to an end the civil obligations or matrimonial bond. The provision clearly institute that the ground can be claimed by the other party but not by the party who himself converted to another religion.

UNSOUNDNESS OF MIND

A person incapable of managing himself and his affairs including the problems of society and of married life can be said to be a person of unsound mind (Kartik Chandra vs. Manju Rani, AIR 1973 Cal 545). Two subgroups as provided under this ground include (*i*) unsoundness of mind of incurable nature and (*ii*) mental disorder that does not amount to unsound mind can be ground for divorce only if it has been continuous or intermittent and is of such a kind and to such an extent that the spouse seeking divorce cannot be reasonably expected to live with the person suffering from the disorder. Where there was allegation by the husband as to wife suffering from mental

disorder and the wife though denying such allegation but did not cooperate with committee of doctors, the adverse inference could be drawn against the wife (T Hari Kumar Naidu vs. Prameela, AIR 2001 AP 46).

LEPROSY AND VENEREAL DISEASE

Section 13 (1) (iv) and (v) lay down these diseases as ground for applying for divorce. **The former reads as** "has been suffering from a virulent and incurable form of leprosy", and **the latter as** "has been suffering from venereal disease in a communicable form". The words "has been suffering from" denote that the disease must be present since some length of time. In context with the duration, Section 14 of the Act provides that "it shall not be competent for any court to entertain any petition for dissolution of a marriage by a decree of divorce unless at the date of presentation of the petition 1 year has elapsed since the date of the marriage". Here, some terms need to be mentioned, namely: **Virulence** implies the power and degree of pathogenecity possessed by an organism to produce the disease. Virulent, therefore, implies extremely malignant (showing widespread dissemination of agent/organism in the tissues and therefore carrying extremely high pathogenecity) or violent or noxious. **Incurable** refers to which is not capable of being arrested by commonly prevalent/known treatment. [It is pertinent to mention that leprosy is presently a curable disease and the patient no longer remains infectious to others after the first dose of multidrug therapy (MDT). Early detection and treatment with MDT averts disability]. **Infection** refers to the state/condition in which the body or a part of it is invaded by a pathogenic agent (organism or virus) that, under favourable conditions, multiplies and produces injurious effects. Infectious, therefore, is the one which is capable of being transmitted with or without contact, i.e. may or may not be contagious. **Communicable** disease implies a disease the causative agent of which may pass or be carried from one person to another directly or indirectly. **Contagious** disease implies a disease that is communicable by contact (direct or indirect) with an individual suffering from it or with some secretion of such an individual, or with an object touched by him/her. **Venereal** disease is an older term for sexually transmitted disease and encompassed those diseases when laws were written, namely: syphilis, gonorrhoea, chancroid, granuloma inguinale, lymphogranuloma venereum. Evolution to the term 'sexually transmitted diseases' (STDs) reflects recognition of the increasing number of infections or conditions that are sexually transmitted, i.e. it is a more inclusive term than venereal disease, which includes conditions as already mentioned plus conditions like AIDS, pelvic inflammatory diseases, and other conditions such as trichomoniasis, genital candidiasis, genital herpes, genital warts, and bacterial vaginitis, etc. [Since mental and physical health is of prime importance in a marriage (as one of the objects of the marriage is the procreation of equally healthy children), that is why in every system of matrimonial law it has been provided that if a person was found to be suffering from venereal disease in a communicable form, it will be open to the other partner in the marriage to seek divorce.]

RENOUNCING THE WORLD

Legal presumption of death of a person may be drawn from the fact that he has not been heard of as being alive **for a period of 7 years** or more by those who would naturally have heard of him. A spouse may seek divorce if the other spouse has not been heard of as being alive for such a period or more. However, such a presumption is rebuttable.

ADDITIONAL GROUNDS FOR WIFE

Additional grounds based on which a women can seek divorce include the following: (*i*) Where the husband, since the solemnisation of the marriage, has been guilty (i.e. convicted by the Court) of rape, sodomy or bestiality, a petition for decree of divorce can be filed by his wife against him. (*ii*) Where a decree of maintenance against the husband has been passed by the Court, and there has been absence of cohabitation between the husband and wife for more than 1 year, the wife can apply for divorce under such a situation.

DIVORCE BY MUTUAL CONSENT

Section 13B lays down various conditions under which divorce can be sought through mutual consent as enumerated below:

- That the parties to the marriage are living separately for a period of 1 year or more [the expression 'living separately' connotes not living like husband and wife. It has no reference to the place of living. The parties may live under the same roof and yet they may not be living as husband and wife (Sureshta Devi vs. Om Parkash, AIR 1992 SC 1904)].
- That they have not been able to live together.
- That the parties should have mutually agreed that their marriage be dissolved.
- That the consent should not be withdrawn by either of the parties within six months or till hearing of the case whichever is earlier.

ALTERNATE RELIEF IN DIVORCE PROCEEDINGS

Section 13A of the HMA, 1955 empowers the Court to award decree of **judicial separation** instead of divorce even after the ground for divorce is proved so that under particular circumstances of a case, real justice could be done to the parties leaving room for reconciliation between them.

Legitimacy

Under Section 112 of IEA, there exists a **presumption in favour of legitimacy of a child** born during the continuance

of a valid marriage between his mother and any man or within 280 days after its dissolution, the mother remaining unmarried. The presumption can only be rebutted if it is shown by competent evidence that the parties to the marriage had no access to each other at any time when the child could have been begotten. It is not true that just because a child was born beyond a period of 280 days, it ipso facto becomes illegitimate. Some of the evidence that can prove beyond reasonable doubt that the child is not legitimate is necessary, as the period of gestation is not exactly fixed either by the medical science or by the law. **An illegitimate or bastard child** is one who is born out of wedlock or not within a competent period after the cessation of the relationship of man and wife, or born within wedlock when procreation by the husband is not possible because of congenital or acquired malformation or illness. **Circumstances under which the question of legitimacy may arise are discussed below:**

- **Inheritance:** A legitimate child born during lawful wedlock can inherit the property of its father. According to the Law of England, a monster, which does not have the shape of mankind, is incapable of inheriting, but there is nothing specific on this point in Indian Law.
- **Affiliation cases:** These are cases that are brought before the Court for fixing the paternity of an illegitimate child upon a certain individual. As per law, the father, under Section 125 of the CrPC, has to support his legitimate child who is unable to maintain himself, irrespective of age. In such cases, blood grouping tests may be necessary. The Court can give directions for the blood grouping if one of the parties involved in the proceedings made an application; if either party refuses to comply with the directions, the Court is empowered to draw any inference from this failure, if it thinks proper to do so under the circumstances. The blood grouping tests are useful to furnish evidence of paternity negatively. In addition to blood tests, the factors like resemblance of features, colour, voice, manner, complexion, etc. may also be taken into consideration. However, children may not resemble their parents at all, and therefore, the absence of likeness of features and colour of the hair or eyes or of transmitted peculiarities does not disprove paternity nor prove illegitimacy. Moreover, cases of **atavism** occur in which the child does not resemble its parents but resembles its grandparents. The Magistrate may make a monthly allowance of any sum depending upon the circumstances of the case for the maintenance of such a child. In determining the amount of maintenance, luxury is not to be taken into consideration, but only the necessities of life like food, clothing and lodging, etc. are to be given priority.
- **Suppositious children:** A suppositious child means a fictitious child. A woman may substitute a living male child for a dead child or a living female child born of her, or may feign pregnancy as well as delivery and subsequently produce a living child as her own, when she wants to extort money or to divert succession to property. Such cases may occur when succession to large estate is involved or when money is to be extorted by blackmailing.

Legal Consequences of Fecundation Ab Extra

Even if a wife conceives a child by the husband through fecundation ab extra, i.e. by the mere deposit of semen on the vulva without penetration, this does not have effect on consummation of marriage. Clarke vs. Clarke (1943, 2 A11, ER, 540) was the first case wherein the Judge ruled in favour of the husband and granted a decree of nullity accepting the evidence of "non-consummation of marriage" on the evidence led by the husband and holding that the birth of the child did not in itself establish consummation. Further that "fecundation ab extra" was an established medical fact previously noted by the courts.

29 CHAPTER

Forensic Psychiatry

After going through this chapter, the reader will be able to describe: Forensic psychiatry | Various 'terms' in the Mental Health Act | Signs/symptoms of mental disturbance with their medicolegal importance | Mental retardation | Psychosis and neurosis | Association of cerebral tumours, pregnancy and epilepsy with psychosis | Personality disorders | Diagnosis of mental illness | True and feigned mental illness | Restraint of the mentally ill | Civil and criminal responsibility of the mentally ill | Criminal responsibility for offence committed during intoxication | Sexual perversions/deviations (paraphilias)

Health is defined as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO, 1948). **Mental health** has been defined as, "a state of balance between the individual and the surrounding world, a state of harmony between oneself and others, a co-existence between the realities of the self and that of other people and that of the environment [Sartorius N (1983), Bull-WHO]. This may amply be substantiated through the words of Kandel (1998), "There can be no changes in behaviour that are not reflected in the nervous system and no persistent changes in the nervous system that are not reflected in structural changes on some level of resolution. Everyday sensory experience, sensory deprivation, and learning can probably lead to a weakening of synaptic connections in some circumstances and a strengthening of connections in others."

In general medicine, diagnosis, treatment and prognosis are comparatively better standardised as compared to psychiatry due to obvious reasons. The road to current version of the International Classification of Diseases (ICD-10 of which chapter V (F) and sub-chapters deal with psychiatric classification of diseases) and the American National System of Psychiatric Classification in the form of current version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) can be traced to many developmental landmarks, which, of course, is not in the purview of this short treatise. These two classifications are mostly similar because American diagnosticians also took part in designing the new ICD-10 classification. However, the two systems are still not fully congruent. For example, social and occupational criteria have been avoided in ICD-10 because of the difficulty of equating these criteria between various cultures, but they have been used in DSM-IV, which is a national classification. DSM-IV is only in one version and more useful in diagnostic criteria for research purposes.

Mental Health Act, 1987

Indian Lunacy Act (1912) has no relevance today, as the advances in medical science have dictated corresponding advancement in the related legal provisions (insanity was formerly thought to be affected by 'the moon', hence the term 'lunacy'—in Latin, *luna* means moon). The Act was amended in 1987 (and was termed as Mental Health Act, 1987) with the aim to consolidate and amend the law relating to the treatment and care of mentally ill persons and to make better provision with respect to their property and affairs. Certain terms as defined in the Act are described in Table 29.1.

Medical officer means a Gazetted Medical Officer in government service appointed by State Government.

Medical officer in charge is a Medical Officer who for the time being is in charge of a psychiatric hospital or a nursing home.

Medical practitioner means a person with recognised medical qualification under the provisions of the Act.

Mentally ill person is a person suffering from mental disorder other than mental retardation, needing treatment.

Mentally ill prisoner is a mentally ill person, ordered for detention in a psychiatric hospital, jail or other safe custody.

Psychiatric hospital or nursing home is a hospital for the mentally ill persons maintained by government or private party with facilities for outpatient treatment and registered with appropriate Licensing Authority. Admitting a mentally ill to a general nursing home is an offence.

Psychiatrist is a medical practitioner possessing a post-graduate degree or diploma in psychiatry recognised by Medical Council of India.

Table 29.1 New Terms Used in the Mental Health Act

Outdated terms	New terms
Asylum	Psychiatric hospital/nursing home
Lunatic	Mentally ill person/mentally challenged
Criminal lunatic	Mentally ill prisoner

Reception order means an order for admission and detention of a mentally ill person in a psychiatric hospital or a nursing home.

Relative includes any person related to a mentally ill person by blood, marriage or adoption.

Various Signs/Symptoms with Medicolegal Importance

In physical medicine, the emphasis is on the pathology of various organs and symptoms and signs consequential to such pathology. In psychiatry, one cannot always attribute the symptoms to the pathology in the brain or any of the other organ systems. Apart from the changes in the 'internal milieu', the psychosocial factors, too, modify human behaviour. The symptoms in psychiatry are the experiences as narrated by the patient, which cause distress to the individual. A cluster of such symptoms may indicate signs of an illness.

SIGNS/SYMPTOMS RELATED TO DISTURBANCES OF CONSCIOUSNESS AND HIGHER CEREBRAL FUNCTIONS

Level of sensorium of the patient and various higher functions may have to be evaluated in relation to certain medicolegal issues. The following description may help in this context:

Consciousness

It may be defined as the awareness of the self and environment. Its knowledge may be gathered from the following:

Confusion: Here the thinking process lacks clarity and coherence. It is seen both in organic and functional disorders.

Clouding of consciousness: There is mild global impairment of cognitive (intellectual) functions in addition to decreased awareness of environment.

Delirium: There is impairment of consciousness along with perceptual and affective (emotional/mood) abnormalities. A delirious patient usually experiences auditory and visual hallucinations and is irritable and restless. Thought content becomes irrelevant or inconsistent. In the early stage, the patient is restless, uneasy and sleepless. In the later stages, he may lose self-control and become excited/impulsive or violent and may commit suicide. Such a person may not be responsible for his acts depending upon the degree of disturbed consciousness. Usually seen in physical diseases, where there is continuous high temperature and sometimes due to overwork, mental stress or drug intoxication.

Twilight state is described as interruption of the continuity of consciousness. Consciousness is clouded and sometimes narrowed (narrowing of consciousness implies restricted awareness of a person towards environment). Despite disordered consciousness, the patient may be able to perform certain actions like driving, walking around, etc., followed by amnesia for the state/event. Twilight states may occur in epilepsy, brain trauma, dissociative disorders, alcoholism, etc. ('Mania a potu' has been described as a situation where a person reacts extremely to small amounts of alcohol due to development of twilight state. Such patients, often have an increased vulnerability due to pre-existing organic brain pathology.) Twilight state may lead to violent behaviour.

Oneiroid state is described as narrowing of consciousness together with multiple scenic hallucinations. Such a state may occur in schizophrenia, but may also be observed in patients under excessive care and dependent upon others. The atmosphere is perceived as strange and dream-like. Unlike twilight states, the contents of oneiroid states are often remembered.

Fugue: A state of altered awareness during which an individual forgets part or whole of his life, leaves home and wanders away. He has a state of complete amnesia for the period of wandering. The fugue is commonly a hysterical phenomenon, serving as a means of escape from some unpleasant situation. Fugues may also occur in depressive illness, schizophrenia and epilepsy.

Coma: There is no reaction to any painful stimuli.

Stupor: Characterised by akinesis and mutism, with relative preservation of consciousness.

Orientation: It may be described as one's awareness in relation to time, place and person. Loss or impairment of orientation does not occur in all the fields simultaneously. In organic psychosis, it is temporal (time) and the situational orientation, which is disturbed first, followed later by disturbance in the orientation to place. Loss of orientation to one's own identity is the last one to be affected.

Attention and concentration: Attention means focusing of consciousness on an experience, whereas concentration is the maintenance of the focus. Narrowing of attention can be seen in cases of anxiety and depression. It is also impaired in schizophrenics when they are absorbed in their hallucinatory experiences or distracted by their internal cues. Fluctuations in attention and concentration are often seen in mania. Fatigue and drug intoxication can alter the attention span.

Memory: It is the ability to store and recall information. For information to be stored, it has to be registered after proper perception. Therefore, registration, retention and recall are the three important components of memory. **Recent** memory is the ability to retain new material over a short span of time, extending from 5 minutes to a few hours, e.g. the patient may be asked what he had taken in the breakfast or

lunch, the names of the persons he met a short while ago, etc. **Remote memory** is the ability to recall information regarding events that happened quite a long time before and is tested by means of the ability to recall details regarding one's hometown, education, marriage, etc.

Amnesias: These can be organic or functional. Organic amnesia can occur due to focal or diffuse brain damage, the lesions being in the hippocampal and diencephalon structures. In these amnesias, it is the recent memory, which is impaired. There is difficulty in learning new material. Examples of these amnesias include retrograde and anterograde amnesias after head injury and Korsakoff psychosis. **In retrograde amnesia,** the memory for events just before the head injury is lost. Sometimes, it may cover a period of months. As time progresses, this retrograde gap gradually shrinks. **In anterograde amnesia,** the patient fails to remember any new material after the head injury. In Korsakoff's psychosis, apart from recent memory disturbances, **'confabulation'** is noticed wherein the patient gives a coherent but false account of some recent events. In this psychosis, the lesions are in the dorsal nuclei of the thalamus. In amnesia due to diffuse brain damage, the memory is a part of global deterioration in the cognitive functions. It is the recent memory, which is impaired, while the remote memory is intact; but over a period of time, this remote memory lacks details. The amnesia seen in dementia is of this type.

SIGNS/SYMPTOMS RELATED TO DISTURBANCES OF THINKING

Thinking refers to the ideational components of mental activity—processes used to imagine, appraise, evaluate, forecast, plan and create, etc. In the process of thinking, a wide range of psychic phenomena like perception, memory, judgement and abstraction are involved. Thinking may be divided into following types:

- **Fantasy thinking:** This type of thinking allows the person to escape from or to deny reality. It can be seen in both normal and pathological thinking. Everyone occasionally uses fantasy thinking when daydreaming.
- **Imaginative thinking:** It merges fantasy and memory to generate plans for the present or the future.
- **Rational or conceptual thinking:** Here, the thinking uses logic to solve the problem.

Disturbances of thinking can be studied under the following heads.

Retarded Thinking

The process of thinking gets slowed down, e.g. slow thinking noted in depression where the thinking lacks initiation or planning. **Thought blocking** may be seen in schizophrenia, experienced as a snapping off or sudden break in a train of thoughts. **Thought withdrawal** is a disturbance in the control of thought in which the patient feels as if some alien force had intentionally withdrawn thoughts from his consciousness. In **circumstantiality**, the thinking is slow and proceeds with unnecessary trivial details. However, the goal is not lost and the patient is able to convey his ideas. This is seen in obsessional personality disorders and also in epileptic personality change.

Accelerated Thinking

Typically known as fast thinking and may be seen as normal variant. Rapid, excessive and loud speech is characteristic of mania. Rapid rates of speech may be influenced by cultural and situational factors and merely represent rapid thought. For example, auctioneers speak with an astonishing rapidity, likely reflecting a feigned psychomotor skill. Flight of ideas occurs when the flow of thought switches direction frequently and readily.

Form of Thinking

Form of thinking is disturbed in certain types of schizophrenia. Various types of formal thought disorders may include the following:

- **Asyndesis** where connecting links between successive thoughts are missing.
- **Metonymy** is using imprecise approximations in the place of correct word.
- **Inter-penetration of themes** means intermixing of ideas related to reality and fantasy and the concept thus produced lacks clarity and meaning.
- **Derailment** implies sliding of one thought into a subsidiary thought. When a major thought is substituted by a subsidiary one, it is termed substitution.
- **Tangentiality** is a mild form of derailment in which there is breakdown in associations. Loose associations exemplify severe derailment in which the flow of ideas is no longer comprehensible to the listener since the individual thoughts seem to have no logical relation to one another.
- **Perseveration** implies repetition of a sentence or phrase, sometimes several times over, after it is no longer relevant. Commonly seen in delirium.
- **Stereotype** means constant repetition of a phrase or a behaviour in many different settings, irrespective of context.

Content of Thinking

Abnormal beliefs and convictions form the core of thought content disturbances. Normally also, one may entertain certain beliefs and prejudices that, although they clearly contradict one another, are nevertheless held with passion and conviction.

Delusional Disorders

These were previously known as 'paranoia', presently find place under DSM-IV 297 and ICD-10 F22. Because of changing

diagnostic and classification approaches, the terms paranoia and an associated illness, paraphrenia, practically disappeared from the field. (The term 'paranoia' has been derived from Greek, 'paranos' meaning 'distracted'. It implied a variety of mental illnesses especially characterised by delusions of persecution and an abnormal tendency to suspect and mistrust others. The term 'paraphrenia' as described by Kraepelin denoted illness having similarity with 'paranoid schizophrenia' with fantastic delusions and hallucinations but having less personality deterioration and less impairment of volition. Affect was well-preserved in such cases.) Kraepelin regarded paranoia, paraphrenia and paranoid schizophrenia as a relatively discrete group of illness, later referred to as the 'paranoid spectrum'.

The definition of delusion by Mullen based on the earlier concept of Jaspers is widely quoted and accepted. He characterises delusions as follows:

- They are held with absolute conviction.
- The individual experiences the delusional belief as self-evident and regards it as of great personal significance.
- The delusion cannot be changed by an appeal to reason or by contrary experience.
- The content of delusions is unlikely and often fantastic.
- The false belief is not shared by others from a similar socio-economic group.

Delusions are considered to be the product of pathological process in the brain. They are sometimes categorised as 'primary/autochthonous' and 'secondary'. The former appears relatively sudden, whereas the latter is considered to be a further development within the delusional system. For example, the initial belief may be that the police is watching him day and night whereas the explanation for such a belief may rest in the underlying belief that the authorities do not wish that the secret information which he has been holding about the aliens be divulged.

DSM-IV recognises five main subtypes of the illness based on the predominant delusional themes—erotomanic, grandiose, jealous, persecutory and somatic, and mixed types. ICD-10 also recognised these subtypes, and adds litigious and self-referential. Some important subtypes are being described below:

Persecutory and Litigious Subtypes

The word 'persecute' has been derived from Latin 'persecutus' meaning 'pursued'. According to dictionary, it implies hostility or ill-treatment or harassment especially on the grounds of political or religious belief. The persecutory threats may take the form of descriptions of the most elaborate plots involving a variety of known and unknown adversaries. The beliefs are usually extremely stable and increase in elaboration with the passage of time. Suspiciousness, extreme anxiety and irritability are also the usual accompanying features. Individual believes himself to be the centre of focus and malignant attention that would be inexplicable to the normal person. Interactions with family, social agencies or the authorities become increasingly confrontational. Some may also resort to assault and the danger may be profound because the individual will act as though genuinely under severe threat. Situation may at times be provoked by alcohol or other drugs.

Some individuals may complain of profound and persistent sense of having been wronged and therefore, may repetitively seek redress, often through the legal system. Pursuit of 'justice' may sometimes become 'self-reinforcing'. Goldstein has described three typical ways of such 'litigious paranoia', viz.:

(i) hyper competent defendant using the letter of the law up to and beyond its limits but paying no heed to its spirit,
(ii) pursuing vendettas in a divorce proceedings consumed with jealousy and
(iii) 'paranoid complaining witness' who endlessly goes on initiating litigation despite repeated adverse judgements.

Medicolegal Aspects In some cases, individuals may carry out violent actions in a calculated way, believing that a vengeance is being exacted while in others, anger may express itself explosively. Culpability may be determined by the contents of the delusion. Thus, as Goldstein has pointed out, "If the person felt threatened because of a delusional belief and reacted, as he genuinely perceived, in self-defence, his degree of blame may be adjudged to be low. But if he was equally deluded and carefully plotted revenge, this might be seen as highly culpable". However, such a distinction may be met with difficulty either in the clinical situation or at law.

Hypochondriacal Subtype

Modern society, especially the affluent ones, is pre-occupied with the health concerns. However, some may show excessive concern including pathological self-concern. This may take the form of hypochondriasis, in which there is persistent conviction of illness in the absence of objective evidence of its existence and with inability to accept re-assurances. Usually, the individual refers to physical complaints, but reference to psychological complaints may also be there. Various manifestations have been described in literature, viz.:

- **Delusions involving the skin,** i.e. complaining of organisms/parasites crawling over surface of the skin or burrowing under the skin
- **Delusions of ugliness** (dysmorphic delusions), i.e. morbid fear of being ugly or deformed and the complaint being expressed with unremitting delusional intensity
- **Delusions of body odour** or halitosis, e.g. smell may be attributed to escaping flatus, abnormal sweat secretion, or dental problems leading to halitosis
- **Miscellaneous delusions** comprising numerous possibilities of different themes, some more common examples may include the following:
  - Delusion of transmitting non-sexual diseases to others. For example, when people start coughing on entry of an

individual, the individual may believe that he is transmitting tuberculosis to others

- Delusion of sexually transmitted disease wherein the patient develops the conviction that he has venereal disease, often when there is no evidence of risk-taking behaviour having occurred.

In the past, syphilis was probably the greatest fear, but presently it is usually the AIDS. Repeated tests showing negative serology have no re-assuring effect. Interestingly, a few cases of AIDS have been reported wherein delusional illness with hypochondriasis emerged, usually due to direct effects of the virus on the brain.

Jealousy Subtype

Jealousy may be justifiable as seen in normal day-to-day life and usually accepted by society. It may occur in relation to situation and individual's perception (some people habitually vent anger with slight provocation and others usually bottle-up their feelings). This is what is usually understood as **normal jealousy**, and its expression may range from pique to severe rage. Secondly, jealousy may be characterised by impulsive reaction with excessive manifestation because of some personality disorder. This may be due to some overvalued idea/concept (nonpsychotic) wherein there is preservation of self-awareness of emotion and sometimes of its irrationality. It may, therefore, be termed as **neurotic jealousy**. Thirdly, jealousy as occurring in delusional disorder and is characterised by a fixed belief that cannot be swayed by reasoned argument or presentation of evidence to the contrary. This may be termed as **psychotic jealousy**.

Cobb proposed the following features of pathological jealousy, whether it be neurotic or psychotic:

- The jealous thinking and behaviour are unreasonable in expression and in intensity.
- The jealous individual is convinced of the partner's guilt but the evidence is dubious to others.
- A recognisable psychiatric illness is present, which could plausibly be associated with abnormal jealousy.
- In a proportion of cases, the jealous person has habitual personality characteristics of jealousy, suspiciousness and over-possessiveness.
- Pathological jealousy is usually focused on one specific person.

Medicolegal Aspects Severe assault and even murder may be committed under jealousy. The doctor has a duty to warn and protect the partner if the danger seems substantial. **Jealous killer**: This has been called as 'Othello syndrome' [In Shakespeare's tragedy, Othello (a Moor of Venice) was a participant who smothered his wife named Desdemona in a jealous rage inspired by the treachery of Iago, and later killed himself after learning of her innocence]. In this syndrome, the sufferer becomes convinced about his partner's infidelity and initiates seeking proof of occurrence of such activities. In this exercise, he starts examining her clothing and bedclothes, etc. for evidence of semen/seminal fluid. He may also look for evidence of illicit entry into the house or room and may set up elaborate traps. He may evaluate the 'opening windows' as the scope for the entrance or the exit of the lover. Additionally, he may tax her with accusations of infidelity, often projecting that if she admits, he would forgive and forget. Eventually, the partner may succumb to pressure and falsely confess that she has indeed been unfaithful. This may act as the breaking point and the accuser indulges in violence and justifying the same by quoting the victim's confession. [In this context, it needs be mentioned that 'mental agony' caused to the aggrieved party constitutes "cruelty" as envisaged under Section 13 of the Hindu Marriage Act, 1955. This Section deals with "divorce" wherein cruelty by either partner has been mentioned as a ground for dissolution of marriage. Thus, levelling of false allegations by one spouse about the other having alleged illicit relations with different persons outside wedlock amounts to mental agony and therefore, "cruelty" (Jai Dayal vs. Shakuntala Devi, AIR 2004 Del 39).]

Erotomanic Subtype

In this, the individual has strong erotic feelings towards another person and carries persistent, unfounded belief that the other person is deeply in love with him or her. Commonly, he or she is a real person who is unaware of the situation. Previously, it was claimed that the erotic delusions were largely confined to women, especially isolated and frustrated elderly spinsters. However, cases of males are also being reported presently.

Medicolegal Aspects Often the overt behaviour is in the form of harassment. Sometimes, assault and even murder can be committed under aggression. A deluded woman may claim that the doctor or the teacher showed strong erotic feelings towards her and it may be virtually impossible to persuade the public and the authorities about the falsity of accusations. Any professional person dealing with deluded patient must therefore be vigilant and seek collegial help in dealing with such a situation.

Grandiose Subtype

This variety does not find much description in literature, since the individual who is habitually elated, and who may believe himself or herself powerful or rich, is unlikely to seek help, especially psychiatric help. Two types of categories find place in literature:

(i) Those carrying such a profound state of bliss as to neglect self-care.
(ii) The rest are usually those who have committed some offence under delusional influence.

Medicolegal Aspects Presence of grandiosity in any psychiatric disorder has been regarded as a bad prognostic factor

as the individual does not usually seek help, especially the psychiatric help. Even if the treatment is arranged, the abandoning of highly pleasurable beliefs may not be welcomed by some patients. There may be circumstances where grandiose delusions are overtly acted out.

Medicolegal Importance of Delusions

Delusions, without any doubt, reflect mental abnormality. Hence, they have legal bearings. When a person does some act due to the direct effect of delusion he suffers from, then he will not be held responsible for that act. For example, when a person suffering from mental disease kills an innocent person thinking that the innocent person is about to kill him, and to protect himself he hits that person fatally, as a result of which the innocent person dies, then he will not be held responsible for his act. But, by virtue of delusion he suffers from, if he thinks that another person is out to cheat him and on such a false belief if he kills that person then he will be responsible for the killing. This is as per the provision of the law, which provides that a man reserves the right to protect his own life, if necessary at the cost of the life of the person who attempts to kill him. But law does not approve the killing of a cheat. In some circumstances where the person is suffering from partial delusions and commits some act that is not directly related with the effect of the delusion but has indirect bearing, the doctrine of diminished responsibility may be called to play.

SIGNS/SYMPTOMS RELATED TO DISTURBANCES OF PERCEPTION

We perceive various things around us through our sense organs. However, those perceptions have to be understood in their context and have to be associated with the various previously existing experiences, to give them a proper meaning. Sometimes, sensory experiences can be distorted to produce alterations in intensity and quality of the perceptions. An individual may feel the sensations more intensely on account of emotional tension. In depression and hypochondriasis, the individual may become intolerant to noises and hear sounds louder than what they actually are. Changes in quality of perception like distortion of colours, images, etc. are not common. Alterations in the spatial form of objects can be in the form of **micropsia** (objects appearing smaller) or **macropsia** (objects appearing larger).

Apart from sensory distortions, an individual can undergo a sensory experience in the absence of an external stimulus (hallucination) or he may see an object and mistake it for something else (illusion).

Hallucinations

Hallucinations are perceptions in the absence of an external stimulus. They can affect any sensory system, and they sometimes occur in several systems concurrently. When perception is altered, hallucinations, illusions and often delusions are frequently experienced together. Hallucinations are experienced by many normal people under unusual conditions. Between 10% and 27% of population have experienced hallucinations, most often visual hallucinations. **Hypnagogic** and **hypnopompic** hallucinations are common, predominantly visual hallucinations that occur during the moments immediately before falling asleep and during transition from sleep to wakefulness, respectively. They are physiological in nature. In acute bereavement, spouse may complain of hallucinating the voice or the presence of the deceased. After amputations, phantom limb hallucinations are common.

Auditory Hallucinations

Auditory hallucinations range in complexity from hearing disturbed sounds such as whirring noises and muffled whispers to organised sounds/discussions about the patient. Simple auditory hallucinations are commonly associated with organic psychosis such as delirium and toxic and metabolic encephalopathies. Deafness can produce hallucinations consisting of noises or formed music. Auditory hallucinations are classically associated with schizophrenia but are also frequently seen in psychotic mood disorders. **In command hallucinations,** the patient is ordered by hallucinatory voices to do things/acts. Such voices may be frightening or dangerous as they may command acts of violence towards self or others, such as 'jump off the roof, you are not worth anything'. Patients differ in their capabilities to ignore the commands. He may feel impelled to carry out the orders and indulge in crime.

Visual Hallucinations

Visual hallucinations occur in a wide variety of neurological and psychiatric disorders, including toxic disturbances, drug withdrawal syndromes, focal CNS lesions, schizophrenia, etc. Visual hallucinations may range from simple elemental (in which hallucinations consist of flashes of lights or geometric figures) to elaborate visions, such as flock of angels. In delirious states, visual hallucinations of small animals are common. In alcoholic delirium tremens, **lilliputian hallucinations,** i.e. hallucinations of small animals and human beings, are reported (person sees figures in reduced size like midgets or dwarfs). Scenic hallucinations are associated with various psychiatric disorders and also temporal lobe epilepsy.

Olfactory and Gustatory Hallucinations

Olfactory and gustatory hallucinations (hallucinations involving smell and taste, respectively) have most often been associated with organic brain diseases, particularly with the uncinate fits of complex partial seizures. Olfactory hallucinations may also be seen in major depression, mostly as odours of decay, rotting and death, etc.

Haptic Hallucinations

They involve touch. Simple haptic hallucinations, such as the feeling that bugs are crawling over one's skin **(formication)**, are common in alcohol withdrawal syndromes and in chronic cocaine intoxication.

Illusions

Illusions are misinterpretations of real sensory stimuli. They are perceptual distortions in the estimation of size, shape and spatial relations and are common even in the absence of psychiatric disorders, especially when someone is fatigued or excessively aroused. For example, a child seeing monsters emanating from shadows on the walls or hearing ghosts in the sounds of the wind. Illusions may be of three types. **Completion illusions** are seen in human tendency to complete a familiar and unfinished pattern. **Affect illusions** can be understood in context with the prevailing mood state. **Pareidolic illusions** are those where images are seen from shapes, e.g. seeing human faces in the clouds.

SIGNS/SYMPTOMS RELATED TO DISTURBANCES OF EMOTION

The terms 'affect' and 'emotion' are used interchangeably to describe person's mood state. The experience of an emotion, as felt by the individual, is what is termed as 'affect'. When an affective state is maintained for a considerable length of time, it is termed 'mood'. In the clinical assessment of affect, it has to be ascertained whether the mood is sustained or not, responsive to the surroundings (reactivity of mood), whether there is lability of mood and whether the mood is appropriate or inappropriate to the situation.

Anxiety, depression, euphoria, irritability, anger, etc. are common experiences of most people. However, they are also seen prominently in certain mood disorders and other psychopathological states. Apart from quality of the mood what makes a particular affect abnormal is its intensity and duration, and other psychopathological features associated with the mood. For example, all of us tend to feel depressed at one time or the other, but a diagnosis of depression is made only when certain other characters are also evident, such as depressive feelings existing for considerable length of time, sleeplessness, lack of energy, feelings of guilt, etc. Euphoria, elevation and irritability are seen in mania.

Anxiety

Anxiety is a state of subjective restlessness and apprehension, which may or may not be accompanied by symptoms of autonomic over-activity such as palpitation, sweating, tremors and dilated pupils, etc. Anxiety is said to be free-floating when it is not arising out of any situation. Situational anxiety is confined to certain specific situations. Anxiety can be physiological; for example, anxiety at the time of examination or when facing any new situation. Anxiety is a normal emotion and increases in intensity and duration during certain psycho-pathological states. It is also the commonest symptom of various neurotic disorders, such as anxiety neurosis, phobic neurosis, obsessive-compulsive neurosis, etc.

Phobia

A **phobia** is an unreasonable and unwarranted fear of an object or situation. According to Marks, in phobia:

(a) the fear is out of proportion to the demands of the situation;
(b) the fear cannot be explained or reasoned away; and
(c) the fear is not under voluntary control and leads to an avoidance of the feared situation.

In simple phobias, persistent and irrational fears are provoked by specific stimuli. Phobias may develop to almost any object or situation. Common simple phobias include fears of dirt, excrement, snakes, spiders, heights, water, blood, etc. **In complex phobias,** fears related to broad situations are involved. Agoraphobia, the best known, is the fear of open spaces. In social phobias, people become overwhelmingly anxious under some situations.

Obsession

An **obsession** is an idea, image, affect, impulse or movement that appears as a mental content with a subjective sense of compulsion, overriding an internal resistance. The important characters of obsessions are following:

- The individual realises that they are absurd.
- The individual struggles to get rid of those.
- The individual experiences anxiety; to control the anxiety, he indulges in repetitive acts, i.e. 'compulsive acts'.
- The individual knows/realises that it is not part of his personality to entertain such ideas. This is what is termed as **'ego-alien'** character of obsession.

An obsession may be in the form of any of the following:

- **Obsessional thinking:** Obsessional ideas, thoughts, images, memories, questions or ruminations often appear as counter impulses directed against a situation, e.g. the compulsive obtrusion of obscene pictures while praying.
- **Obsessional impulses:** These are impulses urging the person to perform certain actions that obtrude compulsively into the conscious awareness in spite of resistance. For example, obsessional checking of the doors and windows, impulse to utter obscene words, etc.
- **Obsessional acts:** Here, acts are usually carried out on the basis of obsessional impulses or fears; for example, obsessional cleaning and washing because of morbid fear of dirt. The individual feels that by doing obsessional acts

in a prescribed way he can get rid of the fear. As the doubts keep on increasing, the rituals also become endless, ultimately interfering with the patient's daily life.

- **Panic:** Panic attack is a circumscribed episode of severe state of anxiety lasting minutes to hours. Subjectively, panic is characterised by feelings of utter terror. Individual isolated panic attacks may be common in general public. Panic attacks may be seen more regularly and more severely as a part of panic disorder or in association with other anxiety disorders.
- **Irritability:** It is an unpleasant feeling state in which the person feels an inner discomfort. It may exist purely as a feeling or be behaviourally associated with reduced control over temper. Irritable persons often lash out at others, usually verbally but sometimes, physically too.
- **Elated moods:** Elated moods include euphoria, elation, exaltation and ecstasy. They are marked by feelings of well-being, optimism, capability, pleasure and grace. Such moods are often observed when long-sought-after goals are attained and in states of love, religious fervor and spiritual transcendence. Abnormal elated moods are usually seen primarily as a part of **manic states**. Manic states occur in bipolar disorders and as a secondary mania caused by a variety of physical and toxic conditions. Secondary manias may follow specific cerebral insults accompanying systemic disorder or occur after ingestion of some drugs including amphetamines, antidepressants, decongestants and corticoids, etc.

SIGNS/SYMPTOMS RELATED TO DISTURBANCES OF BODY FUNCTIONS

Appetite

Excessive eating (bulimia and polyphagia) can be due to personality trait. It is also observed in anxiety states and sometimes in depression where it is associated with hypersomnolence. Polyphagia with somnolence is also found in hypothalamic lesions. Excessive appetite with weight loss is seen in thyrotoxicosis, while excessive eating with weight gain may rarely indicate acromegaly. Decreased appetite is seen in anorexia nervosa, depression and schizophrenia. In schizophrenia, especially in catatonic states, the patient may refuse to eat or drink anything due to negativism. It is also a feature of any chronic debilitating disease such as tuberculosis.

Thirst

Excessive thirst (polydipsia) associated with polyuria can be due to psychogenic factors, especially anxiety states. Polyuria with polydipsia is also a feature of diabetes mellitus, diabetes insipidus and lesions around the post pituitary. Patients on lithium therapy may complain of polyuria and polydipsia.

Sex

Loss of libido is seen in depression and also in highly neurotic individuals. It can be a feature of chronic debilitating diseases. Neuroleptic drugs may also decrease the sexual derive. Psychiatric disorders known for decreased sexual drive include depressive disorders, substance abuse disorders and marital conflicts. Increased sexual desire (hypersexuality) is seen in mania and certain organic psychoses where the inhibitions are lost. Sometimes, hypersexual behaviour can be a means of self-assurance in anxious and neurotic individuals. Altered sexuality including fetishes, sadomasochism and paedophilia may occur in isolated psychiatric syndromes.

Sleep

Disturbed sleep is one of the commonest complaints. With advancing age, the mean sleep time decreases. The duration and quality of sleep decreases in various psychiatric disorders. Sleep can get disturbed by various physical ailments. Difficulty in falling asleep or early insomnia is characteristic of anxiety. Late insomnia and early morning awakening are seen in depression. Insomnia may also result from ingestion of substances that alter normal sleep–wake cycle, including alcohol and stimulants. Hypersomnia (excessive sleep during night) also associated with somnolence (excessive during day) is observed in Kleine–Levin syndrome.

Somnambulism (Sleep Walking)

This condition is characterised by aimless wandering with incomplete arousal from sleep, attended with acute anxiety. The person may get up from the bed, open the door, walk out a distance and return to the bed to sleep again, having no recollection of the events the next day. In some cases, he remembers the events of the preceding fit and follows them with exact precision during the next fit, though he forgets them in the normal state. In this condition, the mental faculties are partially active and are so concentrated on one particular train of ideas it is said, that a somnambulist is capable of performing remarkable and incredible pieces of work, which would have puzzled him during his waking hours. A somnambulist may thus solve a difficult problem or may commit theft or murder. Somnambulism forms a good defence for exemption/diminished criminal liability if it can be proved that the individual committed the offence during the fit.

Semi-somnolence or Somnolentia

This is half way between sleep and walking and is often called sleep drunkenness. The state of mind is comparable to a person who is suddenly aroused from a deep sleep and the person may unconsciously do some abnormal acts for some period, say for a few minutes.

Impulse

It is a 'sudden and irresistible force compelling a person to the conscious performance of some action without motive or forethought'. Normally, when somebody intends to do any act,

he tries to realise its consequences and often decides whether to do it or not. A mentally ill person has no such balance of mind and may commit any act as soon as the idea strikes to him. Some common impulses are:

Kleptomania	An irresistible impulse to steal articles of small value.
Pyromania	An irresistible impulse to set fire to things.
Mutilomania	An irresistible impulse to maim animals.
Dipsomania	An irresistible impulse to drink at periodic intervals.
Sexual impulses	The person may feel compulsive urge to perform sexual intercourse, which may often be in a perverted way. There may be some psychic problem in the sexual behaviour or the person may be suffering from mental subnormality.
Suicidal and homicidal impulses	May be seen in certain intoxications, e.g. LCD, cannabis, etc.

Automatic Behaviour (Automatism)

Automatic behaviour is an ill-defined term that literally means 'acting without volition' and the word 'volition' is said to mean 'the faculty of will by which the powers are directed towards the attainment of a chosen end'. It may be raised as a defence in a criminal charge. In this, behaviour may be considered as apparently purposeful and complex that occurs without conscious control and usually followed by amnesia for the event. It can occur in association with epilepsy, alcohol/drug usage, hypoglycaemia, and during sleep. The English Law distinguishes such automatisms as 'non-insane automatism' and considers these as falling short of the 'disease of the mind'. However, the concept of automatism is vague one, and our law has no special provision for automatism. Fenwick provides useful criteria for assessing crimes allegedly attributable to an ictal and to a sleep automatism. In an **ictal automatism** (i.e. characterised or caused by a stroke or an acute epileptic seizure):

- there is a known history of epilepsy and previously manifested similar automatisms;
- the act is out of keeping with the character and the circumstances;
- no evidence of premeditation or concealment is there;
- there is loss of memory for the event but not for events before it; and
- witnesses, if available, would describe a disorder of consciousness.

Hypnotism

This is a sleep like condition brought on by artificial means or by suggestions. The person so induced passes into a state of 'trance' in which his mind becomes susceptible to suggestion or command of the hypnotiser. An Austrian physician of 18th century, Franz Anton Mesmer, practiced this art and hence, also called as **mesmerism**. [A hypnotist while arranging a 'variety programme' put a girl of about 19 years of age into a hypnotic state, performing certain other activities during the induction. The girl started having depression and often feeling frightened on awakening. An action for negligence and assault (because of placing hands on the neck during the exhibition) resulted in an award of 1000 pounds for negligence and 25 pounds for assault together with special damages of 107 pounds. The English Hypnotism Act (1952), subsequently made such public exhibitions illegal.]

As to the nature of acts, the fundamental principle holds good that no one can be compelled by hypnotic influence to commit any act of which he/she was not capable in the normal state. Ericksen, attempting such suggestions on 50 test subjects found that they awoke rather than carry out some repugnant act. It is agreeable that if a person volunteers to be hypnotised, he is expected to have anticipated all the consequences of the deed and agreed to become responsible for them. In other words, it may be said that a person cannot take advantage of his/her own misconduct if he violates the law under the effect of hypnotism. Hypnotism, as a defence in the criminal act, is not generally recognised in courts. Evidence extracted under the influence of hypnotism is not accepted by the court, and the status of such evidence may be comparable to situations wherein evidence is obtained under influence of alcohol/drug etc., e.g. the so-called 'truth serum' (i.e., the use of intravenous thiopentone to extract confessions during interrogation by inducing a state of drowsy disorientation).

Mental Retardation (Mental Subnormality)

The Mental Deficiency Act (1927) recognises three groups of mental development defects, namely idiocy, imbecility and feeble mindedness (morons). These are generally grouped under the term **amentia**. This term is now replaced by the term, 'mental retardation' and includes mental subnormality and mental handicap. Mental retardation signifies a condition of retarded, incomplete or abnormal mental development (Table 29.2). It may be present at birth or may become manifest during early childhood. It can be due to congenital or acquired cause and may be associated with stigmata of physical deformity. Various degrees of mental retardation are discussed below.

PSYCHOSIS AND NEUROSIS

The terms 'neurosis' and 'psychosis' have been used for many years to separate mental illnesses into two groups corresponding generally to the fact that whether the 'gross reality testing and personality organisation' are intact or not, respectively.

Table 29.2 Parameters Determining Mental Retardation

Degree of mental retardation (MR)	IQ	Mental age	Adult life and needs
Mild MR, feeble mindedness (Morons)	50–70	6–11 yr	Can achieve social and vocational skill enough for minimum self-support
Moderate MR (Imbeciles)	35–49	3–6 yr	May perform unskilled/semi-skilled work, needs supervision
Severe MR (Idiocy)	20–34	of 3 yr	Cannot speak intelligibly, needs nursing care
Profound MR (Idiots)	< 20	< 3 yr	No/minimum capacity for sensory-motor functioning, needs nursing care

Intelligence quotient (IQ) is defined as the ratio of a subject's intelligence to the average intelligence for his/her age.

Psychosis

The traditional meaning of the term 'psychotic' is considered to be 'loss of reality testing and impairment of mental functioning' manifested by delusions, hallucinations, confusion and impaired memory. According to the American Psychiatric Association, the term 'psychotic' means grossly impaired in reality testing. With gross impairment in reality testing, persons incorrectly evaluate the accuracy of their perceptions and thoughts and make incorrect inferences about external reality, even in the face of contrary evidence. Direct evidence of psychotic behaviour is the presence of either delusions or hallucinations without insight into their pathological nature. Examples may include agitated, inattentive and disoriented behaviour seen in alcohol intoxication delirium; and markedly incoherent speech without apparent awareness by the person that the speech is not understandable.

Neurosis

Neurosis implies a chronic or recurrent nonpsychotic disorder characterised mainly by anxiety. In DSM-III, a neurotic disorder was defined as, "A mental disorder in which the predominant disturbance is a symptom or a group of symptoms which is distressing to the individual and is recognised by him or her as unacceptable and alien (ego-dystonic); reality testing is grossly intact. Behaviour does not actively violate gross social norms. The disturbance is relatively enduring or recurrent without treatment, and is not limited to a transitory reaction to stressors. There is no demonstrable aetiology or factor".

Cerebral Tumours and Psychosis

Mental symptoms may occur at any stage of the growth of cerebral neoplasms. In neoplasms of the prefrontal region, mental symptoms appear before physical signs. Irresponsible and antisocial behaviour, motor aphasia, grasping and sucking reflexes are seen. Temporal lobe tumours cause automatism, a feeling of objects being unreal and strange objects and persons being familiar. Frontal lobe tumours cause lability of feelings, euphoria and depression. Parietal lobe tumours cause the impairment of appreciation of spatial relationship, agraphia, agnosia and right-left disorientation.

Cerebral Trauma and Psychosis

It is commonly realised that a head injury is generally followed by some degree of mental impairment, either temporary or permanent. Damage to the brain at birth or during early childhood may cause mental subnormality or give rise to epilepsy. In adults, mental symptoms following head injury are said to be common in those abusing alcohol. Concussion occurs mostly in closed injuries. The period of unconsciousness may last for hours or days. The clouding of consciousness of some duration occurs on recovery. Some patients, after sustaining a head injury, appear apparently normal but subsequently show automatism (post-traumatic automatism or twilight state). The patient may carry out apparently purposeful acts without recollection of the accident. The late effects depend upon the severity and extent of the damage and the previous personality of the individual. Boxers who have received recurrent head injury develop symptoms of 'punch drunkenness' characterised by lack of coordination, tremors, ataxia, intellectual deterioration, disturbed memory and signs of cerebellar and extrapyramidal lesions.

Pregnancy/Child Birth and Psychosis

Psychosis may occur at any time from the beginning of pregnancy to the end of lactation. During pregnancy, delusions and dislike or hatred towards the husband may occur and the patient may develop suicidal tendency. Postpartum psychosis, following child birth, may take a great variety of forms, the commonest being mania. Infanticide may be committed. Cases arising late in the puerperium may manifest symptoms of depression with delusions of unworthiness and suicidal tendencies. Lactational psychosis may occur after 6 weeks of confinement. It is usually characterised by mental confusion, hallucinations and depression. The patient may develop persecutory delusions, which may eventually lead to suicide or infanticide.

Epilepsy and Psychosis

While epilepsy may exist without obvious intellectual impairment (some epileptics are really quite intelligent), as a general rule, epilepsy is attended by certain degree of mental blunting,

Table 29.3 Differential Diagnosis of True Seizure vs. Pseudoseizure

Features	True seizure	Pseudoseizure (NEAD)
Aura	Common stereotyped	Rare
Timing	Nocturnal common	Only when awake
Incontinence	Common	Rare
Cyanosis	Common	Rare
Post-ictal confusion	Yes	No
Body movement	Tonic-clonic	Nonstereotyped and asynchronous
Self-injury	Common	Rare
EEG	May be abnormal	Normal
Affected by suggestion	No	Yes
Secondary gain	No	Yes

and in some cases definite psychosis. Psychiatric disturbance arising before the motor discharge is known as pre-epileptic confusional state. When it arises after the motor discharge, it is known as post-epileptic automatism and when it replaces the motor discharge by some outrageous act (epileptic equivalent), it is known as masked epilepsy or psychomotor epilepsy (Table 29.3). **In pre-epileptic confusional state,** the patient drifts into an irritable state for a few days before the onset of the fit, which may be accompanied by clouding of consciousness. The patient may have delusions under the influence of which he may perform criminal acts. **In post-epileptic automatism,** there is lapse of consciousness. The patient usually performs acts without volition. He has no recollection of them afterwards when consciousness is regained. The characteristics of such automatism may be as under:

- The automatic action tends to occur after each fit in the same person and is of the same type in each attack. Examples of such actions may include:
  - the person going to some shop, picking up something, and being arrested for theft, or
  - the person micturating in a public place and being arrested for indecency, etc.
- There is usually total amnesia for the act (1) or rarely faint blurred memory of epileptic automatic phase may remain.

In masked or psychomotor epilepsy, there is no convulsion. The mental disturbance causes the convulsions to be replaced by some outrageous act, such as murder (epileptic equivalent). These cases are usually characterised by undue brutal force and by the fact that the component of premeditation is absent. The victim is usually a stranger, there is no motive, and there is absence of accomplices and preparedness. Further, there is usually no attempt on the part of the patient to hide the crime or to escape.

Personality Disorders

According to Theodore Millon, **personality** is considered as an integrated organisation of attitudes, perceptions, emotions, behaviours, and habits that characterise a person's distinctive way of relating to others and to himself/herself. **Personality traits** are considered as enduring patterns of perceiving, relating and thinking about the environment and oneself that are exhibited in a wide range of important social and personal contacts (DSM-IV). The term **temperament** may be referred to those biologically based predispositions (like irritability, optimism, pessimism, melancholy, etc.) that colour one's personality.

SCHIZOPHRENIA

According to Eugen Bleuler (1911), the term 'schizophrenia' was considered parallel to 'dementia praecox' because he considered 'splitting of the different psychic functions' to be the most important feature. The mechanism of 'splitting' (spaltung) was popular in German psychiatry at the time when Bleuler coined the term 'schizophrenia'. Freud expressed some reservations about both the words and Jaspers forwarded the view that 'splitting' could not be observed in some schizophrenic patients. Hence, currently, it is advocated that there is no such thing as a unitary disease called 'schizophrenia' but only a collection of mental symptoms some congenital, some relics from evolution, and other acquired. A brief sketch of descriptive features of schizophrenia is being furnished in Tables 29.4 and 29.5.

Schizoid personality disorder is characterised by a persistent pattern of social withdrawal. They show discomfort in social interactions and are introverted. There is lack of emotional expression. Speech is usually low and monotonous. Such persons characteristically seem to lack interest in the lives and concerns of others. They may seem absorbed in insignificant matters. Psychomotor activity tends to be lethargic and lacking gesture. Their sexual lives may be poor.

PARANOID PERSONALITY DISORDER

The term 'paranoid' has been used as an adjective to indicate various delusional representations/syndromes. The condition is characterised by pervasive suspiciousness, mistrust and hypersensitivity to criticism. Generally, the paranoids get involved in counter-attacks. Hostility is almost always a characteristic feature in them and can be manifested as excessive argumentativeness and confrontation. The term **paranoia**, as differentiated by Kraepelin, is a distinct condition characterised by chronic

Table 29.4 Brief Sketch of Clinical Features of Schizophrenia

Reality disorders	Disorganisation	Emotions	Excitation and depression	Psychomotor disturbances	Cognitive disturbances
Delusions—prominently of enigmatic character, often showing fragmentation in reality experiences. **Hallucinations**—prominently auditory, hearing voices and speaking in the third person is the most specific.	Primarily showing disturbance in 'form and flow of thinking'. Scale compiled by Andreasen shows that 'loosening of associations' is the dominant feature wherein the flow of thinking is interrupted by deviations towards distant or unrelated thoughts. Examples may include: tangentiality, fusion, derailment, muddling, circumstantiality, perseveration, etc.	Inappropriate affect, i.e. expression of affect that is inappropriate in the circumstances, e.g. hollow laughter that is unrelated to any apparent stimulus. Blunted affect, i.e. decreased responsiveness to emotional issues and diminished facial expression, etc.	Excitation is usually in the form of irritability, sleeplessness, agitation, etc. Depression is often a feature of prodromal phase of the illness	Subtle disturbances in motor coordination are common. Catatonic motor disorders are rare [Catatonia exhibits disturbance of voluntary motor activity and posture (hypomobility/immobility or catatonic stupor in extreme cases). Catalepsy may be exhibited wherein uncomfortable and bizarre postures are maintained against attempts for rectification.]	Lack of insight, i.e. failure to understand the nature, severity and significance of the illness and compliance with treatment. Cognition deficits: a wide range of cognitive deficits occur, major being in the realm of executive function (impaired ability to initiate and select self-generated mental activity), working memory, and long-term memory.

Table 29.5 Motor Signs and Symptoms

Catalepsy (synonyms: waxy flexibility, flexibilitas cerea)	Maintaining uncomfortable positions against resistance
Posturing	Maintaining uncomfortable positions that may have a delusional meaning
Stupor	Inability to communicate despite being awake
Akinesia	Inability to move
Mutest	Inability to speak
Echolalia	Repetition of another person's speech
Echopraxia	Repetition of another person's acts
Mannerism	Uncommon conspicuous expression by gestures, speech, or objects
Grimacing	Uncommon conspicuous facial expression
Stereotypy	Repetition of actions
Verbigeration	Repetition of speech
Tic	Rapid movements of facial or limb muscles
Akathisia	Inability to remain seated or standing
Psychomotor retardation	Slowing of mental and motor activity
Psychomotor agitation	Arousal of mental and motor activity (typically by anxiety)

and highly systematised delusional ideas. Schnieder described them as **fanatic psychopaths**, stressing their intensity and rigidity in confrontation with others. Paranoid individuals do not usually go to the doctor to ask for help. Instead, they believe that they need be protected from others as they entertain suspicion that others are acting to harm, exploit, or deceive them. Humorous remarks or jokes may be interpreted as attacks on their character. Pathological jealousy is a common presentation of paranoid individuals.

HISTRIONIC PERSONALITY DISORDER

This disorder is a descendent of 'hysteria' described by Hippocrates 2400 years ago. It was included in scientific medicine by Kraepelin. The first psychoanalytic description of hysterical personality was given by Wittles and refined by Reich. This disorder is characterised by self-dramatisation, excessive emotionality and attention seeking behaviour. Egocentric and demanding interpersonal relationships are typical of this condition. Experts usually distinguish hysterical (healthier) and histrionic (sicker) personalities, where the latter is considered as an exaggeration of the former. A few differences are given in Table 29.6.

NARCISSISTIC PERSONALITY DISORDER

The terms 'narcissism' originated from the Greek myth of Narcissus who was infatuated with his own reflection in the mirror lake. The condition is characterised by an exaggerated sense of self-importance with a lack of positive attitude for

Table 29.6 Differentiating Features of Hysterical and Histrionic Personality

Hysterical personality	Histrionic personality
Neurotic personality organisation	Borderline personality organisation
Integrated identity	Diffuse identity
Predominance of repression	Predominance of splitting
Intact reality testing	Intact reality testing (proneness to distortion)
Integrated superego	Marked superego defects
Strongly bonded families	Disturbed, often broken families
Steady educational and vocational careers	Erratic careers
Capable of maintaining long-term friendships	Chaotic interpersonal relationships
Suggestible in triangular relationships	Diffuse suggestibility
Inauthenticity	Multiple identifications
Changing moods	Frequent dysphoria
Sexual inhibition	Promiscuity, perverse tendencies
Competitiveness with the same sex	Less differentiated behaviour toward sexes
Genital traits	Oral/pregenital traits

others. Grandiosity and constant craving for admiration are its other features. According to Kernberg, narcissistic individuals function on a borderline level. He argues that self-hatred, rather than self-love, lies at the root of pathological narcissism. He further says that **malignant narcissism** may be considered at the extreme end of the continuum and is a combination of narcissistic personality disorder, antisocial behaviour, egosyntonic aggression or sadism directed against others.

ANTISOCIAL PERSONALITY DISORDER

This is characterised by disregard for the safety of oneself and others and violation of the rights of others without feeling remorse. Such individuals are usually unreliable, manipulative, and unable to conform to social norms. Various factors, namely biological, developmental and social, have been implicated. Parental deprivation and deviance during critical period seem to be a critical factor. Robins showed that having a sociopathic or alcoholic father was a powerful predictor of antisocial personality disorder in adult life. Psychological undernourishment may be the adding factor. Apparently, they may appear quite normal, charming, and understanding. However, history reveals the factual position. 'Disinhibitory syndrome' is considered to be an essential feature. The syndrome is characterised by an impaired control of impulses leading to temper outbursts without proper recollection (sometimes known as **short-circuit reactions**). Superficiality of knowledge, failure to learn from experience, and a paranoid view of the world are commonly exhibited. Robins described antisocial personality as beginning around 8 years of age and continuing into adult life. He drew attention to the criminal behavior associated with the disorder. However, later the concept was modified to include personality traits as well as features described originally by Cleckley (his seminal work 'The mask of Sanity' is considered to be the basic text on antisocial personality disorder. He differentiated psychopathic personality from criminality and social deviance behaviour). ICD-10 labels this personality disorder as 'dissocial personality disorder' and reflects personality traits more than overt criminal behavior.

DEPENDANT PERSONALITY DISORDER

The critical feature is the urgent desire to be cared for by others, with dependence, attachment, and fear of abandonment. To achieve their goal, they usually relinquish their own needs, expression of feelings, and even their self-identity. In exchange, they seek others to have the responsibility for their lives. They manifest self-doubt, pessimism, and a need for affection.

DEPRESSIVE PERSONALITY DISORDER

Such individuals are submissive, introverted, and unassertive. Their life-style is marked by pessimism, dejection, and self-reproach. They have a negative view of past and present and generally do not expect things to improve. They show low tolerance to shortcomings and failures. They are prone to guilt and feel inadequate. Factors like sudden loss, inadequate attention from parents and a punitive super ego have been postulated to be instrumental in the causation. Some have suggested that a depressive temperament is genetically related to affective disorder.

MASOCHISTIC (SELF-DEFEATING) PERSONALITY DISORDER

The term 'masochism' was introduced to psychiatry by Kraft-Ebing in 1882. It was derived from the character in a novel by the German author Leopold von Sacher-Masoch. This character endured torture, scorn, and humiliation from a woman in the novel. Reich described the masochistic character as a person who suffered deep frustrations in early developmental stages and expressed this frustration through suffering inflicted by the love 'objects'. Thus, presence of defiance is a typical feature in the masochistic search for love. Such individuals accept and endure humiliation, expecting that others would sympathise with them. In this way, they try to fulfill their expectation of gaining love and care through submission. Masochists believe that by exaggerating their weaknesses, they will protect themselves from aggression by others. The mood of these

individuals mostly remains dysphoric, fluctuating between anxiety and sadness.

SADISTIC PERSONALITY DISORDER

Sadism was originally described as 'desire to inflict pain upon the sexual object' by Kraft-Ebing, after the writings of the Marquis de Sade. Later, many experts dealt with various forms of sadistic behavior that were different from sexual sadism. The term 'sadomasochism' was conceptualised by Kernberg, where in sadomasochistic character included 'help-rejecting complainers' and often having borderline personality organisation. Some authors consider sadistic personality disorder to be complementary to self-defeating personality disorder, because they argue that the person who is prone to abuse others is likely to be masochistic and the person who is repeatedly abused, is likely to be sadistic. Gay (one of the chief investigators of this disorder) found that factors like significant childhood loss and physical, emotional or sexual abuse during childhood were instrumental in leading to such disorder. She observed that such individuals were surprisingly laborious and carried intense long-lasting relationships. They considered abuse of the partner and children as consistent with culturally accepted patriarchal values. Also, they showed pattern of cruel, demeaning, and aggressive behaviour towards others in order to cause suffering and to establish dominance and control.

Change(s) in personality may occur due to experiences of disaster and prolonged exposure to other life-threatening situations. Sometimes, they also occur following a mental disease/disorder, the cause being related to the stressful experience and the perceived damage to the patient's self-esteem. Other factors may include people's attitude towards the illness and previous psychological adjustment. Head injury, cerebral neoplasm, vascular accidents, Huntington's disease, epilepsy, etc. may all cause personality change, especially when affecting frontal and temporal lobes. Systemic diseases involving CNS, endocrine and metabolic disorders, AIDS and chronic metal poisoning may also lead to change in personality. However, such patients do not generally show intellectual deterioration.

Certification of Mental Illness

The certificate in respect of mental illness is a legal document; as per Section 21 of Mental Health Act, it must meet with the following requirements:

- It must be issued in the prescribed form.
- Two medical certificates are required of which one must be from a doctor in the Government service.
- The patient should have been examined not earlier than 10 days before an application for reception is made.
- Each doctor must examine the patient separately without consulting each other and form an independent opinion on the basis of his own observations and from the particulars communicated to him.
- Each certificate should clearly state that the patient is suffering from mental illness/disorder of such a nature and degree as to warrant his restraint in a psychiatric hospital or registered psychiatric nursing home and that such restraint is necessary in the interest of the health and safety of the patient or for the protection of others.
- If it is not practicable to give an opinion on the mental health of the person who is alleged to be mentally ill, the magistrate may permit the observation of such a person in an observation ward of a general hospital, general nursing home or any other suitable place for a period of 10 days at a time, extendable to a maximum of 30 days.

True and Feigned Mental Illness (Insanity)

Symptoms of mental illness are occasionally feigned by persons accused of criminal offence in order to prevent a trial, to procure an acquittal, or to escape the consequences of a business transaction/deed. Personnel in army, navy and other services may do so to escape punishment for neglect of duty or when they desire to leave the service. In most cases, this is fairly easy to detect the malingering, but in some cases it may be extremely difficult and may require prolonged and careful observation (Table 29.7).

Restraint of the Mentally Ill

The humanists react with abhorrence if an individual's freedom is restricted without a justified process. Many mentally ill patients may be admitted as 'voluntary' or 'informal' patients, and most of them are coerced to some extent for accepting hospitalisation. This coercion may be from an employer, a family member or a doctor. In such cases, the treatment and/or hospitalisation may be evaluated on the principle of beneficence. Temporary hospitalisation to regain sanity is a much more preferable alternative to staying chronically sick but free. The **Hawaii declaration** of the World Psychiatric Association provides the following guidelines for such situations:

> No procedure must be performed or treatment given against or independent of a patient's own will, unless the patient lacks capacity to express his or her own wishes or owing to psychiatric illness cannot see what is in his best interest or, for the same reason, is a severe threat to others. In these cases, compulsory treatment may or should be given provided that

Table 29.7 Differentiating Features of True and Feigned Insanity

Points of difference	True insanity	Feigned insanity
Onset	Usually gradual or rarely sudden but almost always without any motive	Always sudden and not without some motive
Predisposing factor(s)	Usually present	Usually absent
Facial expression	Usually peculiar in well-developed cases of insanity	Generally normal even when the person pretends to be insane
Look	Vacant, agitated or worried	Not so
Mood	Excited, depressed or fluctuating	May overact to show abnormality in mood
Signs and symptoms	Usually point to a particular type of mental illness. The individual shows signs and symptoms of insanity irrespective of his conduct being observed or not	Signs and symptoms are not uniform and do not indicate any particular type of mental illness. The individual pretends to be insane only when he is observed, and there may be total absence of symptoms when he thinks that he is not being observed
Physical exhaustion	Minimum, even with continuous overactivity	Gets exhausted like a normal person
Physical manifestations	Manifestations like dry harsh skin, furred tongue, constipation, anorexia and insomnia are present	Manifestations characterising true insanity are absent
Habits	Invariably dirty or filthy	Usually not dirty or filthy, though a false show may be posed to that effect
Repeated examination	Not worried about being repeatedly examined	Resents for fear of being detected

it is done in the patient's best interest and over a reasonable period of time, a retroactive informed consent can be presumed and, whenever possible, consent has been obtained from someone close to the patient. As soon as the above conditions for compulsory treatment or detention no longer apply, the patient must be released, unless he or she voluntarily consents to further treatment. Whenever there is compulsory treatment, there must be an independent and neutral body of appeal for regular inquiry into these cases. Every patient must be informed of its existence and be permitted to appeal to it, personally or through a representative without interference by hospital staff or by anyone else.

IMMEDIATE RESTRAINT

A mentally ill person can be placed under immediate restraint when he develops mental incapacity to the extent that renders him dangerous to himself or to others. Immediate restraint under the personal care of the attendants (e.g., locking the room) may be lawfully imposed either through the consent of the guardian of the mentally ill person or without the consent (if there is no time to obtain the same) to prevent danger. However, the patient may be released whenever he becomes no more dangerous. Immediate restraint may also be exercised in cases of delirium, but the restraint must cease with the subsidence of symptoms.

Admission to the Psychiatric Hospital

VOLUNTARY OR DIRECT ADMISSION

Any individual considering himself to be mentally ill and desiring admission to the hospital can request the doctor in charge of the hospital for being admitted as a voluntary patient. The doctor in charge of the hospital can admit such a patient after being satisfied as to his illness.

In case of a minor, the request is to be made by the guardian. When such a minor attains majority, the doctor shall intimate this fact to the patient and unless a request for continuance as an inpatient is made by him within 1 month from such intimation, the doctor shall discharge him unless the doctor thinks that there still exists need for the continuance of treatment. For this purpose, he should have the opinion of a board of doctors. But the treatment under such circumstances shall be continued for a period not exceeding 90 days at a time.

ADMISSION BY APPLICATION THROUGH A RELATIVE OR FRIEND

- If the mentally ill person is unable to apply himself, a relative or friend can apply to the doctor in charge of the hospital.
- Two medical certificates in support of the mental illness of the person are to be attached along with the application or

the illness may be confirmed by two doctors working in the hospital.
- The doctor can admit such a patient for a period not exceeding 90 days at a time.

RECEPTION ORDER ON PETITION TO THE MAGISTRATE

Here, the petition may be made to the magistrate by the doctor in charge of the hospital or by the husband, wife or other relative of the mentally ill person.

- Where made by the doctor, he must be satisfied that the patient undergoing temporary treatment is suffering from mental disorder of such a nature and degree that his treatment requires to be continued for more than 6 months.
- Where made by the husband or wife or some relative of the patient, he must have seen the alleged mentally ill person within 14 days before the date of application. The application must be supported by two medical certificates (one from the registered medical practitioner and the other from some government doctor, who had examined the patient within 10 days prior to issuing the certificate specifying the need for hospitalisation). Another certificate from the doctor mentioning the physical fitness of the patient to travel is also to be attached.
- A bond by the patient's relative or friend to pay hospital expenses is to be extended.

The magistrate, if satisfied, will make a reception order for the admission of the patient to the hospital. However, magistrate may fix a date for the consideration of the application and may also make such inquiries concerning the alleged mental illness of the patient as he thinks fit and pass order accordingly. Such order holds good for 30 days.

RECEPTION ORDER ON PRODUCTION OF MENTALLY ILL PERSON BEFORE THE MAGISTRATE

Such a situation may be seen under the following two circumstances:

- **Wandering and dangerous lunatic:** The police officer in charge of the police station may take into protection any person wandering at large within the limits of the police station whom he has reason to believe to be mentally ill and produce him before the magistrate within 24 hours excluding the time necessary for the journey. The magistrate, if satisfied, may pass order for his admission into the psychiatric hospital or may get him examined by the doctors and then pass order for his admission. The friend or relative should undertake to pay the maintenance charges of the hospital.
- **Neglected lunatic:** The police officer in charge of the police station is duty bond to report to the magistrate any mentally ill person who is not under proper care and control or is ill-treated or neglected by any relative or other person in whose charge he is. Any private person can also report such incidence to the magistrate. The magistrate may entrust the mentally ill person to the care of the relative or other person legally bound to maintain him. However, if such a person is not available or such a person is unable to do so for any reason, the magistrate may issue an order for reception of such a mentally ill person as an in-patient in a psychiatric hospital.

RECEPTION OF THE MENTALLY ILL PRISONER

A mentally ill prisoner is one who has become mentally ill while undergoing a sentence in jail (prison) for some offence already committed. He can be admitted to the hospital on a reception order from the magistrate after the person's illness is medically certified.

RECEPTION OF ESCAPED MENTALLY ILL PERSON

If any mentally ill person, who is already undergoing treatment in hospital, escapes, he may be retaken into the hospital by any police officer or member of the hospital within a period of 1 month from the date of escape.

RECEPTION AFTER JUDICIAL INQUISITION

If a person possessing huge property turns mentally ill, the High Court or the District Court may pass order of inquisition and arrange reception of the patient to the mental hospital. The court may also appoint some manager for the property and the necessary fees for the hospital may be recovered from the profits or income from the property under the court care.

Discharge of the Mentally Ill from the Psychiatric Hospital

A mentally ill patient may be discharged from the hospital by considering following points:

- Request of the voluntary/direct boarder.
- Viewing the extent of recovery or cure.
- Under orders from the magistrate after looking into the patient's condition and assurance by the relatives for his proper care.
- After judicial requisition confirming the patient to be fit for discharge.

It must be remembered that a mentally ill person has an inherent right to ask for repeated examination of his mental condition in order to secure his release from the hospital if found reasonably cured.

Civil Responsibility of the Mentally Ill

MANAGEMENT OF PROPERTY AND AFFAIRS OF THE INSANE

Chapter VI of the Mental Health Act 1987 provides for the legal proceedings to be followed in cases concerning the protection of person and property of the mentally ill person. On application by any relative of the alleged mentally ill person or by the Advocate General of the State in which the alleged mentally ill person resides or by the Collector of the District on behalf of the Court of Wards, the District Court shall direct an inquisition whether the person alleged to be mentally ill is of unsound mind and incapable of managing himself and his property. The court may also order inquiries concerning the nature of the property belonging to the alleged mentally ill person, the persons who are his relatives or such other matter(s) as seem proper to the court.

After being satisfied as to the mental illness of the person and related issues, the court can appoint a manager/guardian to look after his property, granting him the necessary powers. The court may, if it appears to be just for the mentally ill person's benefit, order that any property, movable or immovable, be sold, charged, mortgaged, dealt with or otherwise disposed off as may seem most expedient for the purpose of raising money to be used for and in the interest of mentally ill person.

CONTRACT

Contract is an agreement enforceable by law. An agreement is simply an accepted proposal. Although an oral agreement is perfectly valid, writing and registration are necessary where so required. Section 6 of the Indian Contract Act provides that 'a proposal is revoked by the death or insanity of the proposer, if the fact … comes to the knowledge of the acceptor before acceptance.' Section 11 of the Act states that 'every person is competent to contract, who is of the age of majority … and who is of sound mind …'. Section 12 of the Act declares a person to be of sound mind for the purposes of contract 'if at the time when he makes it, he is capable of understanding it and of forming rational judgement as to its effect upon his interests'.

'A person who is usually of unsound mind, but occasionally of sound mind (lucid interval), may make a contract when he is of sound mind'. Similarly, 'a person who is usually of sound mind, but occasionally of unsound mind, may not make a contract when he is of unsound mind'.

MARRIAGE AND DIVORCE

The Hindu Marriage Act (Act 25 of 1955) provides for conditions for a Hindu marriage. Section 5 (ii) states that at the time of marriage, if either party:

(a) is incapable of giving a valid consent… due to unsoundness of mind; or
(b) though capable of giving consent, has been suffering from mental disorder of such a kind or to such an extent as to be unfit for marriage and the procreation of children; or
(c) has been subject to recurrent attacks of insanity …

Any marriage solemnised under any of the above circumstances shall be voidable and may be annulled by a decree of nullity under Section 12 of the Act. Another ground for nullity under the same Section is the fact that the consent for marriage was obtained by 'fraud' … 'as to any material fact or circumstance concerning the respondent'; for example, the fact of mental illness or treatment for the same.

Divorce can be granted under Section 13 of the Act on a petition presented by either spouse on the ground that the other party 'has been incurably of unsound mind, or has been suffering continuously or intermittently from mental disorder of such kind and to such an extent that the petitioner cannot reasonably be expected to live with the respondent'.

ADOPTION

Under the Hindu Adoptions and Maintenance Act (Act 78 of 1956), any Hindu male 'who is of sound mind and is not a minor' can adopt a child, with the consent of his wife unless she has been declared by a court to be of unsound mind (Section 7). The same holds good for the female also. Also, the person capable of giving in adoption of a child should be of sound mind.

COMPETENCY AS A WITNESS

Under the IEA 1872, a lunatic is not competent to give evidence if he is prevented by virtue of his lunacy from understanding the questions put to him and giving rational answers to those questions. However, such a person can give evidence during a lucid interval on the discretion of the presiding officer of the court.

VALIDITY OF CONSENT

Section 90 of IPC dictates situations where the consent given by an individual is not valid. This Section explains that if the consent is obtained by coercion, undue influence, fraud, misrepresentation or misconception of facts, the consent so obtained is not proper in the eyes of the law. The Section further says that the consent given by a person who by reason of unsoundness of mind, intoxication or immaturity of age is incapable of understanding the nature and consequences of the act to which he consents is not valid in the eyes of law.

TESTAMENTARY CAPACITY (CAPACITY TO MAKE A VALID WILL)

Testamentary disposition is regulated by the Indian Succession Act (Act 39 of 1925).

The various components comprise:

- an understanding of the nature of the will,
- a knowledge of the property to be disposed of, and
- an ability to recognise those who may have justifiable claims on his property.

Some of the salient points regarding testamentary disposition are as follows:

- The 'will' must be in writing, though it need not be registered.
- It must be signed by testator in the presence of at least two witnesses.
- A legatee cannot attest a will.
- An executor(s) is appointed under the will by the testator to carry out its terms after his death.
- A will can be revoked or modified anytime before the death of the testator.
- A will comes into effect after the death of the testator. It is said to speak from grave and to be 'ambulatory'.
- The testator must be of a 'sound and disposing mind'. Section 59 of the Act states that 'every person of sound mind, not being a minor, may dispose of his property by will'.
- **A will is invalid under the following conditions** (for example):
  - If it is executed under undue influence of any other person.
  - Imbecility arising from advanced age or by excessive drinking.
  - Insane delusions making the testator incapable of rational views and judgement.
- **A will is valid under the following conditions** (for example):
  - Deaf, dumb or blind persons who are not thereby incapacitated for making a will and are able to know what they do by it.
  - Lucid intervals.
  - If testator commits suicide immediately after making the will (in the absence of evidence of mental disorder).
  - Presence of delusions not affecting in any way the disposal of the property or the persons affected by the will.

LIABILITY OF THE THIRD PARTY FOR THE ACTS OF MENTALLY ILL

The third party may be an individual who is caring for the defendant, or may be hospital/health authority, or police/prison authority, etc. under whose custody the mentally ill has been put for some reason or the other. The recognition of the need to treat various mental illnesses as well as addiction to alcohol and drugs with higher degree of sensitivity has been highlighted in recent years by honourable courts. **A case** (The Medical Superintendent, St. Gregorious Mission Hospital, Mannar, Kerala vs. Jessey and Another, R.P. No. 4080 of 2008) may be cited as an illuminating illustration wherein the patient had been admitted to a hospital for treatment of alcoholic psychosis and de-addiction of drugs in May 2000. Five days later, he hung himself in an empty hospital ward, using his *lungi*. In response to complaint of negligence filed by the wife and the minor child of the deceased, district consumer disputes redressal forum directed the hospital to pay them a compensation of ₹ 275,000 along with interest at the rate of 9%. Not succeeding in the State Commission, the hospital ultimately filed Revision Petition in the National Commission. The Commission, re-inforcing the need for hospitals to pay utmost attention to the safety of such patients observed, "Hospitals must pay utmost attention to the safety of patients suffering from mental illnesses without curtailing their movements or freedom. The court laid stress on a different kind of duty expected from hospitals in the treatment of those who may be psychologically unstable. Failure to keep a constant watch over them constitutes negligence."

Criminal Responsibility of the Mentally Ill

The plea of mental illness may be brought forward in charges of murder in order to escape capital punishment. If mental illness is established, the accused person is found 'not guilty', and is ordered to be kept in a psychiatric hospital or psychiatric nursing home, jail or other suitable place of safe custody. A mentally ill person is not punished for his crime, as he is devoid of free will, intelligence and knowledge of the act, but society must be protected against the attacks of a mentally ill person.

The law presumes every individual at the age of discretion, to be sane and to possess a sufficient degree of reason to be responsible for his criminal acts, unless the contrary is proved to the satisfaction of the court. In criminal cases, where mental illness is raised as a plea of exemption from liability, the burden of proving it lies on the defence. The present law on the defence of insanity is contained in **Section 84 of IPC**, which reads, "Nothing is an offence which is done by a person who, at the time of doing it, by reason of unsoundness of mind is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law".

HISTORY OF THE McNaughton CASE AND THE McNaughton RULES

The first important case with regard to criminal responsibility of an insane person came to a court in 1800. However, the case which led to widespread public attention was that of McNaughton (1843). Daniel McNaughton, a 29-year-old Scotsman, had been harbouring a delusion that spies sent by Catholic priests, with the help of Tories (the party then in power in England) were constantly following him, harassing him and hatching a conspiracy against him. He also probably had auditory hallucinations to the effect that Tories were accusing him of crimes of

which he said that he was not guilty. Therefore, he decided to kill the Tories Prime Minister, Sir Robert Peel, making elaborate plans for the crime. Not knowing Peel by sight, McNaughton lay in wait at his residence on 20th January, 1843, and mistakenly shot his private secretary, Henry Drummond, who was leaving the Prime Minister's residence. Ten physicians (9 for defence and 1 for prosecution) found him insane and the jury gave the verdict as 'not guilty by reason of insanity'. He was sent to Bethlem Hospital for life.

The verdict led to unprecedented public (and Royal) outcry. Several days following the verdict, Queen Victoria, herself the target of assassination by the insanity acquittee Edward Oxford, summoned the House of Lords to a special session. At this session, the Lords (14 in number) were instructed to clarify and more strictly define the standards by which a defendant could be acquitted by reason of insanity. The answers given by the 14 judges came to be known as the McNaughton Rules. Section 84 of IPC that deals with the criminal responsibility of the insane is based upon these McNaughton Rules.

CRITICISM OF THE McNaughton RULES

In essence, the McNaughton rules, often referred to as **'right-wrong test'**, lay down that in order to establish defence on the grounds of insanity, it must be clearly proved that "at the time of commission of the act, the accused was labouring under such a defect of reason from disease of mind as not to know the nature and quality of the act he was doing; or if he knew what he was doing, that he did not know that it was wrong or contrary to the law". In India, it had been adopted by the legislation in Section 84 of the IPC as mentioned above.

As advances in psychiatry were made, the McNaughton rules came under increasing attacks as being antiquated. The major basis of this criticism was the argument that some forms of mental illnesses affect a person's volition or power to act without impairing his cognitive functioning, while the McNaughton rules laid stress on the cognitive (intellectual) faculties of the person and did not take notice of impairment of emotional and/or volitional factors. An attempt, therefore, was made to gain recognition of the acts committed under an impulse which the person was deprived of any power to resist. This is the criterion for **'uncontrollable or irresistible impulse rule'**. An attempt to add this rule of 'irresistible impulse' to the McNaughton rules was made in 1924 in the House of Lords, by Lord Justice Darling but was met with severe opposition. In India too, courts have rejected the rule of 'irresistible impulse' as a ground of exemption from criminal responsibility.

DOCTRINE OF DIMINISHED RESPONSIBILITY

The concept of diminished responsibility arose as a means of avoiding the death penalty. The first recorded case, in which the verdict was diminished or partial responsibility ('murder with extenuating circumstances') was that of Alexander Dingwall of Scotland in 1867. It was subsequently used widely in the Scotland Courts, though it was incorporated in the English Law only in the 1957 Homicide Act. Section 2 of this Act enacts that a person shall not be convicted of murder if he was suffering from such abnormality of mind as substantially to impair his mental responsibility (the abnormality of mind may arise from arrested or retarded development, any inherent cause, disease or injury). In such cases, the conviction shall be, not for murder, but for manslaughter. **In India, the principle of diminished responsibility in murder cases** is to be found in exceptions 1 and 4 of Section 300 of IPC. These exceptions read as under:

Exception 1: Culpable homicide is not murder if the offender, whilst deprived of the power of self-control by grave and sudden provocation, causes the death of the person who gave the provocation or causes the death of any other person by mistake or accident.

Exception 4: Culpable homicide is not murder if it is committed without premeditation in a sudden fight, in the heat of passion, upon a sudden quarrel and without the offender having taken undue advantage or acted in a cruel or unusual manner.

DURHAM RULE

This rule was formulated by Judge David Bazelon in 1954 in the Monte Durham Case (Durham vs. US) in the District of Columbia, USA. The rule said that 'an accused is not criminally responsible if his unlawful act was the product of mental disease or mental defect'. The rule is sometimes referred to as "product rule/test". However, the terms 'product', 'mental disease' and 'mental defect' created lot of confusion and therefore, the rule was replaced 18 years later in the Brawner Case (US vs. Brawner, 1972) by nine members of the Court, including Judge Bazelon.

CURREN'S RULE (1961)

This rule postulates that an accused 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. This is similar to Irresistible Impulse Rule as the words 'capacity to regulate his conduct to the requirements of law' amply point out. Again, the terms, 'mental disease' and 'mental defect' were the elements of confusion and therefore, could not survive longer.

AMERICAN LAW INSTITUTE TEST

In the early 1960s, the American Law Institute (ALI) drafted a provision that could reasonably bridge the narrowness of the McNaughton rule and expensiveness of the Durham rule. The ALI standard provided, "A person is not responsible for criminal conduct if at the time of such conduct as a result of mental disease or defect he lacks substantial capacity either to appreciate the criminality of his conduct or to conform his conduct to

the requirements of the law". **Its differentiation from McNaughton's standard** can be visualised by analysing the provision:

- The words 'criminal conduct' used in the ALI standard convey broader concept as compared to 'criminal act' used in the McNaughton rule.
- The words 'as a result of mental disease or defect' used in the ALI standard again embrace a broader spectrum of psychiatric disorders encompassing mental disease as well as defect as compared to the term 'unsoundness of mind' (a vague term) used in McNaughton rule.
- The notion 'lacks substantial capacity' conveys that the ALI standard does not require total lack of capacity and gives the decision-makers significant latitude in weighing the quantitative element of the evidence.
- The expression 'either to appreciate the criminality of his conduct or to conform his conduct to the requirements of the law' used in the ALI standard is noteworthy. The word 'appreciate' in the former part of the expression is specifically intended to address both the cognitive as well as affective/emotional elements of the process of understanding. And the word 'conform' used in the latter part is intended to include the volitional element to the defence of criminal conduct.

Criminal Responsibility for Offence Committed During Intoxication

Offences committed under drunkenness may be dealt with separately from other offences, like murder, etc., because of the external factors including the state of mind under intoxication. Intake of intoxicant may be voluntary, when taken deliberately, or may be involuntary, when taken against one's will, and not deliberate or voluntary drunkenness. Again, the degree of drunkenness or intoxication alone will relieve from criminal responsibility as renders him incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law (Section 85 IPC). The emphasis is on the impairment of the cognitive faculty, as in the case of Section 84. The burden is on the accused to lead evidence, including medical evidence of his drunkenness to prove the degree of intoxication to show that his mind was so affected by drink that he was not in a position to form any intent (mens rea) essential to constitute the offence in question.

Sexual Perversions/Deviations (Paraphilias)

Paraphilias denote aberrant sexual activity or deviantly expressed sexual interests and behaviours that are socially prohibited or unacceptable, or biologically undesirable. As per DSM-IV diagnostic criteria, a patient must have experienced one of the behaviours as enlisted under the category for a period of 6 months or more and the behaviour must cause clinically significant distress or impairment in social, occupational, and/or other significant areas of the individual's functioning. A person who indulges in such acts is known as sexual pervert or deviate. In all these perversions, it is essential to consider the mental state of the individual in collaboration with a skilled psychiatrist. The perversions may be studied under the following classification:

- Perversions (paraphilias) requiring a partner for gratification:
  - Paedophilia
  - Sadism
  - Masochism
  - Frotteurism
- Perversions not requiring a partner for gratification:
  - Voyeurism (scoptophilia)
  - Exhibitionism
- Perversions requiring some object/article as a stimulus for sexual arousal:
  - Fetichism or fetishism
  - Transvestism
- Perversions exhibiting excessive sexual arousal and drive:
  - Satyriasis in males
  - Nymphomania in females

PAEDOPHILIA

It involves sexual contact with a child at least 5 years younger than the perpetrator, the perpetrator being at least 16 years of age or older. Paedophilia involves boys, girls, or both sexes of children and the activities are limited to incest, non-incest, or both. Paedophilia needs to be differentiated from child molestation (the word 'molest' means to trouble or annoy in a hostile manner or in a way that causes injury, and is used usually in relation to sexual behaviours). This term technically applies to individuals who are sexually involved with children. Not all child molesters are paedophiles. The use of mind altering drugs, poor impulse control, or mental retardation may be contributory factors.

Children are naive, allowing easy friendship and responding to requests of the adult. Consequently, paedophiles usually develop non-sexual relationship initially, and in the event of being alone and isolated with the child, start having sexual touching or other behaviours. As documented, most paedophiles molest children known to them; only about 10% molest children who are strangers. Certain peculiar issues in this context may include the following:

(i) Inability of the child for understanding nature and consequences of the act.
(ii) Unawareness of the children that in the event of disclosure, they will be interviewed by police.

(iii) The aspect of being ostracised by other children for having been molested.
(iv) The aspect of perpetrator's being influential because of being elder, i.e. often having a parental or other position of authority with respect to child, etc.

SADISM (ALGOLAGNIA)

In sadism, the person gets sexual gratification by torturing sex partner by beating, biting, whipping or ill-treating the partner. The cruel sexual behaviour has been named sadism after the name of **Marquis de Sade**, who wrote books in which the characters enjoyed being cruel. This perversion may be practiced by either sex but is more common in the males. Multiple injuries may be inflicted on various parts of the body but breasts and external genitalia are commonly selected. In extreme cases, even a murder may be performed. Such a murder is known as **lust murder**. After murder, the sadist may perform sexual intercourse on her **(necrophilia)** or may tear out the genitals or other organs and eat the flesh to satisfy the sexual hunger **(necrophagia)**. The sufferer may be suffering from psychosexual incompetency and may find this as most suitable a process, as there is no resistance or rejection and moreover, the same will not be known to anybody else.

MASOCHISM

It is the reverse of sadism. It may occur in either sex but is common among males. Here, the person gets sexual gratification or arousal by being bodily tortured or abused. The term is derived from Leopold Von Sacher Masoch, an Austrian Novelist who described such characters suffering from this perversion. As a stimulus to write, he liked to be whipped by his wife.

Masochistic asphyxial death (hypoxyphilia) may occur when a pervert creates a state of partial hypoxia in him to experience sexual arousal and orgasm, the so called **sexual asphyxia**. The condition may be achieved by hanging or by strangulation or by using masks, pads on the face or enveloping the face in plastic. There may usually be signs of associated sexual activity, such as transvestism, rubber fetishism, pornography and bondage, etc. After experiencing orgasm, the constricting/suffocating mechanism may not be released due to some malfunctioning of the device and thus resulting in death (auto-erotic death).

FROTTEURISM

It involves uninvited touching or rubbing against another person for sexual gratification. Frotteurs carry out paraphilic behaviour in crowded places/environments such as bus stands, subways, sporting events, crowded bars, etc. Fantasy is an extensive component of frotteurism, i.e. they think that touching behaviour will not offend victims, and furthermore, victims will find touching pleasurable. The parts practiced for rubbing are usually buttocks, thighs, breasts, or genital areas, etc. Some frotteurs may wear plastic wrap around their organ during episodes of frottage so that their trousers will not be stained by the ejaculate and they can proceed to work. This is also punishable under Sections 290 and 291 IPC.

VOYEURISM (SCOPTOPHILIA)

Voyeurism is amongst the most common of the paraphilias. Voyeurs seek situations/choose environments where they can see others disrobe, have sexual intercourse, or carry out some type of intimate sexual relationship such as peering into apartments or crowded residential neighbourhoods, etc. He may approach windows of homes, conceal himself in departmental stores or dressing rooms and in some cases, may install equipment in his own house so that he can "peep" on those who visit the house. That is why they are also sometimes called as **"peeping toms"**. A few voyeurs may prefer to observe their own wives being seduced by other men. In extreme cases, the pervert may get sexual gratification by inducing his wife to sexual intercourse with another person and by observing the same. This is sometimes called as **troilism**.

EXHIBITIONISM

As the name suggests, the term applies to acts mostly practiced by males and are characterised by recurrent compulsive urges to expose their genitals to another person, with or without performance of masturbatory acts. Occasionally, women may expose themselves in public. Frequently, they expose themselves many times to a series of women until their fear of apprehension, or guilt about the inappropriateness of their behaviour leads them to stop the activity. They may cease exposing themselves for weeks and/or months, and then initiating a new series of exposure. Exhibitionism is an obscene act punishable under Sections 290 and 291 IPC.

FETISHISM

The hallmark of fetishism is long-standing compulsive sexual interest in nonliving objects that are used to generate sexual excitement. In the ordinary human life, males do report having interest and attraction to object worn by their sexual partners such as brassieres, panties, garter belts, hose, and boots/socks, etc. However, fetishism is an extreme exaggeration of an attachment to objects and the individual may go to extremes to obtain fetishistic objects (e.g., breaking into houses to steal the things/objects). As the fetish object becomes more ingrained in an individual's sexual arousal pattern, he may hold or fondle with fetish object items and may masturbate in them. A fetichist who is fascinated by female hair may follow females with long hair and resort to cutting them. In extreme cases, relationship of fetishist with the fetish objects may become dominant, with conflict usually erupting between the partners as to the extent of intrusion of the fetish object.

TRANSVESTISM (EONISM)

This is the name given to a perversion in which the males find sexual pleasure in wearing the female garments. It is also known as eonism, the term being derived from the name of a Frenchman, Chevalier d'Eon de Beaumont, who practiced this perversion. This perversion is sometimes found in females who dress themselves in male attire. The clothes worn are usually underclothes and are usually expensive and alluring type. The manner in which the individual walks, sits, and stands reflects his gender motor behaviour.

Obscene telephone calls (telephone scatologia): Obscene telephone callers are generally heterosexual males who call known or unknown females to carry out sexually provocative conversations. Presently, they are likely to be apprehended, generally by the Caller ID technology that automatically lists the telephone number of the caller.

SATYRIASIS

It is excessive sexual desire, arousal and drive in case of males. These subjects are liable to commit sex offences or pervasive acts.

NYMPHOMANIA

Excessive sexual desire, urge or drive in a woman. Some of such women may turn lesbianists.

ZOOPHILIA

It involves activity relating to sexual arousal by repetitively carrying out sexual activities with animals. The ease of accessing animals for sexual purposes and the low risk of apprehension for such activity forms the basis for involvement in this undesirable activity.

COPROPHILIA AND UROPHILIA

These are perversions in which sexual excitement is provoked by the sight or odour of faeces or urine, etc.

Infundibulation/stigmatophilia: Boring holes, body piercing, and the wearing of rings through the skin. 'Prince Albert ring' inserted through the urethra of the penis around the glans though makes sexual intercourse impossible but is said to intensify the sensation during ejaculation induced by masturbation.

Narratophilia: Achievement of sexo-erotic arousal by listening to accounts of specific types of sexual activity.

Pictophilia: Achievement of sexo-erotic arousal by seeing sexually explicit pictures.

Hybristophilia: (Greek hybridzein = to commit outrage) is the sexual attraction to a person who has committed an outrageous crime. Alternatively, the partner may be encouraged by the hybristophile to commit a crime.

Chrematistophilia: Gaining of sexual pleasure from being charged for, or forced to pay for sexual services, even to the point of being robbed by the sexual partner.

Formicophilia: A special form of zoophilia, in which sexo-erotic arousal is achieved through small creatures, such as ants, snails, frogs or creeping insects, touching the genitalia, perianal region, or breasts.

Klismophilia: An erotic activity with a rubber fetish.

Mysophilia: Sexual arousal associated with smelling, touching or even chewing or sucking a smelly soiled garment.

Gerontophilia: The sexual preference for an aged partner.

“The injurious effects of a thing need not be construed as a result of the use of a bad thing; it is actually the result of abuse of the thing.”

Section 2 Forensic Toxicology

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Basic Considerations in Drugs/Chemicals

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30 CHAPTER

Basic Considerations in Drugs/Chemicals

After going through this chapter, the reader will be able to describe: Meaning of the terms pharmacology, drug, dose, therapeutic dose, effective dose, lethal dose, etc. | Routes of administration | Pharmacokinetics of drugs/chemicals | Pharmacodynamics of drugs/chemicals | Mechanism of action of drugs/chemicals | Analytical methods in toxicology | Classification of poisons

The term **Pharmacology** is derived from the Greek word 'Pharmacon' meaning 'drug' and 'Logos' meaning 'science'. So, in short, it can be regarded as a 'science of drugs'. However, in the present scenario, it is better to consider it as an integrative rather than autonomous science and as such, it borrows heavily from the knowledge of many allied scientific disciplines. Next come the questions—"What is a science?" and "What is a drug?". The word **science** has been derived from the Latin word 'scientia', meaning knowledge. In the broadest sense, science may be regarded as the application of all available mental and physical resources in order to better understand, explain, quantitate, and predict normal as well as unusual natural phenomena. The term **drug** has traditionally been defined as a chemical substance used for the treatment, cure, prevention or diagnosis of a disease in human beings or animals. However, this definition would not serve our purpose in the present times. WHO scientific group (1966) redefined the drug as, "any substance or product that is used or intended to be used to modify or explore physiological systems or pathological states for the benefit of the recipient". This definition fits well in the present scenario and encompasses contraceptives (which alter the physiological system to prevent pregnancy, which is not a disease), vaccines or antisera (which alter the pathological state by formation of antibodies), or general anaesthetics (which are not used for any cure or prevention of a disease), etc. **Dose** is the required amount of drug in weight, volume, moles or international units that is necessary to provide a desired effect. In clinical practice, it is called **therapeutic dose;** while for experimental purposes (in animals), it is called as **effective dose**. **LD_{50}** means a dose that is lethal to 50% of the subjects. **ED_{50}** means a dose that can provide 50% of the maximum response. **Therapeutic window** implies an optimal range of plasma concentration at which most of the patients experience the desired effects. **Therapeutic index** is expressed as LD_{50}/ED_{50}.

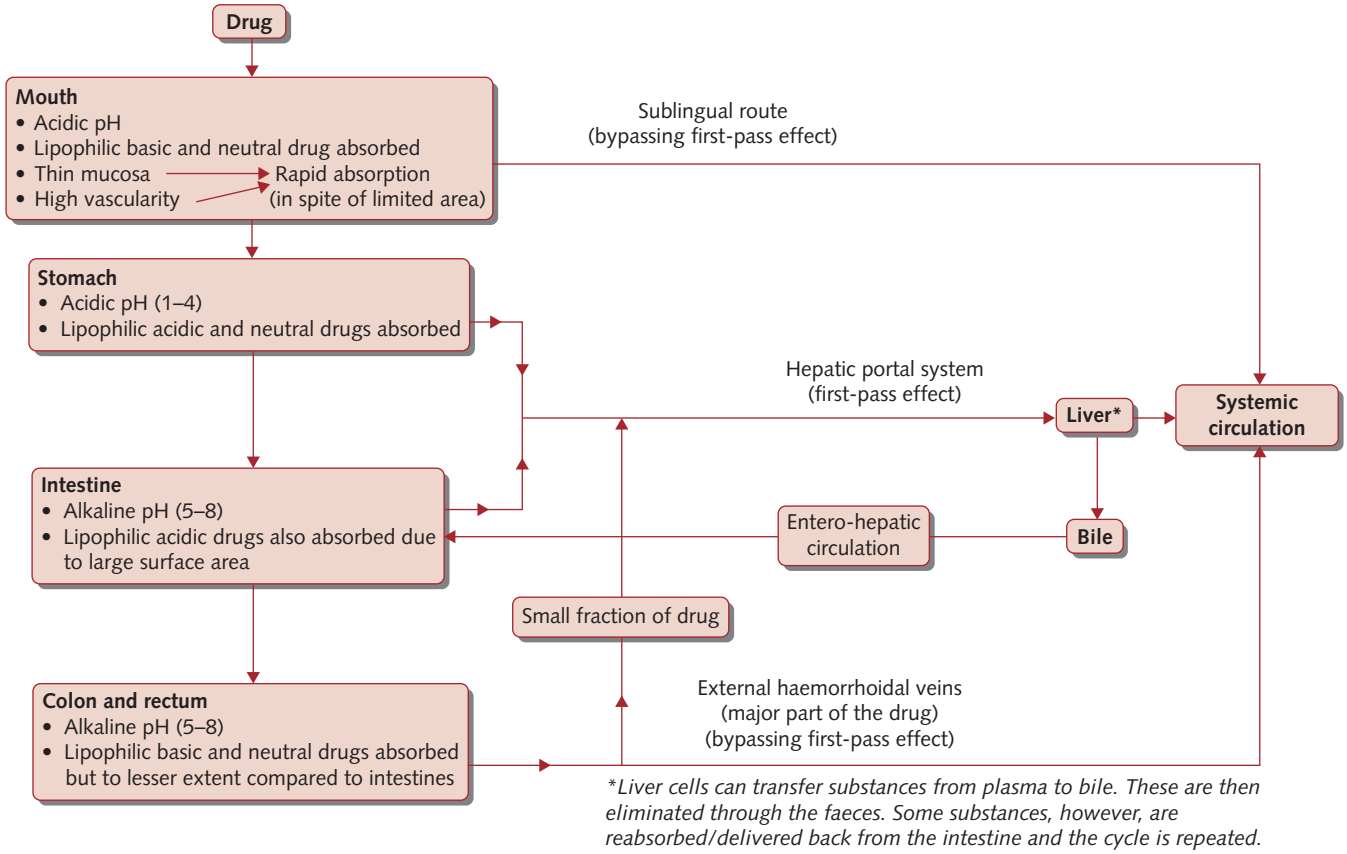
Routes of Administration

Drugs/chemicals are not administered as such but are formulated in different forms for administration. **Excipients** are pharmacologically inert substances that are added to the preparation either to add bulk to the active drug or to mask (or lessen) the unpleasant taste. **Vehicles** are substances that are used to dissolve or suspend the drugs to make them better applicable or more palatable. For the drug or chemical to exert its pharmacological effects, it needs to be carried to its site of action. This usually entails their translocation or biotransportation across the cell membranes, and the journey depends upon the different routes of their administration. Major routes are as follows:

(A) Enteral routes ('Enteron' means 'intestine'): These may include (Flowchart 30.1):

(a) **Oral** (briefly speaking, the absorption of drugs/chemicals from GIT is mainly by passive diffusion through the lipid sheath)—It is most commonly used method, being safe, convenient and painless. However, action is slow and erratic as they have to pass through portal circulation (first pass degradation) to reach systemic circulation.

(b) **Sublingual**—The drug/chemical is placed beneath the tongue or crushed in mouth and spread over the buccal mucosa. It shows rapid absorption and directly passes into the systemic circulation, bypassing the portal circulation.



Flowchart 30.1 Enteral route of drug administration.

(c) **Rectal**—It can be useful in patients/victims having nausea and vomiting. First pass degradation is largely bypassed as a major portion of the drug is absorbed from the external haemorrhoidal veins.

(B) Parenteral routes (Routes other than enteral are called parenteral): These may include:

(a) **Intravenous**—The drug/chemical enters into systemic circulation directly, bypassing first pass degradation, i.e. quick onset of action and lesser dose requirement to achieve the desired plasma concentration.

(b) **Inhalation**—The drug/chemical when given in vaporised form or as a spray of suspended microfined particles is absorbed by simple diffusion from the mucous membrane of trachea and lungs. Absorption is faster and onset of action is quick due to larger surface area of the alveoli and high vascularity.

(c) **Intramuscular**—The drug/chemical is injected into the muscle. Absorption is rapid as compared to oral route and 'depot injections' can also be given for obtaining sustained effects.

(d) **Subcutaneous**—The drug/chemical is injected into the subcutaneous tissue under the skin. It shows slower absorption for a longer period as compared to intramuscular. Depot injections can also be given. Further subcategorisation may include:

(i) **Intradermal**, i.e. the drug/chemical is injected into the outer layers of the skin. Diagnostic tests and allergic sensitisation testing is done using this route.

(ii) **Pellet and biodegradable implants**, i.e. the drug/chemical is implanted under the skin in the form of a pellet or packed in biodegradable tubes to provide uniform but slow release of the chemical.

(iii) **Dermojet injections**, i.e. subcutaneous needle-less injection of a drug/chemical by means of a high velocity jet projected through a microfined orifice.

(C) Routes to achieve high local concentration: Some routes that are used to achieve high local concentration of certain drugs/chemicals, while minimising their systemic absorption, may include the following:

(a) **Intraperitoneal**—Drug/chemical is injected into the peritoneal space, showing rapid absorption due to larger surface area.

(b) **Intrathecal** (intraspinal)—The drug/chemical is injected into subarachnoid space. After diffusing from the lumbar sac, the drug/chemical passes into the subarachnoid space, bypassing blood–brain barrier and blood–CSF barrier.

(c) **Epidural**—The drug/chemical is injected through a vertebral interspace between dura of the spinal cord and the lining of the spinal canal.

(d) **Intramedullary**—The drug/chemical is injected into bone marrow. Action is very fast, as the vascular spaces of bone marrow communicate directly with the large veins.

(e) **Intracardiac**—The drug/chemical is injected into the heart through the left fourth intercostal space.

(f) **Intra-articular**—The drug/chemical is injected into the joint space to ensure higher concentration in a joint.

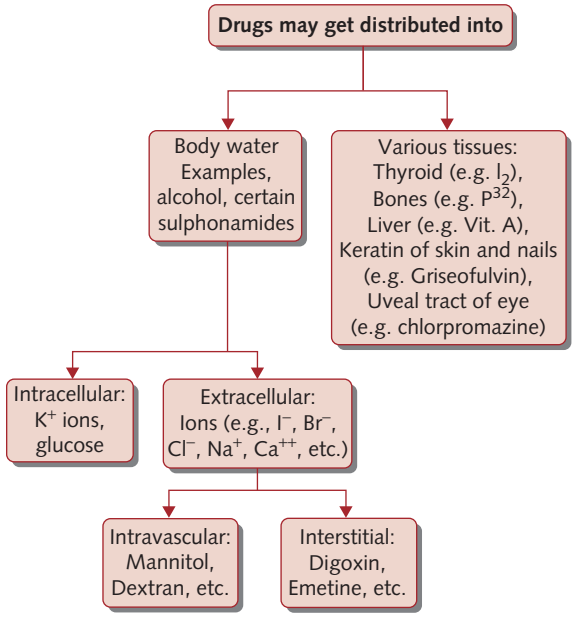
(D) Topical routes (Absorption of most drugs/chemicals through the intact skin is poor as the keratinised epidermis behaves like a barrier to the permeability. However, the underlying dermis is quite permeable to many lipid-soluble drugs/chemicals):

(a) **Transdermal**—Adhesive matrix containing drug/chemical is usually applied to chest, abdomen or mastoid region. The chemical is delivered at the skin surface through diffusion, for percutaneous absorption into the circulation.

(b) **Conjunctival**—Ointments or isotonic aqueous solutions of drugs/chemicals are instilled onto the conjunctivae for local effects.

(c) **Aerosols**—The drug dissolved in a liquid is put inside a cylindrical container and is then fitted with a propellant gas under pressure. A push at the valve releases a "measured drug" through a microfined orifice in the form of mist.

(d) **Suppositories, pessaries and bougies, etc.**— These contain drug mixed with glycerin or gelatin or hard soap, etc. These remain solid at room temperature but become soft and absorbable at body temperature.



Flowchart 30.2 Once absorbed into the blood, a drug/chemical is distributed to all organs including those not relevant to its pharmacological or therapeutic effect. It may be evenly, or unevenly or selectively distributed in different body compartments. Barriers, namely, "blood-brain barrier" and "blood-cerebrospinal fluid barrier" exist between the blood and the brain fluid, and blood and CSF, respectively. In the brain, endothelial cells of the capillaries are joined by so-called tight junctions rather than having extensive slit-pores between them as is the case in most other capillaries of the body. In addition, these brain capillaries are also enveloped by less permeable cells known as the glial cells.

Pharmacokinetics (What the Body does to the Drug/Chemical)

Absorption means the movement of the drug/chemical into the blood stream from its site of administration. This is followed by **distribution,** i.e. movement of molecules of the drug/chemical from the blood into the tissues (Flowchart 30.2).

Biotransformation means enzyme-catalysed chemical transformation of drugs/chemicals within the living organism. The metabolites thus formed are much less lipid soluble, hence not re-absorbed from the renal tubules and thus are finally excreted. Various pathways are described below:

- **Chemical pathways** include the following:
  - Phase I reactions—These are degradative in nature, i.e. drug is diminished to a smaller polar/nonpolar metabolite by introduction of a new group, mainly through microsomal system. Examples may include oxidation, reduction or hydrolysis.
  - Phase II reactions—These are conjugative in nature. Usually through microsomal, mitochondrial or cytoplasmic enzymes.
- **Enzymatic induction:** Repeated administration of certain drugs/chemical causes stimulation or induction or growth of smooth endoplasmic reticulum leading to increased microsomal enzyme activity and accelerated metabolism. Occurs mainly in the liver.
- **Enzymatic inhibition:** One drug inhibits metabolism of another, causing increase in circulating levels of the slowly metabolised drug and therefore, prolongation and potentiation of its pharmacological effects.

Elimination of drugs/chemicals from the body takes place either in unchanged form or as water-soluble metabolites. Both the processes of metabolism and excretion are essential for the elimination of drugs/chemicals. **Major routes** of excretion are (*i*) renal, (*ii*) biliary, (*iii*) faecal, and (*iv*) alveolar. **Minor routes** of excretion are (*i*) milk, (*ii*) skin, (*iii*) hair, (*iv*) sweat, and (*v*) saliva.

The rate and the pattern of drug elimination ensues any of the elimination kinetics as shown in Table 30.1. Figure 30.1 depicts the processes involved in renal excretion.

Table 30.1 Different 'Orders' of Elimination Kinetics of Drugs/Chemical

First-order kinetics	Zero-order kinetics	Mixed-order kinetics (Michaelis-Menten kinetics)
Majority of drugs obey this order elimination kinetics	Hardly a few drugs obey this order elimination kinetics, e.g. ethyl alcohol metabolism follows this at virtually all plasma concentrations	Some important drugs (e.g., phenytoin, digoxin, warfarin, etc.) obey this order elimination kinetics
• A constant fraction of the drug is eliminated at a constant interval of time • The rate of drug elimination is directly proportional to the plasma concentration • $t_{½}$ of a drug following first-order kinetics would always remain constant irrespective of the dose	• A constant or fixed quantity of the drug is eliminated per unit time • The rate of elimination proceeds at a fixed pace, independent of plasma concentration of the drug • $t_{½}$ of such a drug will never be constant	It is a dose-dependent kinetics where smaller doses are handled by first order kinetics but as the plasma concentration reaches higher values (due to increase in the dose), the rate of drug elimination becomes zero order because the metabolising enzymes or the elimination processes get saturated at higher concentration

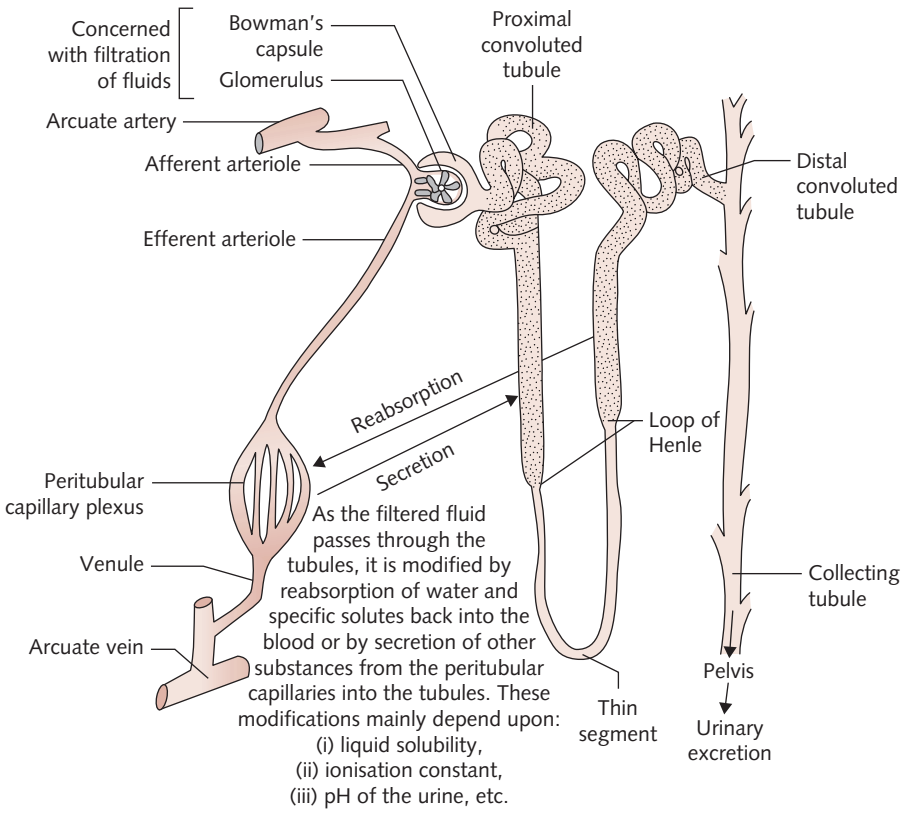


Fig. 30.1 Basic kidney processes that determine the composition of urine. Urinary excretion rate of a substance is equal to the rate at which the substance is filtered minus its reabsorption rate plus the rate at which it is secreted from the peritubular capillary blood into the tubules.

Pharmacodynamics (What a Drug/Chemical does to the Body)

Effect of a drug denotes the type of response produced by the drug, whereas **action** denotes how and where the effect is produced.

Effects of a drug may be classified primarily into desirable/beneficial effects and undesirable/untoward or adverse drug responses (ADRs). The latter can further be studied under the subcategories, viz.:

(A) Expected undesirable effects (Type A ADRs)

(a) **Side effects**—Undesirable effects seen even with therapeutic doses of the drug and are usually mild and manageable.

(b) **Secondary effects**—Indirect consequences of the main pharmacodynamic action of the drug.

(c) **Toxicity**—Exaggerated form of side effects that occur predictably either due to overdoses or after prolonged use of the drug.

(B) Unexpected undesirable effects (Type B ADRs)

(a) **Drug allergy**—An acquired, abnormal immune response to a substance that does not normally cause a reaction. This may further be of various types:
  (i) Type I (immediate type) through IgE,
  (ii) Type II (auto or accelerated) through IgG and IgM,
  (iii) Type III (delayed) through predominantly IgG, and
  (iv) Type IV (cell mediated) through T lymphocytes.

(b) **Genetically determined ADRs,** i.e. pharmacogenetic variations.

(c) **Idiosyncratic drug responses**—Unexplained or poorly explained harmful and sometimes fatal reactions that occur in a small minority of individuals.

Modified drug effects after repeated administration of a single drug can be studied under different heads, i.e. **drug tolerance** —the need to increase the dose in order to produce the pharmacological response of equal magnitude and duration. [Cross tolerance implies tolerance among drugs belonging to the same category, whereas reverse tolerance implies greater response to a given dose after repeated use.] Drug tolerance can further be subclassed as:

(A) Innate (natural or congenital)—Genetically determined lack of sensitivity to a drug.

(B) Acquired—This is further subdivided into:

(a) Drug disposition tolerance—due to pharmacokinetic reasons, i.e. the drug reduces its own absorption or increases its own metabolism through microsomal enzyme induction.

(b) Cellular adaptive tolerance—may be due to drug-induced changes in the receptor density or impairment in receptor coupling to signal transduction pathways.

(c) Tachyphylaxis—acute development of tolerance after a rapid and repeated administration of a drug at shorter intervals, which may be due to gradual depletion of agonist from storage sites or change in sensitivity of target cells.

Effects of a drug may be modified after concurrent administration of two different drugs and can be studied under the following different heads:

(A) Summation—When two drugs elicit the same response but with different mechanisms, their combined effect is equal to the sum of their individual effects.

(B) Additive effects—Both drugs acting by the same mechanism, i.e. $a+b=(a+b)$.

(C) Synergism—Combined effect is more than the algebraic sum of the individual effects, i.e. $a+b>(a+b)$.

(D) Drug antagonism—Conjoint effect is less than the sum of the effects of individual drugs, i.e. $a+b<(a+b)$. This can be brought about by various mechanisms, namely:

(a) Chemical antagonism—Chemically opposite drugs.

(b) Physiological/functional—Two agonists acting at different sites counterbalance each other by producing opposite effects.

(c) Biological/pharmacokinetic—When one drug affects the absorption, metabolism or excretion of the other drug and effectively reduces the concentration of the active drug at its site of action.

(d) Receptor antagonism—Two drugs acting on the same receptor.

(E) Drug combination—It may be brought about by:

(a) Drug–drug interactions—An alteration in the effectiveness or toxicity of one drug due to the action of another simultaneously administered drug.

(b) Fixed dose combinations.

Mechanisms of Action of Drugs/ Chemicals

RECEPTOR MEDIATED MECHANISM

A receptor is a specific macromolecular protein (membrane bound or intracellular), which is capable of binding with the specific group(s) of the drug/endogenous substance. [Binding may be reversible (H bonds, Van der Waal's bonds, electrostatic bonds, etc.); irreversible (covalent bonds), or stereoselective (optical isomers). The term **affinity** denotes capability of a drug to form a complex with its receptor, whereas **efficacy**/ intrinsic activity denotes the ability of a drug to trigger response after making the drug-receptor complex.] On the basis of affinity and efficacy, drugs can broadly be classified as:

Agonists—Drugs having high affinity as well as intrinsic activity and therefore, can mimic the effects of the endogenous substances after combining with the receptor.

Antagonists—Drugs having only the affinity but no intrinsic activity. These drugs bind to the receptors but do not mimic, rather block or interfere with the binding of an endogenous agonist.

Partial agonists—Drugs having full affinity to the receptor but with low intrinsic activity and hence, partly as effective as agonists.

Inverse agonists (negative antagonists)—Drugs having preferential affinity for the inactive state of the receptor and therefore, producing an effect opposite to that of an agonist even in its absence.

NONRECEPTOR MEDIATED MECHANISM

It can further be categorised as:

(A) By chemical action

(a) Neutralisation (antacids neutralising gastric acid).

(b) Chelation (combining of metallic ions with certain heterocyclic ring structures so that the ion is held

by chemical bonds from each of the participating rings).

(c) Ion exchange (these are substances that replace certain negative or positive ions that they encounter in solution).

(B) By physical action

(a) Osmosis (the passage of solvent through a semipermeable membrane that separates solutions of different substances).

(b) Adsorption (attachment of a substance to the surface of another material).

(c) Protectives (an agent that mechanically protects the part of the body to which applied).

(d) Demulcent (an agent that soothes the part or softens the skin to which it is applied).

(e) Astringent (an agent that has a constricting or binding effect, i.e. one that checks haemorrhages or secretions by coagulation of proteins on a cell surface).

(C) By counterfeit or false incorporation mechanisms: for example, replacement of PABA by sulfa drugs thus making the folic acid synthesised by bacteria ineffective.

(D) By protoplasmic poison effect, causing death of bacteria.

(E) By formation of antibodies (for example, vaccines).

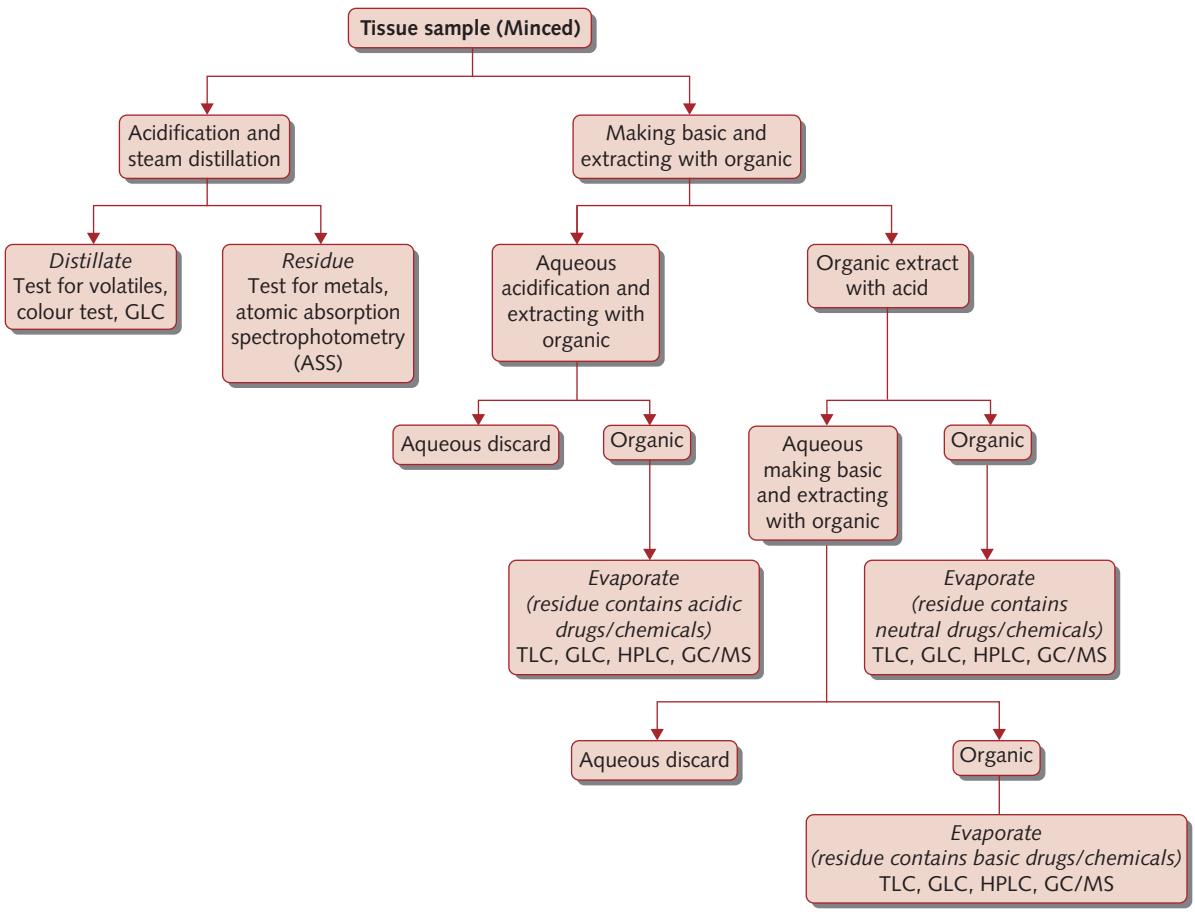
(F) Through placebo action—Placebo is a pharmacodynamically inert and harmless substance given to a person in a form similar to the actual medicament and that gives relief from the subjective symptoms of the patient.

Analytical Methods Used in Toxicology

QUALITATIVE ASSAYS

Colour tests: Using various reagents, information is gathered depending upon the development of colouration related to drug(s).

Thin layer chromatography (TLC): Chromatography is a separation technique, which utilises partitioning characteristics of different chemical substances (may be a solid, liquid or gas) in different media. TLC involves the movement by capillary action of a liquid phase (usually an organic solvent)



Flowchart 30.3 Schematic presentation for separation of toxic substances/chemicals from tissues by steam distillation and differential solvent extraction. There is no substitute for an unequivocal identification of a specific chemical substance that is demonstrated to be present in tissues from the victim at a sufficient concentration to explain the death or injury with a reasonable degree of scientific probability or certainty.

through a uniform layer of stationary phase (usually silica gel) held on a rigid support. Compounds are separated by partition between mobile and stationary phase.

QUANTITATIVE ASSAYS (Flowchart 30.3)

Gas Chromatography (GC): It offers a way of simultaneously separating, identifying and measuring drugs and other organic poisons. Most commonly employed to quantitate blood levels of volatile liquids such as ethanol, ethylene glycol and methanol.

High pressure liquid chromatography (HPLC): A high pressure (1000–6000 psi) pump facilitates movement of the specimens through the columns packed with chromatographic adsorbents. The effluent steam passes through a detector, usually an ultraviolet spectrophotometer and the appearance of the drug in the solvent is signaled by a recorder peak (the size of the peak is proportional to the concentration of the drug in the sample). It can be used to separate and analyse complex mixtures.

Mass spectrometry (MS): This is usually combined with gas chromatography (GC-MS) and is considered to be the best technique for quantitative analysis of a wide variety of chemicals. Its capital as well as operational costs restrict its use greatly.

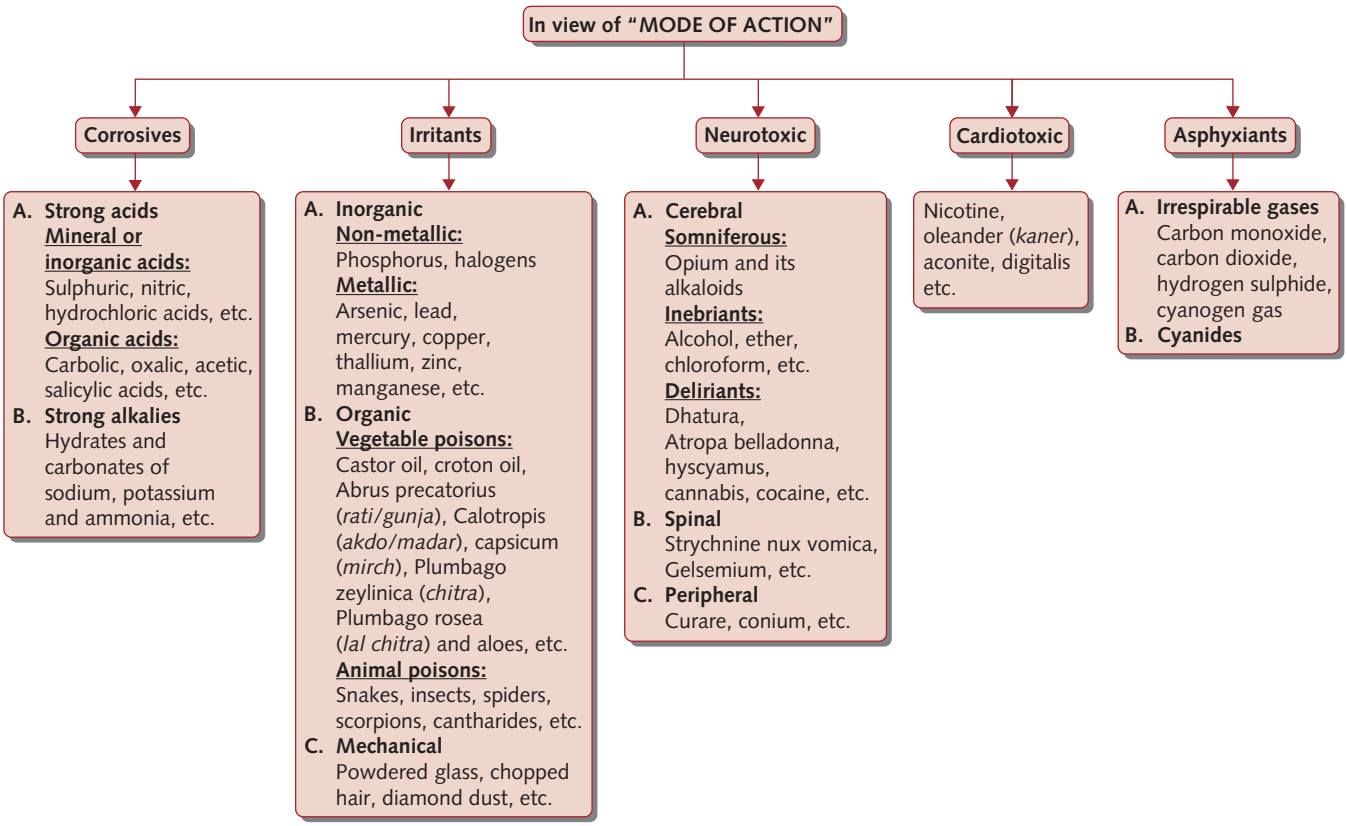
Radioimmuno assay (RIA): It involves mixing of known quantities of drug specific antibody with known amount of radioactively labelled drug that allows analysis of the precipitate with a gamma counter. Usually employed for detection of drugs in extremely low blood concentrations (e.g., ***Cannabis***, LSD, paraquat, etc.).

Enzyme-mediated immuno assay (EMIA): It is based on the principle that the amount of drug present is proportional to the inhibition of an enzyme-substrate reaction. It is used mainly in emergency because of its speed in providing information. It eliminates the complex separation phase necessary in RIA.

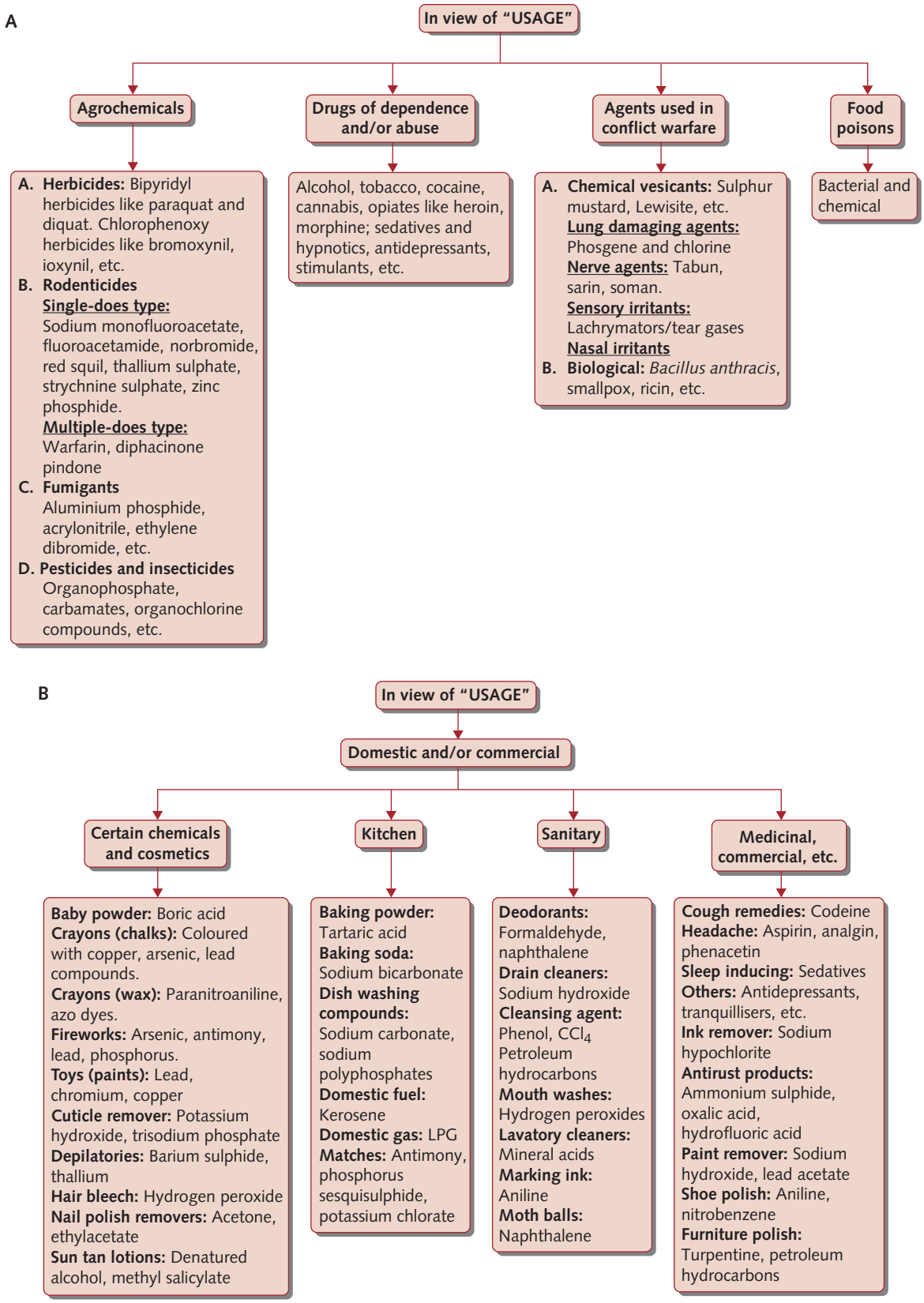
Atomic absorption spectrophotometry (AAS): It is considered as the best method for detecting inorganic elements (arsenic, lead, mercury, thallium, etc.). However, the equipment is costly. Inductively coupled plasma atomic emission spectroscopy (ICPAES) is a new development that allows simultaneous multi-element analysis.

Neutron activation analysis (NAA): It is based on the principle that many substances become radioactive when exposed to bombardment by neutrons. The induced radioactivity is highly specific of the elements contributing to it. However, it requires the use of a nuclear reactor, which is very expensive.

Ultraviolet spectrophotometry (UVS): It is based on the principle that many drugs when in solution will absorb UV radiation. The degree of absorption depends upon the chemical structure of the drug, its concentration in the solution and the wave length of the ultraviolet rays (UVR).



Flowchart 30.4 Classification of poisons according to mode of action.



Flowchart 30.5 Classification of poisons on the basis of usage.

Table 30.2 In View of 'Manner of Poisoning'

Characteristics	Suicidal	Homicidal
Accessibility to poison	Easy and free	Not particular
Cost	Cheap	Not particular
Colour and taste	Tasteless or of pleasant taste	Colourless, odourless, tasteless
Solubility in food/drinks	Not particular	+ve
Signs and symptoms	Nil/few	Resemble disease(s)
Onset of action	Quick	Slow
Clinical diagnosis	Difficult	Difficult
Antidote availability	Nil	Nil
Metabolism and excretion	Not particular	Rapid
Death	Painless	Definite
Postmortem detection	Not particular	Difficult
Examples	Aluminum phosphide, organophosphate and carbamate compounds, organochloro compounds, opium, barbiturates, potassium cyanide, copper sulphate, etc. (according to availability and use in the particular place/region)	Aconite, thallium, organophosphate and carbamate compounds, organochloro compounds, and arsenic, etc.

Of all the homicides, those committed through poison are the most difficult to discover, to prove objectively. However, the background of proven poison murders leads one to say that homicidal poisoning is usually an intimate or 'household' crime, customarily preferred when the victim and the poisoner share the same domicile, an arrangement that furnishes the necessary proximity and privacy for the lethal potion to be administered (in the absence of witnesses).

Classification of Poisons

No classification of poisons is entirely satisfactory as many poisons fall into more than one group. Accordingly, classification/placement depending upon the mode of action, usage, and nature of poisoning has been furnished so as to make it convenient for the readers to have some immediate idea about the antecedents/scenario of the substance under reference (in view of 'Mode of Action' Flowchart 30.4; in view of 'Usage' Flowcharts 30.5A and 30.5B and in view of 'Manner of Poisoning' Table 30.2).

ACCIDENTAL POISONING

The term 'accident' may be considered to imply an unintended, undesigned and unexpected happening; especially one resulting in loss or injury. The 'designation' of accident commonly reflects involvement of human, instrumental, toxic, and/or environmental conditions leading to its occurrence. In the words of Stephen, "An accident is an incident that takes place out of the ordinary course of events, which no man of ordinary prudence could anticipate and provide measures to check it". It may be exemplified like this: A intending to kill B, mixes poison in B's food but C, his cousin, takes the food and dies. Here C's death is an accidental act in the sense of being an act of misfortune. However, A is not exempted from killing C despite the fact that he never intended to kill him, since the act of killing a human being is an illegal act; A is as much liable for killing C as he would have been had B taken the poison, by invoking the principle of transfer of malice.

Circumstances of accidental poisoning, therefore, defy any circumscribed description. Following may be cited as some instances:

- Contamination of food/drink
- Consuming a poison for a medicine by mistake
- Wrong medication, over-medication
- Snake bite
- Bite(s) by honey bee, wasp, spider, scorpion, etc.
- Poisoning at home, gardens/agricultural fields, etc.
- Road side poisoning from some stupefying agents like *dhatura*, cannabis, chloral hydrate, etc.
- Abortifacients like ergot, calotropis, plumbago, etc.
- Aphrodisiacs like cantharides, cocaine, alcohol, opium, cannabis, etc.

31 CHAPTER

Intricacies of Forensic Toxicology

After going through this chapter, the reader will be able to describe: Historical background of forensic toxicology | Concept and scope of toxicology | Definition of 'poison' and its implications | Statutes on drugs/poisons in India | Sections of IPC concerned with poisons and poisoning | Factors modifying action of drugs/chemicals | Concept of fatal dose | Evidence of poisoning in the living and the dead | Techniques of obtaining samples and interpretation of results | Relative toxicity of drugs/chemical

Forensic Toxicology

This section is not intended to be a complete treatise on Forensic Toxicology, but rather an overview of some important aspects and characteristics of chemical injuries to the human beings as they pertain to Forensic Medicine. The entire discipline of toxicology is a paramount science and influences human life right from inception and even after death, should exhumation be required.

Poisoning has been used by man for murder and suicide as long as recorded history. The Egyptians and the Greeks knew that certain plants have capacity to inflict death on a victim. The Greek philosopher Socrates was executed by the State through the use of hemlock, a plant poison. Around the year 200 B.C., a Greek handbook on poisons was written; it discussed in an informed way the poisonous qualities of opium, henbane, some fungi, aconite and other substances. The author divided poisons into those that act quickly as opposed to those that act slowly.

In ancient India, poisons were known (arsenic, aconite and opium). They were used by women to get rid of oppressive husbands. The *Mahabharata*, which is usually ascribed to the fifth or sixth century B.C., mentions that Bhim Sen, the Hindu Samson, was poisoned by his cousin Duryodhan whom he had defeated in a duel. In a semi-historical legend of mid-India, it is narrated that the grandfather of Asoka, Chandra Gupta, a contemporary of Alexander the Great, sent to the latter monarch in the guise of a present, a fascinating girl who was a 'poison-maiden' fed on poison until she was so saturated with venom that her embrace would prove fatal to an ordinary mortal. The mere conception of the idea of such a Borgia-like siren would imply considerable familiarity with poisoning. In Mohammedan times, poisoning was a recognised form of capital punishment, and was unusually rife in harem intrigues and against political foes and prisoners.

The first textbook on poison was written in 1814 by Matthew Joseph Orfila, a Spanish chemist, who is considered to be the **Father of Toxicology**. Orfila extracted arsenic from human tissues using a procedure for identification, developed several years before by James Marsh. This evidence was used in the court (1840) to convict Marie Lefarge of a homicidal poisoning. This was the first time that the toxicological data had been used as evidence in the trial. Orfila was also the instructor of Robert Christison, a British physician, who is considered to be the **'First British Toxicologist'**. Alexander O Gettler, Chief Forensic Toxicologist of the laboratory at New York (established in 1918), is considered to be the **'Father of American Toxicology'**.

A VIEWPOINT

The poisoner is a murderer who has gone through a long and deliberate process of cold premeditation. It is a crime as deliberate, as cold-blooded, as malicious and as premeditated as is a covert bombing. This viewpoint may be illustrated by the following anecdote:

A representative of a foreign government was causing severe inroads into security and covert operations in a part of Europe. It was arranged for him to meet with a supposed government official in order to discuss further subversion. The 'official' was an officer of the threatened country under a low-profile cover. The meeting was held at a modest restaurant. The food was good and the coffee was excellent and plentiful. The next day, there was another meeting; a modest meal, a small amount of wine, and a sheaf of documents passed over to the foreign representative from the 'official'. The two parted and official disappeared. Back home, everyone knew he had

taken a brief business trip to the nation's capital and had returned on schedule. Two days after the final meeting of the pair, the foreign representative had flown to Vienna, where he suddenly became violently ill with nausea, vomiting and diarrhoea. He showed severe rhinitis and bronchitis, and a temperature of about 100° F. He complained of severe pains in the back, arms and legs. He was in turn anxious, uneasy and then delirious. His sleep pattern was erratic; the bronchitis became worse. In a week he was dead. At autopsy, the doctor noticed that the hair of the head, pubis and axilla pulled out very easily. The diagnosis was severe polyneuritis complicated by fulminating pneumonia. Because the pathologist seemed so sure of his diagnosis, no chemical tests were performed on the tissues or body fluids of the deceased.

One part of the story was never known to the attending physician. During both meals with the victim, the official had excused himself 'to visit the latrine'. Instead he had spoken to the headwaiter about his friend's 'diabetes', giving the headwaiter several tablets to put into the coffee and wine of his friend to counteract the excess sugar. He explained to the waiter that his friend was embarrassed about his condition and hated to make a public display of his disability. For $10 bill each time, the cooperation of the headwaiter was ensured. The tablets were fast-dissolving forms of thallium acetate, a delayed action poison. There are other poisons that can be used in a variety of ways for evildoing. Fortunately, the state of the art in analytical chemistry has advanced to a degree that virtually any poison can be identified quantitatively if adequate tests are run.

Concept and Scope of Toxicology

The word 'toxicology' is derived from the Greek word 'Toxicon', which was used as a poisonous substance to arrowheads. The term connotes unwanted effects created by certain substances. The extent of the effect may be varying. It may be trivial or grave. Pruritic skin rash may be considered as a trivial toxicity, whereas respiratory depression may be considered grave. However, conventionally the term toxicity points towards a grave effect. The damage produced by the substance may lead to death of the individual, and the effect is then described as fatal. The substance inflicting toxic effect may be a drug, an insecticide or pesticide or any chemical substance in the environment. (Methyl isocyanate leakage at the Union Carbide plant in Bhopal in 1984 resulted in high mortality and morbidity.) Major subspecialties of toxicology may be enumerated in Table 31.1.

Chemical injury may be considered as the destruction or alteration in cellular and organic function produced by nonbiologic substance. In the strictest sense, any substance causing injury to the human life by chemical reaction produces a chemical injury. The statistics stand as a witness to the growing rate of chemical death. The term **poison** may need description at this juncture. A poison is any substance that on introduction into the living body or on bringing into contact with any part thereof will produce ill effects or death by its local or systemic action or both. In fact, every substance is theoretically capable of producing toxicity and every drug is potentially a poison when used erratically. **Paracelsus,** the sixteenth century Swiss chemist, was right when he said that there is no substance in this world that is not a poison. It all depends upon the dose and the mode of administration. **Forensic toxicology** is a hybrid of analytical chemistry and fundamental toxicologic principles. It is concerned with the medicolegal aspects of the harmful

Table 31.1 Scope and Functional Components of Toxicology

Specialty	Major functional components
Clinical	Causation, diagnosis and management of established poisoning in humans
Pharmacological	Assessing the toxicity of therapeutic agents
Forensic	Establishing the cause of death or intoxication in humans by analytical procedures and with particular reference to legal implications
Occupational	Assessing the potential of adverse effects from chemicals in the occupational environment and the recommendation of appropriate protective and precautionary measures
Environmental	Assessing the effects of toxic pollutants, usually at low concentrations, released from commercial and domestic sites into their immediate environment and subsequently widely distributed by air and water currents and by diffusion through soil
Regulatory	Administrative function concerned with the development and interpretation of mandatory toxicology testing programmes and with particular reference to controlling the use, distribution and availability of chemicals used commercially and therapeutically
Laboratory	Design and conduct of in vivo and in vitro toxicology testing programmes
Product	Assessing the potential for adverse effects from commercially produced chemicals and formulations and recommendation on use patterns and protective and precautionary procedures
Aquatic	Assessing the toxicity on aquatic organisms of chemicals discharged into marine and fresh water
Toxinology	Assessing the toxicity of substances of plant and animal origin and produced by pathogenic bacteria

effects of chemicals upon the human beings. It may be broadly labelled as 'toxicology in the courtroom'. The realm of forensic toxicology of necessity is no longer limited to criminal proceedings. Environmental toxicology, occupational toxicology and pesticide toxicology are becoming more prominent in the legal process. The forensic aspects of toxicology are needed in these new activities, the level of expert testimony being dismal at the moment.

Statutes on Drugs/Poisons in India

Several legal provisions have been enacted for the regulation and control of manufacture, sale and possession of drugs and poisons. Some important ones are discussed as follows.

DRUGS AND COSMETICS ACT (1940)

The Drugs Bill having been passed by the Central Legislative Assembly, received the assent of the Governor General on 10th April, 1940; and it came on the Statute Book as the Drugs Act, 1940. However, after amendment in 1962, the words 'and Cosmetics' were added and therefore, now it stands as 'The Drugs and Cosmetics Act, 1940'. Some definitions of common usage as appearing under this Act are as under:

- **Drug** includes:
  - all medicines for internal or external use of human beings or animals and all substances intended to be used for or in the diagnosis, treatment, mitigation or prevention of any disease or disorder in human beings or animals, including preparations applied on human body for the purpose of repelling insects like mosquitoes;
  - such substances (other than food) intended to affect the structure or any function of the human body or intended to be used for the destruction of vermin or insects which cause disease in human beings or animals, as may be specified from time to time by the Central Government by notification in the Official Gazette;
  - all substances intended for use as components of a drug including empty gelatin capsules and
  - such devices intended for internal or external use in the diagnosis, treatment, mitigation or prevention of disease or disorder in human beings or animals, as may be specified from time to time by the Central Government by notification in the Official Gazette.
- **Cosmetic means** any article intended to be rubbed, poured, sprinkled or sprayed on, or introduced into, or otherwise applied to the human body or any part thereof for cleansing, beautifying, promoting attractiveness, or altering the appearance, and includes any article intended for use as a component of cosmetic.
- **Manufacture** in relation to any drug (or cosmetic) includes any process or part of a process for making, altering, ornamenting, finishing, packing, labelling, breaking up or otherwise treating or adopting any drug (or cosmetic) with a view to its sale or distribution but does not include the compounding or dispensing of any drug, or the packing of any drug or cosmetic in the ordinary course of retail business and 'to manufacture' shall be construed accordingly.
- **Misbranded drugs:** A drug shall be deemed to be misbranded:
  - if it is so coloured, coated, powdered or polished that damage is concealed or if it is made to appear of better or greater therapeutic value than it really is; or
  - if it is not labelled in the prescribed manner; or
  - if its label or container or anything accompanying the drug bears any statement, design or device that makes any false claim for the drug or which is false or misleading in any particular.
- **Adulterated drugs**

  A drug shall be deemed to be adulterated:
  - if it consists, in whole or in part, of any filthy, putrid or decomposed substance; or
  - if it has been prepared, packed or stored under insanitary conditions whereby it may have been contaminated with filth or whereby it may have been rendered injurious to health; or
  - if its container is composed in whole or in part of any poisonous or deleterious substance that may render the contents injurious to health; or
  - if it bears or contains, for purposes of colouring only, a colour other than the prescribed; or
  - if it contains any harmful or toxic substance that may render it injurious to health; or
  - if any substance has been mixed therewith so as to reduce its quality or strength.
- **Spurious drugs**

  A drug shall be deemed to be spurious:
  - if it is imported under a name that belongs to another drug; or
  - if it is an imitation of, or is a substitute for, another drug or resembles another drug in a manner likely to deceive or bears upon it or upon its label or container the name of another drug unless it is plainly and conspicuously marked so as to reveal its true character and its lack of identity with such other drug; or
  - if the label or container bears the name of an individual or company purporting to be the manufacturer of the drug, which individual or company is fictitious or does not exist; or
  - if it has been substituted wholly or in part by another drug or substance; or
  - if it purports to be the product of a manufacturer of whom it is not truly a product.

Cognisance of Offences

No prosecution under these provisions shall be instituted except by an Inspector (or by the person of aggrieved or by

a recognised consumer association whether such person is a member of that association or not).

Penalty for Manufacture or Sale of Drugs in Contravention to Legal Provisions

In case of an adulterated, spurious or any drug that when used by any person for or in the diagnosis, treatment, mitigation or prevention of any disease or disorder is likely to cause ill health or is likely to cause such harm on his body as would amount to grievous hurt within the meaning of Section 320 of IPC, shall be punishable with imprisonment for a term that shall not be less than 5 years but that may extend to a term of life and with fine that shall not be less than ₹ 10,000.

DRUGS AND MAGIC REMEDIES (OBJECTIONABLE ADVERTISEMENT) ACT (1954)

The objective of this Act is to ensure that ethical standards are maintained when drugs are advertised by the manufacturers. Advertisements offending decency or morality can be banned under this Act. Also, those claiming magical powers for certain drugs, e.g. enhancement of potency, miraculous cure for some incurable diseases, etc., can be taken to task. Magical remedies include the use of talismans or charms such as "*mantras*", "*kavachas*", etc.

NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES ACT (1985)

In India, the statutory control over narcotic drugs is exercised through a number of Central and State enactments. Central Acts, namely, the Opium Act (1857), the Opium Act (1878) and the Dangerous Drugs Act (1930) were enacted long time ago. With the passage of time and the developments in the field of illicit drug traffic and drug abuse at national and international level, many deficiencies were noticed in those existing laws and an urgent need was felt for the enactment of a comprehensive legislation on narcotic drugs and psychotropic substances and make provisions for the implementation of international conventions relating to narcotic drugs and psychotropic substances to which India has become a signatory. This Act of 1985, therefore, repealed all the previously mentioned Acts. However, in recent years, India had been facing a problem of transit traffic in illicit drugs. The spillover from such traffic has caused problems of abuse and addiction. Hence, the Act of 1985 was again amended in 1989 and 2001. The Amendment Act is applicable to the whole of India and also applies to all citizens of India who are outside the territory of India and to all persons on ships and aircrafts registered in India, wherever they may be. (This has been effected in pursuance of the UN convention against Illicit Traffic in Narcotic Drugs and Psychotropic Substances, 1988.)

Narcotic drug means coca leaf, cannabis (hemp), opium poppy straw and includes all manufactured drugs.

Opium derivative means:

- Medicinal opium, i.e. opium that has undergone the processes necessary to adapt it for medicinal use.
- Prepared opium, i.e. any product of opium obtained by any series of operations designed to transform opium into an extract suitable for smoking and the dross or other residue remaining after opium is smoked.
- Phenanthrene alkaloids, namely, morphine, codeine, thebaine and their salts.
- Diacetylmorphine, i.e. the alkaloid also known as diamorphine or heroin and its salts.
- All preparations containing more than 0.2% of morphine or containing any diacetylmorphine.

Poppy straw means all parts (except the seeds) of the opium poppy after harvesting whether in their original form or cut, crushed or powdered and whether or not juice has been extracted therefrom.

Psychotropic substance 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. (This Schedule has been given at the end of the Act, mentioning proprietary, nonproprietary names and chemical composition of such substances.)

Offences and Penalties

Whoever in contravention of any provisions of this Act or any rule or order made or condition of a license granted thereunder produces, possesses, transports, imports inter-State, exports inter-State, sells, purchases, uses or omits to warehouse **poppy straw** or removes or does any act in respect of warehoused poppy straw shall be punishable as given in Table 31.2.

Table 31.2 Quantum of Punishment against Contravention of Provisions

Where the contravention involves small quantity	Rigorous imprisonment for a term that may extend to 6 months, or with fine that may extend to ₹ 10,000 or with both
Where the contravention involves quantity lesser than commercial quantity but greater than small quantity	Rigorous imprisonment for a term that may extend to 10 years, and with fine that may extend to ₹ 1 lakh
Where the contravention involves commercial quantity	Rigorous imprisonment for a term that shall not be less than 10 years but that may extend to 20 years, and shall also be liable to fine that shall not be less than ₹ 1 lakh but that may extend to ₹ 2 lakh

Similar provisions have been enshrined in the Act in relation to **opium derivatives, cannabis plant** and **cannabis,** etc. **Small quantity** in relation to narcotic drugs and psychotropic substances has been defined as any quantity lesser than the quantity specified by the Central Government by notification in the Official Gazette. Specific quantities amounting to small and commercial have been given in the Act itself. NDPS Act also provides for enhanced punishment for repeat offences, especially after previous conviction. Death penalty for certain offences after previous conviction has also been mentioned under Section 31A of the Act.

RELATED LEGAL PROVISIONS

Section 176 of IPC: Provides punishment for omission to give notice or information (including that of a poisoning case) to the public servant/police.

Section 177 of IPC: Provides punishment for furnishing false information (including that of a poisoning case).

Section 201 of IPC: Provides punishment for causing disappearance of evidence of offence (e.g., destroying sample of gastric lavage, clothing carrying evidence of poison etc.)

Section 202 of IPC: Provides punishment for intentional concealment of information of offense (including that of a poisoning case).

Section 269 of IPC: Negligent act likely to spread infection of disease dangerous to life.

Section 270 of IPC: Malignant act likely to spread infection of disease dangerous to life.

Section 271 of IPC: Disobedience to quarantine rule ('quarantine' means period of isolation imposed upon persons or animals that have arrived from elsewhere or been exposed to infectious or contagious disease).

Section 272 of IPC: Adulteration of food or drink intended for sale.

Section 273 of IPC: Sale of noxious food or drink.

Section 274 of IPC: Adulteration of drugs.

Section 275 of IPC: Sale of adulterated drugs.

Section 276 of IPC: Sale of drug as a different drug or preparation.

Section 277 of IPC: Fouling water of public spring or reservoir.

Section 278 of IPC: Making atmosphere noxious to health.

Section 284 of IPC: Prescribes for rash or negligent act (or omission) in relation with poisonous substance so as to endanger human life or to be likely to cause hurt or injury to any person.

Section 299 of IPC: Culpable homicide including that caused through administration of some poisonous substance.

Section 300 of IPC: Murder including that caused through administration of poisonous substance with the intention of causing death.

Section 304-A of IPC: Causing death by rash or negligent act (or omission) including that caused through poisoning.

Section 324 of IPC: Makes simple hurt more grave and liable to a more severe punishment—where it has been inflicted by one of the means described in the Section such as 'fire' or 'any heated substance', poison or any substance that is deleterious to the human body to inhale, swallow, or to receive into the blood.

Section 326 of IPC: It is similar to Section 324 with the only difference that the words 'grievous hurt' have been substituted for the word 'hurt' providing enhanced punishment.

Section 328 of IPC: Provides punishment for causing hurt by means of poison or any stupefying, intoxicating or unwholesome drug or other thing with the intent to commit an offence (i.e., *anything* when used in unwholesome state/composition can act as a poison).

Factors Modifying the Action of Poisons

The toxicity that a chemical causes depends upon its inherent properties (physico-chemical as well as pharmacological), the quantity or concentration involved, the route of absorption and the factors specific to the individual who is exposed. A discussion of the factors that can influence the toxicity of a given poison is presented below.

FACTORS ATTRIBUTABLE TO THE POISON ITSELF

Quantity

It is logical that a larger dose will produce more rapid and intense poisoning. However, if larger doses result in vomiting, these may not develop poisoning with the same intensity. Secondly, a resultant effect may be different with the larger dose as compared to the smaller one. Therefore, the cause of death with the larger dose may be shock, whereas with a smaller dose over a longer period, the cause of death may be different.

Physical Form

Gaseous or volatile poisons are very quickly absorbed and are thus rapidly effective. The rate of absorption and the effect is quicker when a poison is taken in liquid form than when taken in solid form. In case of solid poisons when the same is taken in the powdered form, the absorption and action is quicker than when taken as a lump. Some poisonous vegetable seeds may pass through the GIT as such when taken intact due to their impermeable pericarp. But when taken crushed, they may be rapidly fatal.

Chemical Form

Chemically, pure metallic arsenic and mercury are not poisonous because these are insoluble and are not absorbed. But white arsenic (arsenious oxide) and mercuric chloride are deadly poisonous.

Concentration/Dilution

Generally speaking, in concentrated form, poisons are absorbed more rapidly and are also fatal more rapidly. But this is not true with some poisons. A dilute solution of oxalic acid is less corrosive but is more rapidly absorbed and hence may be more rapidly fatal.

Route of Administration

The rapidity of the action of a poison depends upon the mode by which it is introduced into the system. Sublingual, inhalation and intravenous routes allow most rapid absorption of poisons. Poison is also quite rapidly absorbed through intramuscular, subcutaneous, rectal, urethral and vaginal routes. A poison ingested into the stomach acts more rapidly than when administered into the rectum, since the absorptive power of the stomach and the small intestine is greater than that of the large intestine and the rectum. Injured skin absorbs poisons quicker than intact skin.

Cumulative Action of the Poison

Preparations of cumulative poisons (poisons that are not readily excreted from the body and tend to pile-up in different organs of the body for a long period) may not cause any toxic effect when entering in low doses. However, when such poisons enter the body over a long period even in low doses, may ultimately cause harm when their concentration in different tissues reaches high level due to their cumulative effect.

Drug Interactions

Drug interactions are known to occur due to changing of one drug or increasing the response to another drug or food material. A patient on treatment with a monoamine oxidase inhibitor may suffer severe respiratory depression to ordinary doses of pethidine. Drugs like sedatives, hypnotics, tranquilisers, antihistamines can markedly increase the effect of alcohol. Evaluation of the hazards from exposure to multiple chemicals can be much more demanding than is the case for a single chemical (see Chapter 30 also).

FACTORS ATTRIBUTABLE TO VICTIM

Condition of the Stomach

- Presence of food stuff in the stomach acts as diluent of the poison and hence protects the stomach wall from the corrosive and irritating action of the poison. Dilution of poison also delays the absorption. Some specific type of food has specific action on absorption of the poison. Fatty food usually delays the process. However, presence of fatty food may accelerate the process of absorption in case of phosphorus.
- In case of pyloric stenosis and where gastrojejunostomy operation had been performed, poisons which are absorbed from the intestine are absorbed late due to emptying of stomach in case of former and due to repeated backward flow of the stomach contents from the jejunum to the stomach through the anastomosis.
- Absorption is usually rapid where the stomach is empty. Conversely, a substance may be destroyed in the gastrointestinal tract and may not manifest toxicity, e.g. d-tubocurarine or snake venom.

Condition of the Body

Physiological variables and pathological conditions may alter or modify the effects of poisons. Some important factors are as under:

- **Age, sex, body weight, etc.:** Some poisons are tolerated better in some ages and badly in some other ages. Opium and its alkaloids are tolerated better by elderly people but badly by children and infants. A well-built person with good physique and health will tolerate the action of a poison better than a weak subject.
- **Presence of any disease:** Broadly speaking, a healthy and vigorous person is less likely to succumb to the effects of poison than one who is enfeebled by disease. In certain disease conditions, some drugs are tolerated exceptionally well, e.g. sedatives and tranquilisers are tolerated in high doses by maniac and deliriant patients. Similarly, digitalis, tobacco or tartar emetic, even in a small dose, may produce symptoms of syncope when given to a person having a weak or fatty heart.
- **Habit, tolerance and drug-dependence, etc.:** By the long continued use of some drugs like opium, tobacco, alcohol, cocaine, etc., an individual can tolerate large doses that under ordinary circumstances may be liable to prove fatal. Repeated and continued use of some drugs mentioned above may result in drug dependence (see Chapter 30 also).
- **Idiosyncrasy (hypersensitivity/allergy):** Sometimes, a small or otherwise innocuous dose of a substance may result in severe toxicity. This phenomenon may be explained as an abnormal response of the body and is often an allergic response to the substance. Drugs such as penicillin, aspirin, sulfonamides, sera, etc. may exhibit an allergic response. Proteinous and nonproteinous eatables may also exhibit an allergic response (see Chapter 30 also).
- **Iatrogenic poisoning:** The term iatrogenic poisoning is used to refer to adverse reactions to drugs that are prescribed for a patient by a qualified doctor. The term comes from two Greek words, "iatros" meaning physician and "gene" meaning to produce. Iatrogenic poisoning may be an unavoidable hazard associated with the use of some treatment needed by the patient. In some cases, a patient has an idiosyncrasy or personal peculiarity in his biological make-up that makes him uniquely and unpredictable sensitive to a therapeutic drug. In most instances, iatrogenic

poisoning must be accepted as a normal and proper risk associated with the use of any medical procedure. In small percentage of iatrogenic poisonings, there is the element of carelessness, ignorance, or incompetence. In these cases, ethical, civil and even criminal considerations need to be faced.

- **Conditions surrounding the victim:** Activity, crowding, presence of other people and so on may all have an effect on the response to a poison. Certain poisons are influenced by the temperature or the humidity prevailing in the atmosphere. Atropine, for example, has a much greater adverse effect in a desert situation than in a cool, moist climate.

Concept of Fatal Dose

The clinical toxicologist is concerned with the diagnosis and treatment of the living patient, the analytical toxicologist has the complicated task of laboratory investigation and the autopsy surgeon is concerned with evaluating poison(s) as a cause or a contribution to death. He, therefore, has to collect suitable samples for analysis and, when the laboratory results are available, to interpret them in the light of the knowledge of the history, clinical record and postmortem findings.

Not only the lay public, but lawyers, police, etc., assume that there is a more or less linear relationship between the amount of poison that is administered, the resulting levels in blood and tissues and the degree of disability caused—the terminal disability being death. There is no 'fatal dose' in the sense of a single threshold concentration above which a person dies and below which he survives. Instead, there is a range of levels, the upper and lower margins of which may vary. Instances being recorded where survival occurs well above the upper limit and death occurs below the lower margin. In such cases, there is need to evaluate other nontoxicological data to see if they can modify the circumstances sufficiently to allow an acceptable explanation for the death.

Blood level values can be used as a guide but are not intended to be absolute. This is so because of the factors that can affect a drug or chemical in such a way that it may not reach the same level in every human being. The blood level of a drug or chemical can and does vary even among individuals of about equal weight and receiving similar dosages. The data should be used and interpreted for each case. It may be pointed out that the analytical procedure used may be of such a nature that the concentrations of the chemical and its metabolite(s) are detected and reported as the concentration of the unchanged drug. This may account for differences in the levels reported among similar studies of the same drug. Therefore, the additional tissues/samples like urine, vomitus or lavage need to be analysed. Winek and coworkers reported a case of quinine poisoning resulting in death in which 12 different tissue levels were reported. Methods for isolating, identifying and quantitating toxic materials/substances from the tissues often differ in their degree of specificity, sensitivity and accuracy.

When the amount of poison found is sufficient to cause death, it can then conveniently be stated that the poison was the cause of death. However, there may be circumstances when lethal level of a drug or chemical found in an individual's blood does not by itself establish the cause of death. For example, where a known narcotic addict was 'shot to death', and the analysis of various body tissues revealed levels of morphine that were consistent with levels in other deaths attributed to over-dosage with heroin or morphine. **Failure to find a lethal** dose does not exclude the possibility of the poison being responsible for the death. Frequently, a large proportion of the poison swallowed may be got rid of by evacuation. In this way, the whole alimentary tract may be freed of the poison and only the portion that has been absorbed remains in the body. This absorbed portion is distributed more or less throughout the body. However, only some part of the body can be examined and therefore, the quantity of poison found is only a part of the quantity the body contains, which may not be amounting to a minimum fatal dose. Further, the drug/chemical-protein complexes formed in the plasma and other tissues may be difficult to breakdown without some loss of the drug/chemical. Thus, the values reported are indicative of the amount of a compound recovered and do not necessarily reflect the amount actually present in the tissues.

Evidence of Poisoning in the Living

The recognition of poisoning during life is a matter of importance, both from point of view of physician treating the sufferer, and from that of medical jurist whose duty is to help in unmasking the poisoner. Poisoning is acute when caused by one large dose or several smaller doses repeated at short intervals, or chronic when due to continued absorption of minute quantities of poison. Chronic poisoning is nearly always accidental or industrial and concerns the physician and the public health authorities more than the medical jurist.

There is no single symptom, and no definite group of symptoms, which are absolutely characteristic of poisoning. The symptoms of a disease may simulate acute poisoning. However, if a particular train of symptoms, which is usually caused by a certain substance, is associated with a history of typical poisoning, the suspicion of poisoning must be substantiated. There are three factors that must be considered in approaching a diagnosis of poisoning in the living:

- History.
- Clinical signs and symptoms observed by the attending physician.
- Discovery of poison in the food/vomit/urine/excreta (hence, in suspicious cases, these articles must be preserved in clean, glass-stoppered bottles for chemical analysis).

Evidence of Poisoning in the Dead

In fatal cases, evidence of poisoning needs to be approached on the following lines:

- History (information from the inquest report and from the relatives of the deceased).
- Clinical record (in case of hospitalised victims).
- Postmortem findings.
- Discovery of poison in the viscera and blood by chemical analysis.

Technique of Obtaining Autopsy Samples

Investigation of death from suspected poisoning may depend upon the correctness or otherwise of sampling of fluids and tissues from the body. Specimens of sequestered haematomas like subdural, intracerebral, or any other localised collection of blood that is sequestered from the general circulation tends to retain drugs and alcohol longer. Such blood may, therefore, give a better indication of any drug or alcohol level present when haematoma was formed at the time of the happening (this may be particularly so in obtaining drug or alcohol levels in individuals who have survived for some days after the happening). Usually, the concentration of many analytes increases in both heart and peripheral blood specimens during the postmortem interval. In most cases, femoral blood is more likely to be a better indicator of the perimortem concentration of the analyte.

Further, peripherally located blood (from femoral veins) is preferable because it is relatively isolated from the internal organs in the chest and abdomen (blood obtained from heart or other central regions in the chest and abdomen may have falsely elevated drug levels due to postmortem drug diffusion down concentration gradients—a process referred to as **postmortem redistribution of drugs**). Factors responsible for such a phenomenon may be:

- passive diffusion from the stomach into the nearby organs, including the left lobe of the liver, lower lobe of the left lung, and the heart (that is why it is advisable to collect sample of the liver from the middle of the right lobe, which is relatively sequestered region of the liver);
- separation of drug from its protein-bound sites after death and diffusion into the adjoining tissues;
- tissue autolysis;
- depletion of energy-dependent binding processes;
- postmortem movement of blood as brought about by:
  - gravity (i.e., liquids and blood cells accumulating in the most dependent parts of the body),
  - leakage of blood into soft tissues, and
  - muscle rigidity that may expel blood trapped in the heart and large vessels as rigor mortis develops and subsequently, the pressure resulting from the gases accompanying the putrefactive process also plays its role in variable blood movement, etc.

A sample of **urine** can be obtained by puncturing the fundus with syringe and needle. Alternatively, the bladder can be stretched by pulling upwards with the fingers, then a sagittal incision is given with a knife on the ventral surface. The outcoming urine can be collected directly into the container. If only a small amount is present, then the incision may have to be enlarged and the residual urine syringed out under direct vision. Chemicals/drugs can usually be easily detected in this specimen. Protein-binding factors are not in effect; making extraction of drugs/chemicals simpler than in other body specimens. However, the disadvantages may include the following:

- Some drugs (e.g., morphine) must be hydrolysed for complete recovery from the urine.
- The concentration of agents found in the urine is usually of no significance in evaluating the quantity ingested or the toxicity, since urinary concentrating factors sometimes lead to exhibit large quantities even after therapeutic administration.
- Some drugs are present only as metabolites in the urine, owing to extensive body metabolism and low water solubility (cocaine is excreted unchanged only to the extent of 1–10%, depending upon pH, and the remainder being excreted as the metabolite).

Bile needs to be collected in cases as those for morphine and chlorpromazine, which are concentrated by the liver and excreted into the gallbladder. Direct collection into a bottle is preferred, as bile is usually too viscous to be syringed out. It should be noted, however, that the concentration in bile is of no value in interpreting toxicity, since it represents metabolised drug/chemical that may have been taken up to several days prior to death.

Brain tissue does not accumulate most drugs/chemicals, and the concentrations are usually found to be less than those present in the blood. Exceptions are certain lipophilic agents such as benzodiazepines, benzene and its derivatives and cocaine, which accumulate in the brain to a concentration slightly higher than that found in the blood. The concentrations found in the blood, however, may be used more reliably to assess pharmacologic activity. Cerebrospinal fluid may be required for toxicological analysis, though it may be needed for microbiological and virological studies too. It should be collected by lumbar or cisternal puncture.

Vitreous humour is sometimes useful, especially in bodies with appreciable postmortem decomposition as this fluid resists putrefaction longer than other body liquids. A puncture should be made through the sclera at the outer canthus. This should be placed as far laterally as possible, pulling the lid out. The fluid should be sucked out by syringe slowly.

Other fluids may sometimes be useful to collect. Pericardial fluid, pleural effusion and ascitic fluid can be used for qualitative analysis to identify a range of substances.

In solvent abuse and deaths from gaseous or volatile substances, the toxic material may be isolated from the whole lung. On opening the thorax at autopsy, a lung is mobilised and the main bronchus is tied off tightly. The hilum is then divided, and the lung placed immediately into a metallic container, which is sealed and sent as soon as possible to the laboratory.

The substances that may have been injected are numerous. However, insulin, morphine, heroin, cocaine and other illicit drugs are the common examples. In the notorious, Coppolino case in the United States, products of succinyl choline were identified from around a needle track in the buttock of an exhumed body of a woman leading to the conviction of her anaesthetist husband. It needs to be remembered that when an injection site is sampled, it is essential that a control area from a remote part of the body is also sent to the laboratory. Taking sample from the contralateral side needs caution because, as in both drug dependence and insulin usage, alternate sides may be used at frequent intervals for injection.

- In case of **drug abuse during pregnancy**, meconium and hair can be valuable specimens for drug testing. Meconium (it is the bowel contents of the foetus or the first few stool specimens of the neonate) may serve as a "depot" of drugs ingested by the mother from about the 14th to 16th week of gestation onwards (since meconium only begins to form from about that time and is not normally eliminated until after the delivery). Even if mother shows negative results for toxicology at delivery, the meconium may reflect drug abuse during the pregnancy. Hair usually begins to form during the third trimester of gestation. It remains a valid toxicologic sample for up to a few months after birth.
- **In decomposed bodies,** blood is frequently degenerated into a serosanguineous fluid. Reportedly, the skeletal muscle carries drug levels approximating the antemortem blood drug levels (the sample needs be taken from an extremity to minimise the possibility of altered drug levels due to postmortem redistribution). In badly decomposed bodies, maggots may be collected for toxicology testing.
- **In embalmed bodies,** muscle from the buttock serves as an excellent sample. Explanation may reside in the fact that buttocks remain compressed (as the body is in supine position during the embalming procedure), which helps limiting the amount of perfusion to the posterior areas, preventing formation of artefacts by the embalming fluid to an appreciable extent. **In exhumed bodies**, the only blood available may be caked blood from the heart chambers, aorta, or common iliac arteries. Heart, liver, brain, lung, kidney, hair, nails, and various other tissues may be collected depending upon the circumstances of the case.

Rarely, **Radiology** may come to the help as in the case of George Markov murder in London, where a political assassination was carried out by the introduction of a tiny sphere, presumably injected from an air weapon concealed in an umbrella. The sphere drilled out to carry a potent toxin and was located by X-ray at autopsy after small skin puncture was noticed.

Interpretation of Results

An approach to the problem of interpretation usually begins with the review of data abstracted from the literature or culled from the previous cases investigated by the toxicologists and others. Sometimes, it becomes obvious that a quantitative analysis is not necessary. Extensive damage done to the tissues by strong acid or alkali does not require a quantitation of the acid or alkali but rather, a qualitative identification. The same may hold true in case of asphyxiant gases. However, where therapeutic agents or other chemicals are involved, a quantitation of the concentration present in the various tissues can serve to elucidate the cause and manner of death.

In a living person, the concentration of a poison is lower in the venous blood as compared to arterial because tissues may take up the compound from the arterial supply, the concentration then being lower in the venous return. Similarly, portal blood may have a substantially higher concentration of a substance that is being absorbed from the intestine before it is extracted by passage through the liver. After death, variation in concentration is caused by uneven destruction by enzymatic and microbiological activity and from diffusion from sites of higher concentration as the barriers formed by living cell membranes breakdown after death and small molecules may move easily through the tissues into vascular channels.

The oral route is the common route for administration of a drug/chemical therapeutically as well as for other purposes, i.e. suicidally or accidentally. The gastric contents, lavage or vomitus may reveal the presence of solid or dissolved dosage forms, or the colour/odour or both may suggest a liquid dosage form. The total amount of drug found is helpful in establishing whether the death was accidental or suicidal.

The finding of a drug/chemical in the stomach does not necessarily indicate ingestion of that substance. Certain drugs may enter the body via another route and be secreted into the stomach (enteric circulation). The amount found in the stomach may suggest the possibility, i.e., a low concentration may represent the increment from the enteric circulation rather than the remainder from ingestion. Basic drugs (narcotics, cocaine, amphetamines) can reach high concentrations in the stomach even when given parenterally. (It is important to point out that with most analytes, the presence of drug in the gastric contents is not sufficient proof that it was the agent or one of a combination of toxicants that caused the death. It must be documented that sufficient absorption of the substance occurred to result in a toxic concentration of the analyte in blood and/or liver. The liver concentration and/or brain

concentration of an analyte is extremely important in the determination of the involvement of an analyte in the cause of death.)

Drugs taken in combination can be more toxic than if considered separately. Knowledge of the interaction of toxicants will be paramount to proper interpretation of the toxicity of any analyte. Most analytes are more toxic in the presence of alcohol. Unfortunately, information about most combinations of analytes is not well-known and the toxicologist must quite often deal with them on an individual basis. Experience with similar cases or published reports may come to the help of the toxicologist.

The conclusion that death was caused by poison depends on evaluation of history, clinical, toxicological and anatomical findings. It should be remembered that even in case in which the suspicion of poisoning is strong, death may have been due to causes other than the administration of poison. Postmortem appearances indicative of disease or injury may be found coexisting with appearances indicating death from poison. In such like cases, the fact of existence of the disease or injury may be important as bearing on the question of suicide or homicide. **It has been wisely held by Christison** that in cases where a poison has not been detected on chemical analysis, the court, in deciding a charge of poisoning, should weigh in evidence the symptoms, the postmortem appearances and the circumstantial evidence.

Failure to find/detect any poison in the viscera/other specimen of the individual whose death was allegedly due to poisoning may be due to the following causes:

- The decedent may have been unusually susceptible to the deleterious effects of the chemical(s) in question.
- The whole of the poison has been removed from the stomach and intestines by vomiting and purging, and after absorption has been detoxified, conjugated and eliminated by the kidneys and other channels or the quantity present is below the detection limits.
- The absorbed poison might not have been evenly distributed in various organs, which varies with different poisons and also with its mode of administration, e.g. blood levels are higher than liver levels when the poison is administered by intravenous route.
- The detection of highly potent toxic substance with low lethal dose is quite difficult.
- Interference of proteins, fats and decomposition products sometimes makes the detection difficult or rather impossible.
- Sometimes, the treatment given to the victim may alter the poisonous substance and make its detection difficult or even impossible.
- The isolated material may be different than the suspected to be consumed because certain compounds are altered in the body by biochemical process, e.g. detection of phenobarbitone when primidone has been taken and the finding of oxazepam when diazepam has been taken.
- Certain vegetable poisons may not be detected in the viscera as they have no reliable tests, while some organic poisons, especially the alkaloids and glucosides, may, by oxidation during life or by putrefaction after death, be split up into other substances having no characteristic reactions sufficient to reveal identification.
- It is possible that whole of the poison disappeared from the lungs by evaporation. This is especially likely to occur in case of very volatile, for example, gaseous poisons.
- Neglect to submit certain matters or a sufficient quantity thereof, for analysis.
- Lack of reliable means of extracting the poison from substances containing it or no satisfactory tests available for its identification.
- Biological toxins and venoms that may be protein in nature cannot be separated from body tissues. Immunoassay procedures can detect these compounds.
- Normal metabolic degradation of the chemical can reduce its blood concentration during a prolonged survival interval in which respiratory complications and hypoxic encephalopathy maintain coma and act as the immediate causes of death and the chemical may have been eliminated completely by that time.
- When a substance that is normally not present in the body is isolated, it indicates exposure to the substance. But when a substance that is normally present in the body is isolated, it becomes necessary to consider a range of values before interpreting the result as exposure to the toxic levels of this substance, i.e. quantitative analysis is required in such cases.
- Want of care or skill on the part of the analyst.

Relative Toxicity of Drugs/Chemicals

Therapeutic index or the ratio of the toxic to the effective dose of a drug indicates the relative toxicity of drugs. Toxicity of the chemicals has been devised depending on the amounts that produce harm (Table 31.3).

Table 31.3 Toxicity Rating of Drugs/Chemicals Depending upon Usual Fatal Dose

Usual fatal dose	Rating*
< 5 mg/kg	6—Super toxic
5–50 mg/kg	5—Extremely toxic
51–500 mg/kg	4—Very toxic
501 mg/kg–5 g/kg	3—Moderately toxic
5.1–15 gm/kg	2—Slightly toxic
> 15 gm/kg	1—Practically non-toxic

*The rating is based upon mortality, and is applicable to the acute toxicity of a single dose taken orally, helping the doctor to have some idea as to the hazardous nature of various poisonous substances.

32 CHAPTER

Duties of a Doctor in Cases of Suspected Poisoning

After going through this chapter, the reader will be able to describe: Circumstances needing reporting to the police | Steps involving management of poisoning | Various types of antidotes | Principles of chelation therapy and various chelating agents

In all cases of poisoning, the doctor must record the preliminary particulars, viz., full name with address, age, sex, occupation, date and time, brought by whom, history, etc. When poisoning is suspected, the doctor's first duty is to guard his patient's interest. He should at once treat him after finding out the nature of the poison so that appropriate and timely treatment is instituted. If the nature of the poison is not known, treatment is instituted on general lines.

If a medical practitioner in private practice is convinced that the patient he is attending to is suffering from homicidal poisoning, he is bound under Section 39 CrPC to communicate the fact to the nearest police officer or magistrate. Noncompliance is punishable under Section 176 IPC. He is not liable for giving notice, if the case has already been reported to the police by the village headman/village watchman or any other officer required under the law to give such information under Section 40 CrPC. Under Section 39 CrPC, some specific Sections of IPC have been mentioned where the public is bound to give information to the police (Section 309 IPC, which refers to the offence of an attempt to commit suicide or Sections relating to accidents have not been included in Section 39 of CrPC, which requires the public to give information of certain offences). However, under Section 175 of CrPC, one is bound to supply such information if he is summoned by the investigating police officer or magistrate. Noncompliance or concealing any such information is punishable under Section 202 IPC. Giving false information on such matters is punishable under Section 177 IPC.

It may be worth mentioning here that Section 43 of IPC stretches the ambit of the expression 'legally bound to do' to a considerable extent. Three categories have been mentioned, viz., (*i*) everything that is an offence, (*ii*) everything that is prohibited by law and (*iii*) everything that furnishes grounds for civil action.

Furthermore, in the present scenario, where the public is better oriented towards legal implications, there is likely to occur an element of bias in the history given by the relatives/attendants due to insurance/pensionary benefits. The circumstances surrounding vehicular/industrial accidents and suicides through hanging/some other means are quite different and apparent from those surrounding the accidents/suicides through poisoning. Mostly, the doctor will not be in a sound position to label the case for one or the other category. It is, therefore, **advisable to report each and every case of suspected poisoning to the police** and the question of suicide/accident/homicide to be considered by the police. This will be in the interest of the doctor and will avoid complications arising thereof at some later stage, i.e. omission to inform or furnishing false information. In accidental poisoning, if there is any indication of danger to the general public, for example, food poisoning from some hotel or contamination of public drinking water, doctor must notify the public health authorities immediately.

In a case of suspected homicidal poisoning, it is advisable to consult another practitioner, preferably a senior one, who may have requisite knowledge and experience. A doctor should take every precaution to prevent the possibility of further administration of poison to the patient. The best way to do this is to remove him to some hospital or nursing home, where the doctor in charge should be informed of the suspicion so that the victim is not allowed to eat anything brought by friends/attendants and no one can obtain access to his medicines except the nursing staff. If, for some reason, he cannot be removed to the hospital, two well-trained nurses may be employed with instructions that nothing should be given to him by anyone except by either of the nurses. If this is also not possible, a near relation or a friend may be taken into confidence and be informed of the suspicion. If the patient happens to be an adult and in full possession of his senses, he may also be warned.

In every case of suspected poisoning, the doctor must keep and maintain the detailed records of the case. He must collect and preserve all the evidence such as the vomited matter or stomach wash and samples of urine and faeces passed in his presence and suspected articles of food, drink or medicine in separate wide mouthed glass bottles or jars with tightly fitting glass stoppers. These bottles/jars should be labelled properly with the name of the patient, the material preserved with date and time of collection, and should be kept under lock and key in his own custody till required for transmission to the chemical examiner. A medical practitioner must also preserve any other evidence of suspected poisoning like a bottle, cup or tumbler in which the poison is suspected to have been mixed or a piece of paper used for dispensing and wrapping the poison. Clothes or bed sheet soiled by vomit, urine or faeces should also be preserved for possible future examination.

Recording of dying declaration is necessitated when the patient is serious. It is preferable to call a magistrate for this purpose, but if death appears imminent, or if there is likelihood of delay in the arrival of the magistrate, the attending doctor is duty-bound to record the statement himself/herself. Even when the statement is recorded by the magistrate, the presence of a doctor is advocated for certifying as to the 'fitness' of the victim to give statement. If the victim dies before the exact diagnosis could be made out, or was brought dead to the hospital, the doctor must report it to the police. And, the doctor conducting the autopsy in such a case must exercise care in collecting viscera and fluid(s) for sending to the Chemical Examiner for toxicological analysis. **Not exercising proper judgement and care in selecting, preserving and dispatching of specimens for toxicological evaluation would lead to grey areas in interpreting ultimate results/findings** (see Ancillary Investigations in the Chapter "Medicolegal Autopsy, Exhumation, Obscure Autopsy, Anaphylactic Deaths and Artefacts").

General Management of Poisoning

Unlike the average clinical case, many overdosed patients are brought to dispensary/hospital in an unconscious/semiconscious condition. Even if a poisoned patient is conscious and alert, he/she is usually uncooperative and even hostile, since the majority of hospital admissions are cases of attempted suicide. Added to these problems is the unfortunate absence of specific signs and symptoms regarding many poisonous substances. Time need not be wasted in fruitless pursuit of the exact identity of the causative agent. The adage 'treat the patient and not the poison' is quite relevant in toxicological cases. Many poisoned patients will recover with simple supportive treatment. A minority may require intensive care. It is always advisable to send samples of blood, urine, vomitus, etc. to the nearest Forensic Science Laboratory for analysis. Sodium chloride is sufficient as a preservative, though sodium fluoride is to be preferred for the blood sample. The main objects include (*i*) removal of unabsorbed poison, (*ii*) hastening elimination of absorbed poison, (*iii*) administration of antidotes and (*iv*) treatment of general symptoms.

BASIC PRINCIPLES OF POISONING MANAGEMENT

- **Supportive Care**
  - Airway protection
  - Oxygenation/ventilation
  - Haemodynamic support
  - Treatment of seizures
  - Correction of temperature abnormalities
  - Correction of metabolic derangements
  - Prevention of secondary complications
- **Removal of Unabsorbed Poison**
  - Gastrointestinal decontamination
    - Induced emesis
    - Gastric lavage
    - Activated charcoal
    - Whole bowel irrigation
    - Catharsis
    - Endoscopic/surgical removal
  - Decontamination of other sites
    - Skin decontamination
    - Eye decontamination
    - Respiratory decontamination
- **Hastening Elimination of Absorbed Poison**
  - Multiple-dose activated charcoal
  - Forced diuresis
  - Alteration of urinary pH
  - Extracorporeal removal
    - Peritoneal dialysis
    - Haemodialysis
    - Haemoperfusion
    - Plasmapheresis
    - Exchange transfusion
    - Hyperbaric oxygenation
- **Administration of Antidotes**
  - Using mechanical (physical) antidotes
  - Neutralisation by chemical binding
  - Physiological antidotes
  - Metabolic antagonism
  - Chelation therapy
  - Immunotherapy

Some of these are explained as follows.

Removal of Unabsorbed Poison

Depending upon the route of entry, the following principles should be applied.

Inhaled Poisons

When a poison has been inhaled, such as carbon monoxide, automobile exhaust, gas from a septic tank, etc., the patient should immediately be removed to fresh air to chase out the poisonous gas through the lungs. A clear airway should be ensured.

Injected Poisons

If the poison has been injected, application of tourniquets, proximal to the point of injection may slow absorption. Some unabsorbed poison may have to be removed through multiple incisions and suction, similar to that commonly advised for snake bite.

Contact Poisons

If poison be spilled or sprayed on skin, eye or wound or be inserted into vagina, rectum or urinary bladder, the appropriate way is to wash it out with plain warm water. If a specific antidote is known, the poison can be neutralised.

Ingested Poisons

The object is to remove the poison from the gastrointestinal tract as soon and as much of the poison as possible. Attempt should be made to minimise its absorption by gastrointestinal decontamination. Whether or not to perform gastrointestinal decontamination and which procedure to use depends on the time since ingestion, the existing and predicted toxicity of the ingestant; the availability, efficacy and contraindications of the procedure; and the nature, severity and risk of complications. The various modes of gastrointestinal decontamination may include the following:

Administration of Milk or Water A toxin can be diluted by administering milk or water (only to conscious patients) within a few minutes of ingestion. Milk may be given to all patients except those ingesting phosphorous and strong acids. It acts by diluting the poison and by demulsifying toxins. Dilution is effective in ingestion of alkalis and weak acids.

Induction of Emesis Emesis can be induced at home by mechanical stimulation of the posterior pharyngeal wall. However, this is not an effective way of removing poison. A chemical induction of emesis can be achieved by the use of syrup of ipecac. **Ipecac** is obtained from the dried roots of the *Cephaeli ipecacuanha* plant and contains various alkaloids of which emetine and cephaeline predominate. Ipecac stimulates emesis primarily by directly irritating the gastrointestinal mucosa, although a central neurogenic mechanism also contributes. The dose of syrup of ipecac is 15 ml for children and 30 ml for adults. If the vomiting does not occur, dose may be repeated after half an hour. Induction of emesis is helpful if it is done within 1 hour of ingestion. **Contraindications** for the induction of emesis include acid or alkali ingestion, petroleum distillate ingestion, patients with convulsions or those who may develop convulsions and patients with altered sensorium.

Activated Charcoal It has comparable or greater efficacy, and is less aversive and invasive than ipecac or gastric lavage. It is a preferred method of gastrointestinal decontamination in many situations. Activated charcoal is prepared as a suspension in water, either alone or with a cathartic. Because of its large surface area, it adsorbs several poisons and, therefore, reduces their absorption. The optimal dose of activated charcoal is ten times the amount of poison ingested. However, since the latter is not known in most cases, it is given in a dose of l–2 gm/kg body weight using 8 ml of diluent per gram of charcoal. Palatability may be increased by adding a sweetener (sorbitol) or a flavouring agent. It is advisable to administer one dose of charcoal before performing a gastric lavage. This halts the further absorption of poison and allows the removal of charcoal-toxin complex to be evacuated with stool. The complex can also be removed from the stomach by induced emesis or lavage. Charged (ionised) chemicals such as mineral acids, alkalis and highly dissociated salts of cyanide, fluoride, iron, lithium and other inorganic compounds are not adsorbed well by charcoal.

Gastric Lavage (Stomach Wash) Gastric lavage is the preferred method of emptying the stomach and should follow the instillation of one dose of activated charcoal. It is effective if performed within about 4 hours of ingestion. However, in cases of ingestion of phenothiazines, antihistamines, tricyclic antidepressants or salicylates, a good amount of poison can still be recovered after several hours of ingestion because all these drugs delay gastric emptying.

Procedure: Gastric lavage may have dangerous sequelae if performed clumsily. The patient should be prone or semiprone on his left side with head hanging over the edge of the bed and face down, supported by an attendant/assistant so that the mouth is at a lower level than larynx (Trendelenburg and left lateral decubitus position) to prevent aspiration. The dentures, if any, must be removed. The airway must be clear and a mouth gag with a central hole is necessary especially in unconscious patients to prevent the rubber tube being bitten by the teeth. **In adults**, gastric lavage may be done by **stomach tube**. It is a flexible rubber tube about 12.7 mm in external diameter and about a metre and a half in length. It is sufficiently stiff to pass without kinking. A funnel is provided at the upper end and a suction bulb to suck out fluids is also provided. The lower end is blunt and rounded to avoid any injury when it is being passed and is perforated by more than one opening on its sides to allow the administered fluid to enter the stomach easily. The distance between teeth and the cardiac end of the stomach is about 45 cm in adults. Therefore, at a point about 50 cm from the lower end, the tube is marked so that the operator may have some indication when the lower end of the tube has reached the stomach.

The tube should be lubricated with liquid paraffin/glycerine and passed through the hole in the middle of mouth gag, over

the tongue and down the oesophagus. At about the mark, the tip of the tube should be lying in the stomach and one must make sure of this by dipping the funnel end in water. If the tip is in the air passage, bubbles of air will be found coming out of the funnel end in the water. After testing, about half a litre of plain water is run into the funnel, which is held above the level of patient's mouth. The fluid enters the stomach by gravity. The funnel is then lowered below the level of patient's stomach over a receptacle to allow gastric contents to siphon-off. The first washing should be preserved for chemical analysis. A small quantity of a saturated solution of a common salt can be added to the sample as a preservative. The process is then repeated with warm water or other fluid preferably containing an appropriate antidote until the returning fluid is of the same colour as the lavage fluid. Some of the antidote or other suitable solution may be left in the stomach to deal with the effect and after effects of whatever small quantities may have escaped lavage or are later excreted in the stomach. Before the stomach tube is withdrawn, it should be pinched to prevent aspiration of material into the lungs. **In children,** a tube of narrower calibre and shorter length is used. A Ryle's tube may serve the purpose. About 25 cm length is necessary to reach the stomach and this distance should be marked. When the tube has reached the stomach, 20 or 50 ml glass syringe is attached to the upper end of the tube and the stomach contents aspirated. Tap water containing antidote (if available) is then introduced. The stomach should be washed repeatedly to increase the total quantity of poison removed. Before the tube is withdrawn, it should be pinched to prevent aspiration of material into the lungs. During the lavage, the child should be lying prone, the foot-end of the bed elevated and the head slightly extended, turned to one side and supported by some assistant. **Contraindications** to gastric lavage include corrosives (except carbolic acid), convulsants, foreign body ingestion, petroleum distillates, oesophageal varices, etc.

Whole Bowel Irrigation It is performed by administering a bowel cleansing solution containing electrolytes and polyethylene glycol orally or by gastric tube at a rate of up to 0.5 L/h in children and 2.0 L/h in adults until rectal effluent is clear. The patient must be in sitting position. It may be appropriate for those who have ingested foreign bodies, packets of illicit drugs, slow-release or enteric-coated medications and agents that are poorly adsorbed by charcoal, e.g. heavy metals. It is contraindicated in patients with bowel obstruction.

Cathartic Salts The most effective cathartic is sorbitol in a dose of 1–2 gm/kg body weight. Alone, cathartics do not prevent absorption of the ingestant and should not be used as a method of gut decontamination. Their primary use is to prevent constipation following charcoal administration.

Endoscopic or Surgical Removal of Poisons It may be useful in rare situations, such as ingestion of a potentially toxic foreign body that fails to transit the gastrointestinal tract, a potentially lethal amount of heavy metal or agent that have coalesced into gastric concretions (barbiturates, glutethimide, heavy metals, lithium, meprobamate, sustained-release preparations).

Hastening Elimination of Absorbed Poison

Once a poison has been absorbed, its systemic effects can be reduced by accelerating its removal from the body. Various techniques employed to enhance the elimination of poisons from the body are discussed below:

Interruption of Enterohepatic Circulation

A few poisons are secreted into the bile and are reabsorbed in the gut. This is known as enterohepatic circulation and may be seen in phenobarbital, carbamazepine, glutethimide and some organochlorine poisonings. Cholestyramine reduces this reabsorption by binding with the poison in the gut thereby reducing the plasma concentration of the poison.

Enhancing Urinary Excretion

The technique for enhancing urinary excretion of a poison is known as **forced diuresis**. The principle is that by diluting the urine, the concentration gradient between the blood and the tubular fluid is reduced and, therefore, less tubular toxin is reabsorbed. For forced diuresis, normal saline or Ringer lactate and diuretics may be instituted to maintain an hourly urine output of 4–5 ml/kg body weight.

Since the renal tubular epithelium is more permeable to unionised solutes, an alteration of the urinary pH may help in increasing excretion by reducing the unionised solute in the tubular fluid. This is the basis for acidic or alkaline diuresis. **An alkaline diuresis** is effective in enhancing the elimination of several drugs including phenobarbital, salicylates and lithium. To affect an alkaline diuresis, 5% dextrose in half normal saline containing 20–40 meq of bicarbonate may be instituted. During this therapy, acid-base, fluid, and electrolyte parameters need to be taken care of.

Dialysis

A few toxins can be removed effectively by dialysis. These include phenobarbital, salicylates, theophylline, methanol, quinine and lithium. **Haemodialysis,** if available, is five to ten times more effective than peritoneal dialysis. **Haemoperfusion** is another technique in which blood passes through an external filtering device that contains charcoal or a synthetic resin that adsorbs poison. It is beneficial in cases of barbiturate, salicylate, theophylline, paraquat and meprobamate poisoning.

Administration of Antidotes

An antidote is defined in Webster's New Collegiate Dictionary as a remedy to counteract the effects of a poison. Remedies,

in this sense, are usually visualised to be specific chemical entities but this definition may be broadened to include nonspecific measures such as charcoal haemoperfusion, dialysis and so on.

Specific therapy of a case of poisoning involves the use of antidotes that counteract the pathophysiology produced by a toxin. According to their modes of action, they may be enumerated as follows.

Physical or Mechanical Antidote

They prevent the action of the poison mechanically, without destroying or inactivating the damaging actions of the poisons. Examples may include: **demulcents** like egg albumin, starch or barley water or even milk. They have a soothing action and form a protective layer on the mucous membrane of the stomach to protect it from the action of poison. **Adsorbents** like activated animal charcoal that has the capacity to adsorb poisons (e.g. alkaloids) in the pores so that the poison cannot come in contact with the wall of the stomach and is thus prevented from being absorbed. **Diluents** like water or milk or similar drinks that dilute the poison and in that way delay absorption. **Bulky food** like boiled rice or boiled vegetables act by getting admixed with the poison and thereby allow smaller amount of poison to be available to the stomach mucosa for absorption.

Chemical Antidote

These are substances that disintegrate and inactivate poisons by undergoing chemical reaction with them. Examples may include the following:

Weak Noncarbonate Alkalies (for Acids) In case of poisoning with corrosive acids, alkalies act as neutralisers. However, strong alkalies should not be used as they can damage the stomach further. Carbonate alkalies are also avoided because these on reacting with acids produce CO_2, which inflates the stomach and thus stomach may get ruptured if inflated much with gas.

Weak Vegetable Acids (for Alkalies) In case of poisoning with strong corrosive alkalies, weak vegetable acids like citric acid, acetic acid (vinegar), etc. may be used. **Freshly prepared ferric oxide (for arsenic poisoning)** may be used. With arsenic, it forms ferric arsenate, which is not absorbed.

Potassium Permanganate It is an important chemical antidote owing to its oxidising properties. A dilute solution of potassium permanganate 1:10,000 is commonly used in strychnine, nicotine, aluminium phosphide and opium poisoning for gastric lavage. It may also be used in poisoning by oxidisable substances such as phosphorus, hydrocyanic acid, cyanides, morphine, atropine and other alkaloids. **Tincture iodine** (15 drops to a half of glass of warm water) may be used to wash out the stomach, as it precipitates most alkaloids.

Physiological or Pharmacological Antidote

These are substances that exercise their own action on different systems of the body, producing signs and symptoms opposite to the signs and symptoms (or actions) produced by the poison(s). Examples may include naloxone for morphine, neostigmine for *dhatura* and barbiturate for strychnine, etc.

- **Reducing metabolism to toxic agents:** Some antidotes inhibit the metabolism of a nontoxic compound into active toxic compounds. For example, methyl alcohol produces its toxicity after its conversion by alcohol dehydrogenase into formaldehyde and formic acid. Alcohol dehydrogenase has more affinity for ethyl alcohol than methyl alcohol. Therefore, use of ethyl alcohol reduces the toxicity of methyl alcohol.
- **Enhancing metabolism to a nontoxic agent:** Some antidotes may increase the metabolism of a toxin so that it does not accumulate in the body. N-acetylcysteine provides sulphydryl groups that are rate limiting for glutathione-based liver enzymes, which is important in the detoxification of toxic intermediates of paracetamol.
- **Enhancing concentration of naturally present agent:** An antidote may increase the concentration of a natural substance, which is the target for the poison; for example, pralidoxime increases the concentration of cholinesterase and therefore can counter the manifestations of organophosphate poisoning.
- **Competition at the receptor site:** Several antidotes displace the toxin from the receptor site at which it acts for its toxic manifestations. For example, organophosphorus insecticides produce toxic effects because of accumulation of acetylcholine at the synapse and excessively stimulating the acetylcholine receptors. Atropine, which is a blocker of the muscarinic receptors of acetylcholine, finds beneficial effect in organophosphorous insecticide poisoning.

Universal Antidote

Universal antidote is actually a combination of physical and chemical antidotes. When the exact nature of the poison consumed is not known, then the universal antidote may be used. Constituents include the following:

Activated charcoal	2 parts
Magnesium oxide	1 part
Tannic acid	1 part

(According to toxicology experts, the use of activated charcoal alone would be much more effective, and some evidence suggests that the addition of milk of magnesia and tea actually interferes with the absorptive activity of charcoal.)

Chelation Therapy

The word 'chelation' comes from the Greek for a 'claw'. The term is often used to describe those agents that exercise their

action on complex of certain metals. They act on the absorbed metallic poisons. They form chelate with the metallic poisons that are freely available in circulation and help their early excretion from the body. The important amongst them are described below:

Dimercaprol (2,3-dimercaptopropanol) It is commonly known as British Anti-Lewisite or **BAL** (It was developed by the Britishers during the Second World War and had been shown to be effective against the effects of 'Lewisite'; hence the name). It is used in treatment of certain heavy metal poisoning. It derives its name from the fact that it was developed as an antagonist to Lewisite—a war gas. Many heavy metals such as arsenic and mercury have a great affinity for thiol (-SH) groups and combine with them in the tissues, depriving body of the use of enzymes whose activities depend upon thiol groups. Dimercaprol has two unsaturated SH radicals in it and hence if this is present in the body, it may satisfy the affinity of the heavy metal for thiol groups and so indirectly giving shelter to the enzyme system of the tissues. The compound, found by the heavy metal and the dithiol dimercaprol, is relatively stable and is excreted through urine. **Dose** is usually 3–5 mg/kg body weight, as a preparation of 10% BAL with 20% benzyl benzoate in arachis (ground nut) oil. It is given deep intramuscularly at 4 hourly interval for the first 2 days, followed by twice daily for 10 days or until recovery. **Side effects** include nausea, vomiting, lacrimation, tingling of extremities, etc. To avoid these side effects, the patient may be premedicated with 25 mg of ephedrine sulphate, half an hour before the injection.

Presently, two other related drugs are available. Dimercaptosuccinic acid (DMSA) and dimercaptopropanesulphonic acid (DMPS). These possess the same dithiol chelating grouping as dimercaprol, but the molecules as a whole are more hydrophilic. Unlike dimercaprol, DMSA and DMPS can be used orally and have a better therapeutic index than the older drug.

Ethylenediaminetetraacetic Acid (EDTA) It is a cyclic organic acid that combines with sodium to form its sodium salt. When this salt combines with calcium, it forms a stable chelate known as disodium calcium edetate or disodium calcium versenate. It has great affinity for lead. It is, therefore, treatment of choice for inorganic lead and tetra-ethyl lead intoxication. The chelated lead is excreted in the urine. The calcium complex of EDTA is given by slow intravenous infusion in isotonic glucose saline. **The usual adult dose** is 1 gm twice daily (mixed with 5% glucose saline, in concentration of not more than 3%) at 12 hourly interval, given by slow intravenous drip, each time for over a period of 1 hour, for periods up to 5 days. If necessary, the therapy can be repeated after an interval of 1 week.

Penicillamine (Cuprimine) It is a degraded product of penicillin and has a stable SH radical in it, which helps to combine with the free metal in the circulation. **Dose** usually is 30 mg/kg body weight per day, given orally in four divided doses for 7 days or 1–3 gm in slow normal saline drip for 2–4 days. It is used in copper poisoning encountered in industry and also in lead and mercury poisoning. An important therapeutic use of penicillamine is in hepatolenticular degeneration (Wilson's disease), which is caused by a disorder of copper metabolism.

Desferrioxamine It is a specific antidote for iron. The recommended dose is 1 gm intramuscular in adults, followed by 500 gm 4 hourly for two doses, and finally 500 gm 4–12 hourly up to a maximum of 6 gm in 24 hours. It can also be given as an intravenous infusion (15 mg/kg/h) in Ringer lactate.

It is used both for unabsorbed iron in the stomach and the intestine as well as for the absorbed iron. **Dose** usually is 8–12 gm daily in divided doses, given orally. For absorbed part of the poison, 2 gm is given intravenously with 5% laevulose solution.

Immunotherapy

Specific antibodies that can combine with some toxins have been developed and these act as antidotes. For example, digoxin antibodies (Fab fragments) combine with digoxin to produce an inert complex. Another example is the use of snake antivenin in patients with snake bite.

Treatment of General Symptoms

The treatment of symptoms should be applied as indications arise. In many cases, especially if the poison is unknown, the symptoms afford significant clue to the best treatment of the case. Steps should be taken to counteract by general means the particular symptomatic effect of the poison. The treatment should be directed towards general measures to support life of the patient and to reduce suffering.

33 CHAPTER

Corrosive Poisons

After going through this chapter, the reader will be able to describe: Classification of corrosives and mechanism of action | Vitriolage and its medicolegal importance | Comparison of features of mineral acids | Medicolegal aspects of mineral acid poisoning | Source, clinical findings, diagnosis, management and medicolegal aspects of carbolic acid, oxalic acid and salicylic acid poisoning

Corrosive is a substance that has a corroding and destructive effect on the human body. It is almost exclusively locally acting and has very few systemic effects with the exception of generalised shock. Acids are potent desiccants with the ability to produce a coagulation necrosis and eschar formation of injured tissue. On the other hand, alkalies produce a liquefaction necrosis of tissue. Acids are identified by their pH value, which ranges from 0 to 14; acidic substances are listed from strongest (0) to weakest (6). Neutral pH is 7. Alkaline pH ranges from 8 through 14. Classification is as under:

- **Strong acids**
  - **Mineral/inorganic acids:** sulphuric, nitric, hydrochloric acid (Table 33.1).
  - **Organic acids:** carbolic acid, oxalic acid, acetic acid and salicylic acid.
- **Strong alkalies:** hydrates and carbonates of sodium, potassium and ammonia.

Table 33.1 Differentiating Features of Sulphuric, Nitric and Hydrochloric Acid Poisoning

Features	Sulphuric acid	Nitric acid	Hydrochloric acid
Physical and chemical properties	Colourless, oily, heavy, nonfuming, odourless, hygroscopic liquid, having a painful burning taste. It carbonises organic substances	When pure, colourless, fuming liquid with a pungent odour and painful burning taste. With organic matter (protein), it produces picric acid, thereby staining the tissues yellow (xanthoproteic reaction)	Colourless, volatile, odourless liquid, has a burning sour taste
Sources	Industries, commerce, chemical laboratories	Industries, commerce, chemical laboratories	Industries, commerce, laboratories, etc. (a normal digestive fluid of stomach)
Action	Locally—corrosive (directly); indirectly—shock. Asphyxia—when the acid or the vomitus trickles down the respiratory tract. Chemical peritonitis due to perforation of stomach, malnutrition due to resultant stricture of oesophagus	Locally—corrosive, respiratory distress (when fumes are inhaled); indirectly—shock due to pain	Locally—corrosive, inflammation of respiratory tract due to inhalation of fumes. Indirectly—shock due to severe pain

Table 33.1 (Continued)

Features	Sulphuric acid	Nitric acid	Hydrochloric acid
Fatal dose	5–10 ml (conc.)	10–15 ml (conc.)	15–20 ml (conc.)
Fatal period	12–24 h or more	12–24 h or more	18–36 h or more
Signs and Symptoms			
Burning pain in mouth, throat, oesophagus and stomach, spreading over the abdomen	Present	Present	Present
Erosion of the skin/ appearance	Over angles of mouth, lips, fingers with blackening and excoriation	Over angles of mouth, lips, fingers with yellowish colouration	Usually no erosion. Epidermis may fall-off after a few days
Erosion of mucous membrane of mouth and tongue	Similar as in case of skin	Similar as in case of skin	Similar as in case of skin
Difficulty in speech and swallowing	Present as the mouth is filled with saliva, mucus and corroded matter	Same	Same
Teeth	Chalky white, brittle	Yellowish surface-coating	No significant change
Eructation	Present	Present	Present
Thirst	Present (each attempt to drink is followed by renewed retching and vomiting)	Same	Same
Vomiting	Present	Present	Present
Vomitus reaction	Strongly acid	Strongly acid	Strongly acid
Altered blood	Present	Present	Present
Tenesmus	Present	Present	May be present
Tenderness over abdomen	Present	Present	Present
Stiffness of abdomen	May be due to chemical peritonitis due to perforation of the stomach	May be due to distension of the stomach or less commonly due to rupture of the stomach	May be due to distension of the stomach
Perforation or rupture of stomach	Common	Less common	Uncommon
Urination	Suppressed	Suppressed	Suppressed
Nature of stool	Mucus with altered blood present	Mucus with altered blood present	Mucus with altered blood may be present
Cause of death	Shock, perforation of stomach, peritonitis, laryngeal spasm due to trickling of acid or vomitus or malnutrition due to stricture of the oesophagus	Shock, perforation of stomach, peritonitis, laryngeal spasm due to trickling of acid or vomitus or inhalation of vapour	Shock, laryngeal spasm, pulmonary oedema due to trickling of acid or vomitus or inhalation of vapour of the acid

(Contd.)

Table 33.1 (Continued)

Features	Sulphuric acid	Nitric acid	Hydrochloric acid
Postmortem appearances	• There occurs gross corrosion of skin with blackish or chemical charring of the affected area. Lips, angles of mouth or fingers of the hand and sometimes the chin, front of abdomen and chest show such corrosion due to trickling of acidulated saliva. Corrosion with mild to moderate degree of charring is present in the mucous membrane of the oesophagus	• There is usually corrosion with yellowish discolouration of the skin of affected areas due to xanthoproteic reaction. Similar changes are noticed on the mucous membrane of mouth, tongue and oesophagus	• Here, there may not be much corrosion of the skin. There may be damage of the skin over these areas with brownish parchmentisation. The mucous membranes of the mouth and tongue show reddish brown corrosion. Similar changes may be seen on the inner surface of oesophagus
	• Depending upon the amount and concentration of the acid, the wall of the stomach is soft, swollen with blackish corrosion, desquamation, haemorrhage, ulceration and perforation with occasionally the whole stomach changing to blackish disintegrated mass	• The stomach wall is swollen, soft with desquamation, haemorrhage and ulceration. Perforation is not as common as in case of sulphuric acid. Xanthoproteic reaction is not much prominent due to altered blood, which causes dark brown discolouration of the mucous membrane of the stomach	• The stomach is soft, swollen with congestion, desquamation, haemorrhagic points and ulceration of the inner surface. It contains altered blood with mucous shreds
	• When there is perforation of the stomach with leakage of the acid, extensive corrosion of the nearing organs/tissues with development of chemical peritonitis occurs	• When there is perforation of the stomach with leakage of the acid, corrosion of the nearing organs/tissues with development of chemical peritonitis occurs	• Perforation is uncommon as mentioned above
	• The upper digestive tract after the ingestion of the concentrated acid presents a black swollen, dried and charred appearance of typical **carbonisation** due to extraction of water from the tissues and conversion of haemoglobin into acid haematin	• Upper part of the small intestine shows signs of irritation	• The upper part of the small intestine shows signs of irritation
	• If the vomitus is inhaled, there occurs mild to moderate corrosion of the respiratory tract with congestion and oedema of the lungs.	• In case the vapours are inhaled, there occurs congestion of the respiratory tract with congestion and oedema of the lungs. Similarly, if vomitus is inhaled same findings usually occur.	• In case the vapours are inhaled, there occurs congestion of the respiratory tract with congestion and oedema of the lungs. Similarly, if vomitus is inhaled, same findings usually occur.
Materials to be preserved from the dead	In all these cases, stomach with contents, a loop of upper part of the small intestine, half of liver, half of each kidney are preserved in one container. Corroded areas of skin are preserved in another container. Acid-stained clothes are preserved separately.		
Preservatives	Viscera and skin are preserved in absolute alcohol or rectified spirit, and the clothes are sent without any preservation.		

Mechanism of Action

The extent and severity of chemical injury to the GIT depends upon the interactions of three factors: the corrosive nature of the ingested substance, the quantity and concentration of the ingested substance and duration of contact. When the corrosive agents come in contact with body, there occurs an intense inflammatory reaction in the first 4–7 days. If the patient survives this period, the granulation stage will follow, in which fibroplasia and formation of the collagen starts. During the second and third week, when the tissue is weak, there is a chance of perforation. At the third week, the cicatrisation stage starts and excessive formation of scar tissues will result in stricture. **Their action is characterised by** (*i*) extraction of water from the tissues, (*ii*) coagulation of cellular proteins and (*iii*) conversion of haemoglobin into haematin.

Mineral or inorganic acids are corrosive agents in their concentrate forms. When diluted, they act as irritants and when very much diluted and taken by mouth, some of them may act as stimulants to the digestive process. (Alkalies produce liquefactive necrosis with rapidly penetrating tissue injury and a higher risk of perforation of the oesophagus and stomach than do the acids. Liquids tend to produce superficial, often circumferential burns over a larger surface area, while solids and tablets cause localised but deeper burns.)

Diagnosis and Management

RADIOLOGY

In view of the high rate of perforation, water soluble contrast agents are suggested for evaluation. The effects of acid ingestion on stomach have been described in three stages. In the acute stage (1–10 days), there may be air in the gastric wall, which is an ominous sign of an impending perforation. In the subacute stage (11–16 days), the atony, dilatation and rigidity of the antrum and pylorus are seen. In the chronic stage, stenosis and contraction of the antrum and pylorus occur.

Chest and abdominal X-rays and routine laboratory testing should be obtained to evaluate for aspiration, perforation and organ dysfunction.

ENDOSCOPY

Because of easy availability of endoscopy and the better results, it is preferred to the radiological examination during acute stages of acid and alkali ingestions. It is better performed 12–24 hours after ingestion and is used to document the site of injury and its severity. **It has been reported** that the squamous epithelium of the oesophagus is relatively resistant to acid burns, while the columnar epithelium of the stomach is very susceptible. That is why perforation of stomach is much more frequent than perforation of oesophagus. The duodenum is even more susceptible, but it is usually protected by pyloric spasm. In alkali burns, the squamous epithelium of the oesophagus is most severely affected.

TREATMENT

It includes immediate dilution with milk or water. Administration of a weak acid (carbonated beverage or citrus juice) or base (antacid) is also acceptable.

- Do not administer emetics to a patient who has ingested a corrosive agent.
- Do not perform a gastric lavage, as it may lead to perforation of the stomach or oesophagus.
- Do not give the patient anything orally for 2–3 days after the initial administration of water or milk. Later on, fluids and electrolytes may be given.
- Concomitant prophylactic broad-spectrum antibiotic use is recommended.
- The role of glucocorticoids is controversial.
- Oesophageal stricture or gastric outlet obstruction may require subsequent dilatation or surgical reconstruction.
- Laparotomy is required for patients with gastric perforation and peritonitis.
- Skin lesions need to be washed with soap and water followed by application of some ointment.
- Eye involvement necessitates copious irrigation with water or normal saline. Referral to an ophthalmologist is advisable.

Medicolegal Aspects

- Accidental poisoning is common as many of these agents are found in various household products. Inquisitive toddlers, in particular, are vulnerable to injury from alkaline corrosives, which are often kept under the sink or in old soda bottles in many households. Each year, more than 26,000 American children under 6 years of age ingest such corrosive chemicals—mostly household products such as detergents and drain openers. Accidental poisoning can also occur in adults, the acid being taken by mistake for medicine (nitric acid may be an exception, it being a yellowish liquid giving off very irritating fumes, which render it improbable that anyone could take the acid by mistake for something else).
- In accidental ingestion, the intense acidity usually warns the unintentional taker of his mistake, causing him to spit out the acid. This may be accompanied by a good deal of spluttering, with the result that some of the acid dribbles over the chin and spots of it almost invariably fall on the clothing, producing characteristic stains. Furthermore, attempts to wipe the burning stuff from the lips may result in spreading it over the surrounding area and cheeks, etc.

- Suicidal poisoning is rare in the present scenario. (Why anyone should deliberately select such an agonising form of death, is difficult to understand. However, a determined suicide is up to anything. Even in intentional swallowing of the acid, intense pain may result in spluttering and consequent staining about the mouth, chin and clothing, etc.).
- These acids are not suitable for homicidal purposes as the effects are too immediate and violent. Homicidal victims are usually incapable infants or intoxicated persons.
- Acute exposure to the vapours in the industries may lead to death due to respiratory distress.
- Prolonged exposure to the vapours of these agents in industries may lead to respiratory complications.
- In fatal cases of poisoning by these acids, no trace of the poison may be discoverable in the viscera, especially if the victim had survived for a couple of days or more. Salts of these acids being common constituents of food and medicine, it is important to ascertain whether any of these acids is present in the free condition. The quantity of free acid present is specially important in HCl poisoning, as this acid is contained uncombined with bases in the gastric juice to the extent of about 0.2% or more.
- Sometimes, disposal of the dead body may be effected by throwing the dead body into the acid with an attempt to dissolve the body of the victim as was practised by John George Haigh, the so-called **acid bath murderer**. However, the attempt was not completely successful and the sketchy remains, viz., a part of the left foot, a few bone fragments, some gall stones and a partially dissolved set of dentures recovered from the acid tank enabled to establish the identity of the victim.

Organic Acids

Organic acids differ from inorganic acids in two major respects: (*i*) they are weaker in action and (*ii*) they are usually absorbed into circulation and so have both local and remote action.

CARBOLIC ACID (PHENOL/PHENIC ACID)

Phenol or carbolic acid is a corrosive aromatic hydrocarbon that is widely used as a disinfectant and in the formulation of industrial solvents. It is obtained from coal tar by fractional distillation. Pure carbolic acid consists of long, colourless, prismatic, needle-like crystals, which turn pink on exposure to light. It has a characteristic odour, the so-called **phenolic odour**. It is freely soluble in boiling water, alcohol and oils. Crude commercial carbolic acid is a dark brown liquid containing several impurities like cresol. Household phenol contains 5% phenol in water. Although known as an acid, it does not turn blue litmus red and has no acid reaction. However, it is called an acid because it forms carbolates (salts) when acted upon by strong bases.

Phenol absorption can be the result of cutaneous exposure, inhalation of its vapours and ingestion.

Fatal Dose

10–15 gm is the usual range of the fatal dose.

Fatal Period

Death may occur within a few hours due to respiratory or circulatory failure or within some days due to renal involvement.

Clinical Features

Poisoning from carbolic acid is known as **carbolism**.

- Skin exposure may produce a burning sensation followed by tingling, numbness and anaesthesia. A white, opaque eschar is produced that falls off in a few days leaving a brown stain.
- Inhalation of vapours may be responsible for respiratory tract effects in the form of laryngeal and pulmonary oedema leading to distressed breathing and cyanosis.
- Ingestion causes a strong odour of carbolic acid in the patient's mouth. Immediately following swallowing, there is an intense burning sensation in the mouth and throat. This is followed by abdominal pain and vomiting. The mucous membranes of the lips and the mouth become hard and white.
- Owing to its rapid absorption, gastrointestinal symptoms are followed by CNS and CVS symptoms. Giddiness and insensibility deepening to coma may be shown. Transient CNS stimulation may occur initially in some cases. **Pupils** become small and contracted, the temperature drops to subnormal, the skin becomes cold and clammy, the pulse small and thready, and respiration slow and laboured. Metabolic acidosis occurs in severe cases.
- Renal failure may occur due to direct toxicity as well as due to hypotension and haemolysis. Usually, there is oliguria and albuminuria. When fresh, the urine may not have any change in colour or may be slightly greenish. The urine contains trace of free carbolic acid and the metabolic products of phenol, namely, hydroquinone and pyrocatechol. In the voided urine, these substances are oxidised turning it to a dark, smoky green colour on standing. The urine is scanty and contains albumen and blood casts because of severe irritation of the kidneys. All these urinary findings are grouped together under the term **carboluria**. This used to serve as a warning of the toxic action of carbolic acid when it was being used as an antiseptic dressing in the past. (Lord Lister used it extensively for his 'aseptic' surgery. Today however, it has been largely superseded by safer alternatives.)

Diagnosis

- The urine may show red blood cells, proteins and casts.
- Add a few drops of 10% ferric chloride in urine. A violet or blue colour indicates the presence of phenolic compounds.
- The urine containing carbolic acid also reduces Benedict and Fehling solution.

Management

It consists of decontaminating the skin with extensive irrigation where there has been **cutaneous exposure**. The area should be thoroughly washed with soap solution or 25% alcoholic solution and be treated with some vegetable oil. If need be, the area may be washed again after sometime with soap water.

When ingested, cautious gastric lavage is recommended. (Though the acid corrodes the gastric wall, it also hardens it; in contrast to the softening produced by the other corrosives and therefore, gastric lavage can be done.) It may be carried out with 20% alcohol or glycerol or with some vegetable oil like groundnut oil or castor oil. Alcohol has an advantage in that it dissolves the amount of phenol from the mucous membrane and submucous layer of the stomach. But it makes the absorption of the phenol easy and as such needs to be excreted out quickly. Sodium or magnesium sulphate solution can be used for the lavage because they form harmless salt when react with phenol. **Demulcent** drinks like milk, barley water or egg albumin are also advocated. For **efficient excretion**, intravenous infusion of fluid with sodium bicarbonate solution may be given.

Postmortem Appearances

Externally

The contaminated areas of the skin may appear reddish (whitish discolouration usually does not persist for long), necrosed and sometimes denuded and ulcerated. These changes are notable at the expected sites like fingers, angles of mouth, lips, chin, etc. The mucous membrane of mouth is hyperaemic with desquamation and haemorrhagic points. **Phenolic odour** is usually perceptible. The other poisons that are perceptible by their odour may include acetic acid, phosphorus, hydrocyanic acid, opium, alcohol, ether, chloroform, chloral hydrate, paraldehyde, kerosene, DDT, endrin, nicotine, etc.

Internally

The oesophagus may appear reddish and parchmentised. The mucous membrane of the stomach shows prominent rugae. It is thickened, more or less brownish in colour and looks leathery. The contents appear dark brown mixed with mucus, imparting smell like that of phenol. Perforation of the stomach is unusual due to the wall getting tough in consistency. Phenol passes through the wall of the stomach and thus the surrounding structures may appear necrosed with hardening and parchmentisation. Signs of irritation will be seen in the stomach even when the acid is not swallowed but absorbed from the mucous or skin surface. Similar changes may be noticed in the upper part of the small intestine. The heart may appear flabby. Degenerative changes become apparent in the liver and the kidney, if the death is prolonged.

Materials to be Preserved

In addition to the usual viscera and dress stained with vomitus, any doubtful container from the place of occurrence should be preserved as such. For viscera, saturated solution of common salt is used as preservative. Alcohol or rectified spirit should not be used as they mask the detection of the poison. Blood may be preserved without any preservative.

Medicolegal Aspects

Carbolic acid and a large number of its derivatives are used as antiseptics, disinfectants, caustics, germicides and preservatives. Being easily available, phenol was at time a popular suicidal agent. Of late, these substances have been displaced by safer agents. Phenol is rarely used for homicidal purposes though the cases are on the record in which it has been placed in drinking water and even baked in bread. Its characteristic smell usually renders such attempts abortive. Accidental poisoning is uncommon, though cases sometimes may occur through taking the crude acid by mistake. Some accidental cases may be traced to medicinal use of this substance. Children may take it, as it is a common household disinfectant. Chronic intoxication may occur from inhalation of vapour from industrial sources. Phenol in dilution or mixed with some other agent may be used to cause abortion.

During life, the burns upon lips, chin and cheeks, etc. appear white/dirty white and the destroyed epidermis is bleached and hardened. After death, on drying, these burns will undergo the usual darkening and parchmentising. Even when the acid has been absorbed from some mucous or skin surface, signs of irritation will be seen in the stomach; those of corrosion will be absent.

Carbolic acid may normally occur in the urine in traces in the form of phenol-sulphonate of potassium, derived from the digestion of albuminous substances or of their putrefaction. Engel estimated that the quantity of carbolic acid excreted by a healthy man living on a mixed diet is 15 mg in 24 hours.

OXALIC ACID (ACID OF SUGAR)

It is colourless, prismatic crystalline substance resembling magnesium and zinc sulphate (Table 33.2). It is bitter in taste and soluble in water. It vapourises on heating and sublimates on cooling of the vapour.

Different Sources

Metal cleaning agents and stain removers containing oxalic acid. Leather, dye and book binding industry. Rhubarb (particularly leaves), spinach, lichen, onion, cabbage, etc.

Table 33.2 Differentiating Features of Oxalic acid, Magnesium Sulphate and Zinc Sulphate

Features	Oxalic acid	Magnesium sulphate	Zinc sulphate
Taste	Sour and acidic	Bitter and nauseating	Bitter and metallic
Heat	Sublimes	Not so	Not so
Reaction	Strongly acid	Neutral	Slightly acid
Reaction with sodium carbonate	Effervescence but no precipitate	No effervescence but a white precipitate	No effervescence but a white precipitate
Ink/iron stains	Bleaches	Not so	Not so

Mechanism of Action

It has two distinct effects, local and remote. When given in a strong solution, it exerts a corrosive action on the mucous membranes. After absorption, it carries a strong remote (systemic) effect producing hypocalcaemia, nephrotoxicity, etc. It combines with serum calcium to form insoluble calcium oxalate thereby resulting in hypocalcaemia. The reduction in available calcium leads to muscular stimulation with convulsions and ultimately leading to collapse.

Fatal Dose

15–20 gm usually causes fulminating poisoning and death.

Fatal Period

In case of fulminating poisoning, death may occur within a couple of hours. In case of hypocalcaemia, death may occur within 12 hours. In case of uraemia or renal failure, death may occur between 2 days and 2 weeks.

Clinical Features

Oxalic acid does not cause much corrosion of the skin locally. There may be some degree of irritation and if the person survives, cuticle may fall off after a few days. There is a sour taste in the mouth, burning in the throat and stomach within a short time. Vomiting is severe and black in colour due to altered blood. Large doses may cause rapid death from shock. Where the death occurs shortly, intestinal tract may not be affected, but when life is prolonged, there is pain and tenderness over the abdomen, and purging and tenesmus may appear. After absorption, signs of collapse and prostration soon appear. Hypocalcaemia causes tingling and numbness of fingers and limbs. There is muscular tenderness, irritation and tetanic convulsions. Respiration is slow. There is bradycardia with weak, irregular pulse. Ventricular fibrillation may lead to death.

If the patient survives hypocalcaemia, there may be toxic nephritis. In such cases, there is uraemia with scanty urination, haematuria, albuminuria, **oxaluria** (presence of calcium oxalate crystals in the urine is termed as oxaluria. These crystals have the shape of an envelope when seen under a microscope). In fatal cases, death usually occurs within 2 days to 2 weeks.

Management

- Since the degree of corrosion is not as severe as in mineral acids, a soft stomach tube can be passed with care and the stomach washed out using calcium lactate (2 teaspoonful per lavage).
- Demulcent drinks should be given to protect the mucous membrane of the stomach from the corrosive action of the acid.
- Bowel wash by enema and purgatives to remove the unabsorbed poison from the gut.
- Calcium gluconate may be given by mouth or 10 ml of a 10% solution slowly intravenously, which will combine with the oxalic acid in the circulation and will thus save the calcium of the blood.
- In severe cases, parathyroid extract may be given. Urinary output should be checked to detect the possibility of renal damage and fluid intake controlled as the circumstances may be.
- Rest of the treatment is symptomatic.

Postmortem Appearances

These vary with the degree of concentration of the acid. Whether the mucous membrane is reddened or bleached depends upon the concentration of the acid; the stronger the solution, the more likely is bleaching to occur.

Externally

The skin and the lips are not much corroded. But the mucous membrane of the mouth and tongue are corroded, swollen, sodden, bleached or brownish with occasional desquamation and haemorrhagic points.

Internally

Same picture may be noticed inside the mouth and oesophagus. The stomach is swollen and soft. The wall is reddened or, perhaps more frequently, bleached. On this background of red or dirty white, numerous dark brown or almost black streaks may be found running along the length of the stomach, often forming a network over the surface. (The formation of brownish streaks is due to the action of the acid on the blood in the vessels of the stomach wall. The stomach presents such a characteristic appearance that a single glance will suffice to say that the oxalic acid was responsible for death.) There are desquamation and haemorrhages. Perforation is rare. Cloudy areas of deposits of calcium oxalate, partly amorphous, partly crystalline, may be

found. Stomach may contain glairy, brownish, gelatinous mucus mixed with altered blood. The upper part of the small intestine may show signs of irritation. Kidneys also show signs of irritation. There may be evidence of toxic nephritis, the degree of which depends upon the period of suffering.

Medicolegal Aspects

Oxalic acid and its salts are used extensively in industry in bleaching materials for straw hats and vegetable fibres, wood, leather, ink stains, etc., and for discharging dyes in calico printing. Because of its easy availability at home, it has occasionally been used for suicidal purposes. Its homicidal use is not easy due to its detectable taste, early onset of signs and easy chemical detection in vomitus and body organs. Accidents may occur at home in children when it is taken by mistake for epsom salt (mag sulphate) as purgative.

Two potassium salts of the acid are in common use in arts, viz., binoxalate and quadroxalate. Both are sold under the names of 'salt of sorrel' and 'essential salt of lemons', and both are nearly as poisonous as oxalic acid itself. Alkaline oxalates are found in many plants, for example, in wood sorrel and its Indian variety *Rumex vesicarius* (*chuka*), spinach, rhubarb, cabbage, lichens and guano. Hence, it may gain access to the body through food and drugs of vegetable origin. It often occurs as a constituent of human urine, 0.02 gm being excreted in 24 hours. According to Hodgkinson et al., 9–24 mg oxalates are excreted daily, and the oxalic acid content of the tissue seldom exceeds 1.0 mg/100 gm. Hence, in cases of alleged poisoning by oxalic acid or an oxalate, the postmortem appearances and the determination of quantity of poison may be of importance.

SALICYLIC ACID

This is an odourless, crystalline solid substance with a sweetish acid taste. It is mostly used externally for treatment of skin diseases. It causes marked irritation of the gastric mucous membrane and also has a remote action after absorption. Its important preparations are sodium salicylate, methyl salicylate (**oil of wintergreen,** which is actually a liniment containing 25 gm methyl salicylate per 30 ml) and acetyl salicylic acid (**aspirin**). Salicin and methyl salicylate are naturally occurring forms of salicylates found in the leaves and bark of a number of plants, especially the willow tree (*Salix alba vulgaris*).

Clinical Features

- In therapeutic doses, aspirin is absorbed rapidly from the stomach and small intestine, but in overdose, absorption may occur more slowly and the plasma salicylate concentration may continue to rise for up to 24 hours.
- Salicylates stimulate the respiratory centre in the medulla and increase the rate and depth of respiration. As a result, CO_2 is eliminated from the lungs resulting in **respiratory alkalosis**. In an attempt by the body to compensate, bicarbonate accompanied by sodium, potassium and water is excreted in the urine. Dehydration and hypokalaemia result.
- A variable degree of metabolic acidosis develops because of interference with carbohydrate, lipid, protein and amino acid metabolism by the salicylate ions. Inhibition of citric acid cycle enzymes causes an increase in circulating lactic and pyruvic acids.
- A primary toxic effect of salicylates in overdose is uncoupling of oxidative phosphorylation resulting in hyperpyrexia and sweating. Fluid loss is enhanced because salicylates stimulate the chemoreceptor trigger zone and induce nausea and vomiting.
- CNS: development of acidaemia allows salicylates to penetrate tissues more readily and leads, in particular, to CNS toxicity characterised by excitement, tremor, delirium, convulsions, stupor and coma—the so-called **'salicylate jag'**. Tinnitus and deafness are attributed to increased labyrinthine pressure and/or an effect on the hair cells of the cochlea. Coma occurs in terminal stages.
- Renal involvement may be shown by proteinuria, sodium and water retention, and tubular necrosis.
- Salicylate intoxication may be accompanied by hypoprothrombinaemia due to a warfarin-like action of salicylates on the physiologically important Vitamin K_1 epoxide cycle.

Fatal Dose

Salicylic acid	70–80 gm
Sodium salicylate and acetyl salicylic acid	15–20 gm
Methyl salicylate	10–20 ml

(Methyl salicylate is particularly toxic because of rapid absorption and one teaspoonful [5 ml] contains the equivalent of 6.9 gm of aspirin.)

Fatal Period

The fatal period in case of salicylic acid may vary from 4 to 7 days, in case of sodium salicylate 1–3 days and in case of methyl salicylate 12–24 hours.

Management

- Stomach wash can be useful even though several hours may have elapsed since ingestion because blood levels of aspirin may continue to rise several hours after ingestion. Gastric lavage must preferably be done with sodium bicarbonate solution. Activated charcoal suspension can be administered in the usual manner.
- Forced alkaline diuresis can be helpful in eliminating aspirin or other salicylates from the body. Sodium bicarbonate in a dose of 1–2 meq/kg may be given intravenously initially with subsequent administrations as required.

- IV fluids and electrolytes depending upon the situation.
- Vitamin K_1 can be given if there is severe hypoprothrombinaemia.

Postmortem Appearances

These include evidence of haemorrhagic gastritis, subpleural and subpericardial haemorrhages, pulmonary and cerebral oedema, signs of renal irritation and congestion of viscera.

Medicolegal Aspects

Accidental poisoning with aspirin in children has been reported. This has however become relatively rare since the introduction of paracetamol (acetaminophen). In adults, accidental fatalities are uncommon except those cases involving hypersensitivity reactions. In neonates, infants and children, salicylate intoxication may occur inadvertently through placental transfer, breast milk or by the application of teething gels to the gums. Suicidal poisoning with this drug is again uncommon these days.

- Studies in the United States have suggested an association between Reye syndrome and the use of salicylates. Salicylates, therefore, should not be used in children under the age of 12 years unless specifically indicated for a childhood rheumatic condition.
- Aspirin hypersensitivity: Sometimes a single therapeutic dose of aspirin may provoke a fatal hypersensitivity reaction. Within minutes of ingestion, there occurs acute vasomotor rhinitis, angioneurotic oedema and urticaria. Laryngeal oedema may rapidly result in death. Treatment involves the immediate administration of adrenaline (subcutaneously) and corticosteroids.
- Aspirin has been reported to have an unexpected salutary effect in the form of inhibition of platelet aggregation and reduction of risk of thrombosis. In fact, it has been advised that persons who are at risk should take half an aspirin a day as a prophylaxis against heart attack.

Vitriolage

The term vitriolage literally means throwing of the oil of vitriol (concentrated sulphuric acid) on the body of a person with the intention of injuring or disfiguring him out of jealousy or revenge. The method was commonly practised by industrial workers in Glasgow before the Offences Against the Person Act, 1861 was passed in England. For practical purposes, this term is used in all cases of throwing of any corrosive agent on the body of a person. The use of caustic soda, caustic potash and marking nut juice has also been recorded. The usual target is the face of the victim with the idea to cause its disfiguration. This may lead to the destruction of the eyes with permanent loss of vision. There is ulceration and resultant scar formation of the contaminated area of the body. Due to damage of the nerve endings, these are comparatively painless lesions. Evidence of spilling or splashing or pouring of the acid may be available with the involvement of wide area of the body. These injuries heal with scar formation and thus cause permanent disfiguration, loss of eyesight, or contracture, which, if near the joint, may restrict the functions of the joint. Thus, if the victim survives any of such outcomes, the act of vitriolage amounts to the causation of **grievous hurt**. But when the area of involvement is extensive, death may occur wherein the offence will amount to homicide. Immediate treatment consists in washing away the corrosive acid with large amount of water and soap. The raw surface may afterwards be covered with some antibiotic ointment. When the eyes are involved, they should be washed at once with a large amount of water followed by irrigation with 1% solution of sodium bicarbonate.

34 CHAPTER

Nonmetallic and Metallic Irritants

After going through this chapter, the reader will be able to describe: Features, diagnosis and management of poisoning by nonmetallic irritants like phosphorus and its medicolegal aspects | Features, mechanism of action, diagnosis and management of poisoning by metallic irritants like arsenic, lead, mercury, etc., and their medicolegal aspects | Features, mechanism of action, diagnosis and management of thallium poisoning

It is fallacious to sharply discriminate between corrosives and irritants. An irritant poison is only a milder form of a corrosive. In other words, a corrosive, when diluted or taken in a less concentrated form, becomes an irritant. The converse is also true. This group produces mainly manifestations of gastroenteritis with variable involvement of other organ systems.

Nonmetallic Irritants

PHOSPHORUS

There are mainly two varieties—red and yellow. **Yellow (or white) phosphorus** is crystalline and highly toxic. It is translucent, waxy and luminous (glows in dark). It has a garlicky odour and is highly volatile. It ignites at room temperature giving off dense white fumes of phosphorus pentoxide and hence must be constantly stored under water. Till 1931, yellow phosphorus was being used in the manufacture of Lucifer matches. **Red phosphorus** is inert unless contaminated with yellow phosphorus. This is a reddish-brown powder, which has no taste or smell and is nonpoisonous. It is used in the manufacture of 'safety matches'. The tip of the matchstick contains a mixture of potassium chlorate and antimony sulphide. It is ignited by rubbing against the side of the matchbox known as the striking surface, which is covered with a thin layer of red phosphorus and powdered glass.

Yellow phosphorus is an ingredient of tracer bullets, incendiary bombs, smoke screens and air-sea rescue flares. There are several pastes/powders available in India that contain phosphorus (zinc phosphide) used as rodenticides; for example, Ratol. Such pastes are usually mixed with molasses or butter and spread on bread as bait.

Derivatives and related compounds of phosphorus include phosphoric acid, phosphine, aluminium phosphide and zinc phosphide.

Mechanism of Action

Phosphorus is a protoplasmic poison. It affects cellular metabolism comparable to ischaemia produced by embolism during life. Under such anaerobic condition, metabolism of cells may still proceed but will considerably be diminished because of lack of energy. This is what may be termed as **necrobiosis**, which is characteristically manifested in the liver.

Acute Poisoning

Massive intake of phosphorus (over 1 gm) results in **fulminant poisoning**. The dominant clinical feature is peripheral vascular collapse. Death may occur in 12 to 48 hours. **Acute poisoning** results from a moderate dose of phosphorus (0.1–1 gm). Usually, there are two phases—primary, due to local irritant action on the gastrointestinal tract and secondary, due to action of the absorbed poison; there is usually a considerable interval between them. **The primary symptoms** occur usually within 2–6 hours. Occasionally, the onset may be immediate. The initial features include a garlic-like taste, and burning in the mouth, throat, retrosternal area and epigastrium. These are followed by nausea, vomiting and sometimes diarrhoea. The vomitus has a garlic odour and is luminous in the dark.

In most cases, the above symptoms abate temporarily and after a gap of 2–6 days **secondary symptoms** appear due to action of the absorbed poison. The original symptoms return and, in addition, jaundice appears. Abdominal pain increases and distention becomes evident. Vomiting becomes distressing and bleeding tendencies become prominent. Hepatic and splenic

enlargement occurs. In early stages, liver is enlarged due to fatty degeneration (necrobiosis); in the late stages, it is shrunken due to necrosis (acute yellow atrophy). Purpura and epistaxis may follow due to hypoprothrombinaemia. The patient may develop acute renal failure, oliguria and albuminuria. Nervous symptoms develop in the later stages, and these may include headache, restlessness, tinnitus, deafness, impaired vision, convulsion and coma. Priapism is frequent. Death usually follows hepatic and renal insufficiency.

Fatal Dose

A dose of 60–120 mg is usually considered fatal, though vomiting may permit recovery from much larger doses.

Fatal Period

Death may occur from collapse within 24 hours. Symptoms usually last several days, and death may ensue in a week or even longer.

Management

- Oily and fatty substances including milk are contraindicated, as these increase the absorption of phosphorus.
- Stomach should be washed with a weak (1 in 5000 concentration) solution of potassium permanganate, till no more smell of garlic is perceptible. The bowel should be emptied by a brisk purgative. Potassium permanganate acts as a chemical antidote in oxidising the phosphorus and forming harmless compounds, i.e. phosphoric acid and phosphates.
- Intravenous saline is useful to combat shock.
- Calcium gluconate is indicated as the blood calcium is diminished.
- Alkaline reserve is also diminished by phosphorus and administration of sodium bicarbonate has been suggested.
- Dextrose may be needed to protect liver.
- Vitamin B complex, K and C are necessary.

Postmortem Appearances

These differ according to whether death takes place within the first 24 hours or after the lapse of a few days. **In the former case,** the appearances generally are those of a highly irritant poison, consisting of inflammation or erosion of the mucous membrane of pharynx, oesophagus, stomach and intestines. Cloudy swelling of the liver and kidneys may be the other findings. The contents of stomach may be luminous in dark, and the body may smell of garlic. **In the latter case**, the appearances generally show the following.

Externally Jaundice and haemorrhages under the skin and various natural orifices of the body. The body may be emaciated and may smell of garlic.

Internally Fatty degeneration and haemorrhages are the characteristic features. The toxic effects are well-marked in the stomach and intestines. The mucous membrane of the stomach and intestine is yellowish or greyish-white in colour, softened, inflamed or even corroded. Patchy areas of erosion with haemorrhages are common. The contents of the stomach and intestines may have a garlicky odour and may be luminous in the dark. The liver undergoes necrobiosis, as mentioned earlier. Fatty degeneration of the heart muscle, kidneys, etc. may be seen. Subendocardial haemorrhages in the left ventricle are usually present. The blood may appear tarry and its coagulability is diminished.

The viscera should be preserved in saturated solution of common salt and not in spirit as the luminosity is lost.

Chronic Poisoning

This was relatively common among workers in match factories in olden days when Lucifer matches (containing white phosphorus) were in use. Today, however, after the advent of safety matches, incidents of chronic poisoning are rare. Chronic poisoning usually occurs by way of inhalation of fumes of phosphorus over a period of time. Months or even years may elapse before a victim begins to complain. It is supposed that the vapours gain access to the jaw through a carious tooth or an interspace caused by the missing tooth, where the suppurative microorganisms are already present. The initial symptom is toothache, followed by swelling of jaw, loosening of teeth, necrosis of gums and sequestration of bone in the mandible. This is known as **phossy jaw** and was first described by Bristowe in 1862. There may also be systemic manifestations like anorexia, weakness, joint pains, hepatic damage, etc.

Treatment

Teeth of the workers should be regularly examined and if found carious, should be filled-in or extracted. Periodic X-rays of upper and lower jaw should be advised for early detection of bone caries. Removal of the patients from further exposure and surgical excision of sequestered bone is advised.

Medicolegal Aspects

- Intensity of poisonous effect is more if phosphorus is in solution form than when it is in the form of solid lumps.
- Accidental poisoning may occur in individuals (especially children) who get poisoned inadvertently through fireworks, rat pastes, etc. Pregnant women have often been accidentally poisoned by phosphorus, as they take it to induce abortion.
- Suicidal poisoning used to be witnessed in the past, when Lucifer matches were in use. The common method was to soak several match heads in water or brandy and consume the resulting potion.
- Homicidal use is rare. The typical taste, the typical odour and the luminosity in darkness is helpful in establishing its presence in an individual case.
- **Arson:** Sometimes phosphorus, wrapped in a wet rag or covered with dung, is placed on the thatched roof of a house

or among articles that easily catch fire. When the cloth or dung dries, phosphorus ignites resulting in a conflagration. Insurance frauds may be committed in this way.

- Rolled up in a wet cloth or dissolved in carbon bisulphide, it was also employed to set fire to postal letter boxes during the Civil Disobedience Movement in 1932.
- It needs be borne in mind that phosphorus occurs in combination mainly as phosphates in the various articles of food, and in the tissues and fluids of the body. Hence, its detection in these forms has no value for medicolegal purposes. However, its presence in body in the elementary form is sufficient to prove poisoning, as it does not freely occur in nature.
- It can be detected even in putrefied bodies. Other poisons that can be detected in putrefied bodies may include arsenic, antimony, *dhatura*, endrin, strychnine, oleander, nicotine, etc.

Metallic Irritants

Though their overall role in poisoning has come down over the years, many of metals or their salts still cause serious morbidity and even mortality from time to time. However, many of these cases are chronic in nature arising mainly out of industrial or occupational exposure. The important poisons in this group are arsenic, lead, mercury, thallium, copper, zinc, etc. (For copper, zinc and some other essential metals poisoning see Chapter "Food Poisoning".)

ARSENIC

For a long time (until a few decades ago), arsenic had been used as homicidal poison. It was a poison of choice because in both acute and chronic administrations, the signs and symptoms mimic natural disease processes and fail to arouse suspicion of foul play. It also has the advantage of being odourless and tasteless, and therefore easy to administer (Table 34.1 shows various preparations).

Table 34.1 Important Arsenical Preparations

Compound	Uses
Arsenic trioxide (arsenious acid/white arsenic, vernacularly known as ***sankhya*** and ***somalkhar*** in the local language)	Previously used in medicine for treating fever (e.g., Fowler solution contained 1% white arsenic). Now, its use is restricted to rat poisons, flypaper, etc. It is also often used to protect timber and hides from termites
Copper compounds of arsenic [copper arsenite (Scheele's green) and copper acetoarsenite (Paris or Emerald green), known as ***hirwa*** in the local language]	Colouring agents for wall paper, toys, etc.
Sulphides of arsenic [arsenic disulphide (red realgar) and arsenic trisulphide (yellow orpiment)]	Sometimes used as depilatories
Arsenical compounds of lead, sodium and potassium	Weed killer, insecticide, fungicide, etc.
Organic arsenicals (carbarsone, tryparsamide, melarsoprol, etc.)	Treatment of trypanosomiasis, amoebiasis, etc.
Arsine gas (arsenic trihydride or arseniuretted hydrogen)	Industry and laboratory

Metabolism

The toxicity of an arsenic containing compound depends upon its valence state (zero-valent, trivalent or pentavalent); its form (inorganic or organic) and physical aspects governing its absorption and elimination. In general, inorganic arsenic is more toxic than organic arsenic, and trivalent arsenite is more toxic than pentavalent and zero-valent arsenic.

Mechanism of Action

- Acute arsenic poisoning from ingestion results in increased permeability of small blood vessels, and inflammation and necrosis of the intestinal mucosa; these changes manifest as haemorrhagic gastroenteritis, fluid loss and hypotension.
- Toxicity is also attributed to the ability of arsenic to combine with sulphydryl groups in the mitochondrial enzyme systems leading to interference with their action. Arsenate causes uncoupling of mitochondrial oxidative phosphorylation.

Acute Poisoning

- Initially, there is usually a metallic taste in the mouth and some odour of garlic in the breath along with dry mouth and dysphagia. Besides arsenic and arsine poisoning, the garlic odour is typical of phosphorus, selenium, thallium and organophosphate intoxication. Severe nausea and vomiting, colicky abdominal pain and profuse diarrhoea with rice water stools abruptly ensue. There may occur blood in the stools. Stools may frequently contain shreds of mucous membrane and fragments of the poison. Intense thirst is a constant feature, but drinking water accentuates the vomiting. Painful cramps in the legs may develop due to dehydration and the urine may be suppressed.
- Following GIT phase, multi-system organ damage may occur. Delayed cardiomyopathy accompanied by electrocardiographic abnormalities may develop. Collapse sets in, with cold clammy skin, pale anxious face, sunken eyes, rapid

feeble pulse and sighing respiration. Though convulsions or coma may precede death, consciousness is usually retained until near the end. If death does not occur in the first few hours from shock, it may result from acute hepatic or renal failure, which develop over the next few days.

Fatal Dose

The fatal dose of arsenic trioxide is usually in the range of 200–300 mg. In general, the pentavalent form of arsenic (arsenate) is less toxic than the trivalent form (arsenite) because it is less water soluble. The most toxic form is arsine gas.

Fatal Period

When a large dose of arsenic is taken and the poison is quickly absorbed, average fatal period may be 2–4 hours. In the gastrointestinal form, the average fatal period may range from 12 to 48 hours.

Subacute Form of Poisoning

This usually results when arsenic is administered in small doses at regular intervals to cause death by gradual prostration. Symptoms usually manifest in the form of dyspepsia, cough and tingling in the throat followed by vomiting, purging with abdominal pain and tenesmus. Stools are bloody. Feeling of depression and languor may occur. The symptoms of neuritis are more pronounced. The victim complains of cramps in the muscles and is restless. Ultimately, collapse may occur leading to death.

Chronic Poisoning

This is much more insidious in nature. The onset of symptoms usually occurs at 2–8 weeks. Chronic poisoning may occur among persons engaged in smelting and refining of ores and in the subliming of white arsenic in the manufacture of sheep dips, weed killers, paints, dyes, cosmetics, drugs, etc. It needs to be remembered that chronic poisoning may follow acute poisoning, especially when recovery has occurred from a large dose of arsenic. The symptoms may include the following:

- **GIT symptoms** are usually associated with loss of weight, malaise, loss of appetite, salivation, colicky pain, constipation or sometimes diarrhoea and vomiting of glairy mucous tinged with bile. Tongue is usually coated with a thin, white, silvery fur. Circumscribed oedema of lower eyelids and ankles may be seen.
- **Skin symptoms** usually begin with a persistent erythematous flush leading to hyperkeratotic skin and desquamation. Pigmentation is patchy ('raindrops on a dusty road'). Hyperkeratosis is most prominent in the distal parts of the body. A diffuse desquamation of the palms and soles is characteristic. **Mees lines** (transverse white striae of the fingernails, technically called striate leukonychia) may also be seen.
- **Anaemia and leukopaenia** are commonly seen. Thrombocytopaenia is also frequently seen. Anaemia is normocytic and normochromic and is partly caused by haemolysis.
- **Peripheral neuropathy:** Sensory and motor polyneuritis (sensory symptoms usually predominate) manifesting as numbness and tingling in a 'stocking glove' distribution and distal weakness are the important features. Respiratory muscle involvement may also occur.

Diagnosis

When acute arsenic poisoning is suspected, an X-ray of the abdomen may reveal ingested arsenic, which is radiopaque. The serum arsenic level may exceed 7 μg/dl. However, arsenic is rapidly cleared from the blood. Urinary arsenic should be measured in 24-hour specimens collected after 48 hours of abstinence from seafood ingestion. Normal levels of total urinary arsenic excretion are less than 50 μg/dl, excretion of 100 μg or more per day is indicative of poisoning. Urine becomes positive within 6 hours of poisoning and may continue to be positive for about a couple of weeks. Arsenic may be detected in the hair and nails for a prolonged time after exposure. (It was previously thought that it took a week or two for ingested arsenic to appear in the keratinised tissues such as hair and nails, but more sensitive analytic techniques have shown that metal can appear there within hours. The appearance of the metal in these tissues, possibly, is due to its rapid excretion into sweat and sebaceous secretions and spreading into the hair and nails via surface diffusion.)

Management

- Vomiting should be induced with ipecac in the alert patient with acute arsenic ingestion. Gastric lavage may be carried out with warm water. Then freshly precipitated hydrated ferric oxide may be administered with the object of forming ferric arsenite, a harmless salt. Activated charcoal with a cathartic may be tried.
- Aggressive therapy with intravenous fluid and electrolyte replacement may be life-saving.
- Dimercaprol is the chelating agent of choice and is administered intramuscularly at an initial dose of 3–5 mg/kg on the following schedule: 4 hourly for 2 days, every 6 hours on the third day and every 12 hours thereafter for 10 days. Continue the administration of dimercaprol till the urinary arsenic excretion is less than 50 μg/24 hours. There is now increasing evidence that DMSA (succimer) and DMPS (unithiol) may be preferable. They are more effective in reducing the arsenic content of tissues and unlike dimercaprol, they do not cause accumulation of arsenic in the brain. DMSA and DMPS may be given orally (in a dose of 30 mg/kg body weight daily), whereas dimercaprol needs to be given by deep intramuscular injection.

Postmortem Appearances

The character of the appearances depends largely upon the quantity taken and the period that has elapsed before death. **Externally:** Rigor mortis lasts longer than usual. The body sometimes presents a shrunken appearance due to dehydration. The eyeballs are sunken and the skin, chiefly of hands and feet, may be cyanosed. The skin may be found jaundiced. **Internally:** The stomach is primarily the seat of postmortem appearances, although the poison may have entered the body by means other than the mouth. The stomach has been described as having an appearance like that of **red velvet**. The mucous membrane is usually covered with a considerable amount of tenacious mucus often tinged with blood, and is swollen and red in appearance. The colour may be brown than red. The distribution is usually patchy and corresponds to the deposits of stray particles of arsenic, around which the inflammatory changes are evident. Ulceration or erosions may be found, especially at the pyloric end. Submucous petechial haemorrhages are often seen. If decomposition has occurred, the white oxide will have changed in colour to the yellow sulphide and deposits of yellow streaks will be found mainly under the peritoneal coat of the stomach and to a lesser extent in the small intestine. The mucous membrane of the small intestine is usually inflamed in its upper part. It appears flabby and contains large flakes of mucous with little faecal matter.

Petechial haemorrhages under the endocardium of the left ventricle are comparatively common and may be found even when the stomach shows little sign of irritation. They constitute an important finding for diagnosis. They may also sometimes be found in poisoning from phosphorus, antimony, mercury, heat stroke, deaths from acute infections, traumatic asphyxia and poisoning from viper bite.

Liver, spleen and kidneys may show patchy fatty degenerative changes.

Medicolegal Aspects

Though arsenic is odourless and tasteless, it is not really an ideal homicidal poison, because it is relatively insoluble and can be mixed with water or food with difficulty. It is more readily soluble in hot preparations, but on cooling it separates out yielding a gritty deposit. In most homicidal cases, arsenic used to be administered through the mouth after disguising it with articles of food, such as sweetmeat, bread, *dal*, drinks such as milk, tea, coffee, *sharbat*, port wine or with medicine. It has sometimes been given with prepared *paan* or with the tobacco of a cigarette.

- Accidental cases of poisoning sometimes occur from its admixture with drink or articles of food or from its improper medical use. White arsenic has been mistaken for baking powder, soda, salt or flour and has caused mass accidental poisoning. Epidemiological evidence has linked chronic consumption of water containing arsenic at concentrations in the range of 10–1820 ppb with diabetes, vasospasm and peripheral vascular insufficiency culminating in 'blackfoot disease' (a gangrenous condition affecting the extremities).
- Chronic arsenical poisoning with the symptoms of peripheral neuritis broke out among beer drinkers in an epidemic form in Lancashire in 1900. The beer was found contaminated with arsenic.
- Individuals who are in the habit of taking arsenic acquire a certain amount of tolerance. The mountaineers of Styria and Tyrol used it daily with a view to becoming hardier to carry weights and climb mountains.
- Arsenic is eliminated through urine, faeces, skin, hair and nails, and to some extent through the sweat, saliva, bile and milk, etc. After its administration, arsenic appears in urine and faeces usually from 2 to 8 hours. The elimination of these channels continues for a period of 2–3 weeks, after which arsenic is not found in the urine and faeces, although it may be found in the hair and nails. By dividing the hair in small successive lengths from the root upwards and measuring the concentration in different parts of the hair shaft, one may get important information regarding the time that has elapsed since the administration of arsenic, as the most recent doses will be nearer the root.
- In fatal cases of acute poisoning, where the victim has survived for some days, it is hardly possible to find the poison in the viscera. A case is reported in which arsenic was found in the vomit and faecal matter, but was not found in viscera when death occurred after 6 days. A case is also reported where arsenic was recovered from the earth mixed with vomit, but not from the viscera when death occurred 2 days after severe vomiting.
- Arsenic is excreted into the stomach and intestines after absorption, even when administered by channels other than mouth. Hence, detection of arsenic in these organs does not prove that it had necessarily been given through the mouth.

  In a case reported by Stitch, arsenic was detected in stomach contents of a woman who had been poisoned by introduction of a large amount in vagina. In another case of poisoning per vaginum, it was found in the stomach contents and faeces of a woman and also in the organs of her three-month old foetus (Peteroon, Haines and Webster, Legal Medicine and Toxicology; Vol II, 23).
- The greatest concentration of arsenic is found in hair and nails. Its deposition in hair may begin in 15 days after administration.
- Arsenic becomes fixed in the cancellous tissue of bones, chiefly the long bones, owing to the conversion of their phosphates in to arsenates. Its elimination being much slower, its presence can be detected in the bones for prolonged periods. Hence, it is prudent to preserve long bones for chemical analysis in suspected cases of chronic arsenical poisoning when a body is exhumed or when it has reached advanced

putrefaction. (Traces of arsenic were found in femurs removed from the body of the late Fulham of Agra exhumed 14 months after burial. The grave was "*katcha*" one and the lid of the coffin had already given way).

- Arsenic resists putrefaction to a certain extent. In cases of its prolonged administration, the stomach and other tissues may often be well-preserved even some months after death.
- Arsenic is physiologically not a normal constituent of body, but it is widely distributed in nature. It has been found in minute quantities in several varieties of vegetables and apples because of spraying of fruit trees with arsenic preparations.
- Question sometimes may arise whether arsenic can be introduced into the stomach after death and postmortem imbibition can occur in the tissues. The fact of ante- or postmortem imbibition of arsenic can be ascertained by examining the condition of the mucous membrane of the stomach and other tissues.
- When arsenic has been found in exhumed bodies, question may creep up as to whether it had been absorbed from the earth that surrounded the body. It needs be remembered that arsenic found is usually an insoluble salt mixed with lime or iron and hence it cannot percolate into the cadaver buried in such soil. However, to avoid the possibility of any doubt, samples of earth surrounding the coffin or the body should be preserved for chemical analysis.
- Poisoning may occur due to inhalation of arseniuretted hydrogen in factory workers or some circumstance occasioning inhalation of the gas. A case has been reported wherein a man sustained severe injuries including a penetrating wound of abdomen and laceration of left hand from the explosion of a powder comprising of potassium chlorate and arsenic sulphide in a porcelain jar. Six days after the explosion, man's urine was found to contain 0.24 mg of arsenic. Twenty days after this, he developed dermatitis and his hair and nail pairings were found containing 2.4 mg of arsenic. Here, arsenic seems to have entered the system through the wounds and by inhalation of arseniuretted hydrogen that was evolved in the explosion.

Napoleon Bonaparte (Emperor of France from 1804 to 1815) has been the source of attention of the historians because of his flamboyance and daring exploits, and perhaps also because of his tragic demise. On being defeated in legendary Battle of Waterloo in 1815, he was exiled to the very remote island of St. Helena (a British colony in the Atlantic), where he died a mysterious death. His death remained speculative until the scientific authenticity was finally established. Some hair from the scalp were procured by Ben Weilder (a Napoleonic scholar) and handed over to Dr. Antommarchi. Hair samples were submitted to neutron activation analysis, revealing fluctuating levels of arsenic throughout the length of the hair, ranging from 4.4 to 23.0 parts per million. Another sample (comprising just two strands of scalp hair, taken when he was alive) revealed 16.8 ppm of arsenic in one and 33.3 ppm in the other, as demonstrated by graphite furnace atomic absorption spectroscopy. The Heavy Metals Laboratory of Mayo Clinic, Minnesota (USA) states that 1 ppm of arsenic in hair is the baseline level, while any level more than 10 ppm is indicative of significant toxicity.

LEAD (SHISHA)

It tops the list of heavy metals as far as the chronic poisoning is concerned, which is known as plumbism. The twentieth century saw both the greatest ever exposure of the general population to lead and an extraordinary amount of research on lead toxicity. Some of the important salts and their uses are given in Table 34.2.

Metabolism

Elemental lead and inorganic lead compounds are absorbed through ingestion or inhalation. Organic lead (tetra-ethyl lead) is absorbed to a significant degree through the skin as well. Lead is absorbed into blood plasma, where it equilibrates rapidly with extracellular fluid and accumulates in soft and hard tissues. In the blood, about 95–99% of lead is sequestered in red cells, where it is bound to haemoglobin and other components. As a consequence, lead is usually measured in whole blood rather than in serum. The largest proportion of absorbed lead is incorporated into the skeleton, which contains >90% of the body's total lead burden. The half-life of lead in blood is about 25 days, in soft tissues about 40 days and in the nonlabile portion of bone >25 years. Thus, blood lead levels may

Table 34.2 Various Preparation of Lead

Compound	Uses
Lead acetate (sugar of lead)	Previously used in medicine as an astringent and local sedative for sprains
Lead carbonate (white lead)	Manufacture of paints
Lead tetraoxide (red lead or vermilion)	Used as *sindoor* in the parting of scalp hair by married Hindu women
Lead sulphide (*surma*)	Collyrium for the eyes, used mainly by Muslims
Lead oxide (litharge)	Glazing of pottery and enamel ware
Lead oleate (diachylon)	Plaster, abortifacient
Tetra-ethyl lead	Anti-knock for petrol (any substance that is used to prevent detonation in internal combustion engines is called 'antiknock')

decline significantly, while the body's total burden of lead remains heavy.

Lead is excreted mainly in the urine and in the faeces. Lead also appears in hair, nails, sweat, saliva and breast milk. As the body accumulates lead over many years and releases it into the urine only slowly, even small doses can at times lead to intoxication. The total body burden of lead correlates better with the risk of toxicity than the blood concentration alone.

Mechanism of Action

- The toxicity of lead is probably related to its affinity for cell membranes and mitochondria, as a result of which it interferes with mitochondrial oxidative phosphorylation and sodium, potassium and calcium ATPases.
- Lead depresses the enzymes responsible for haem synthesis and shortens erythrocyte lifespan leading to microcytic or normocytic hypochromic anaemia.
- Lead blocks the conversion of δ-aminolaevulinic acid to porphobilinogen by blocking the enzyme aminolaevulinic acid dehydrase. This leads to an increase in δ-aminolaevulinic acid in blood and urine.
- Lead also inhibits ferrochelatase, which results in elevated free erythrocyte protoporphyrin (FEP) levels.

Acute Poisoning

It is very rare. The only common soluble salt is the acetate and this is dangerous only in large doses. Symptoms are similar to those of acute arsenical poisoning, except that diarrhoea is replaced by constipation and the stool is blackened and offensive. The usual symptoms may include metallic astringent taste in the mouth, vomiting associated with colic-like pains in abdomen. There may be purging, more usually constipation. The faeces are offensive and black due to formation of lead sulphide and the urine is suppressed.

When the case is protracted, the patient may suffer from cramps in legs and arthralgia. Collapse precedes death.

Fatal Dose

The average lethal dose is said to be 10 gm/70 kg for most lead salts, while it is l00 mg/kg for tetra-ethyl lead. (However, acute poisoning is very rare.)

Fatal Period

In severe cases, death may occur within 24 hours. Ordinarily, fatal period may extend to 2–3 days.

Management

- Emesis may be tried to remove the unabsorbed lead from the stomach.
- Gastric lavage with magnesium or sodium sulphate is advised. These form highly insoluble lead sulphate preventing its absorption. For the same reason, for removal of the poison from the intestine, sodium or magnesium salt may be given in purgative dose.
- Calcium gluconate 2 gm IV usually relieves abdominal colic and also helps deposition of some lead in bones.
- For elimination of absorbed part of lead, $CaNa_2EDTA$ is given by slow IV infusion in a dose of 30–40 mg per kg body weight, twice on the first day. If therapy is tolerated, it is repeated in the same dose for next 4 days. If necessary, the same regimen may be followed after a gap of 1 week.
- DMSA (succimer 30 mg/kg body weight) is an effective oral chelator and may be given to outpatients.
- Some prefer combined BAL and EDTA. This is because of the fact that on the first day of EDTA therapy, more lead may be mobilised from the bone than is excreted, which may lead to a worsening of the situation. BAL can cross into cells and protect against these increases of central compartment lead. Also, combined therapy produces a greater excretion of lead.
- Penicillamine (0.5–1.5 gm daily) in an adult is an alternative oral chelating agent in those countries where DMSA is not available.

Chronic Poisoning (Plumbism, Saturnism or Saturnine Poisoning)

Chronic lead poisoning may occur in the industrial environment due to inhalation of lead dust or lead vapour arising from burning of paints, battery, smouldering, glass blowing and polishing, enamel factories, dye, cosmetic, etc. It may also occur due to low dose consumption from drinking water supplied through lead pipes, food preserved in tin containers having lead lining, food contaminated with lead in the course of preservation, etc. Poisoning may occur due to prolonged use of vermilion and cosmetics containing lead. Absorption of tetra-ethyl lead through skin is common in people who handle petrol or gasoline.

Chronic poisoning may also result from the mobilisation of lead already stored in the body. As long as the reaction of blood is normal, the stored lead is slowly eliminated from the bones. However, when the reaction changes (for example, in acidosis) there may be sudden mobilisation of stored lead, and symptoms of acute or chronic poisoning may develop.

Clinical Features

In the early phase, chronic lead poisoning is manifested by facial pallor, anaemia, basophilic stippling of red cells, blue line in the gum, retinal stippling and in the later phase by colic, constipation, palsy, encephalopathy, disturbance of genito-urinary and cardiovascular system.

Facial Pallor This is understood to be due to vasospasm, mostly due to contraction of capillaries, particularly about the mouth and is one of the earliest signs.

Anaemia with Punctate Basophilia It is due to impairment in the synthesis of haeme from protoporphyrin and of porphobilinogen from δ-aminolaevulinic acid. Another cause of anaemia may be increased fragility of the RBCs due to loss of intracellular potassium because of increased permeability of the cell membrane. The **basophilic stippling** of the red cells is due to condensation of iron containing ribonucleic acid near the mitochondria. These are stained with basic dyes and hence the name.

Lead line or Burtonian line is a bluish-black discolouration due to subepithelial deposition of lead sulphide granules on the gums at the junction with the teeth. It is seen in not more than 50% of cases, in whom dental hygiene is poor. Its colour is due to the action of hydrogen sulphide liberated by microorganisms from decomposing food material around carious teeth in the presence of circulating lead.

Retinal stippling is noticed by ophthalmoscopic examination showing presence of greyish glistening lead particles, in the early phase of chronic lead poisoning.

Colic and Constipation Colic is often the first symptom that arouses suspicion of plumbism. It generally affects intestines, ureters, uterus and blood vessels. It is spasmodic, intermittent and relieved by pressure. Obstinate constipation (also known as dry belly ache) is associated with it.

Lead palsy is rather a late and uncommon phenomenon. Only about 10% of patients usually suffer from this condition. Lead induced peripheral demyelination is reflected by prolonged nerve conduction time and subsequent paralysis, usually of extensor muscles of the hands and feet **(wristdrop and footdrop)**. It is said to be due to interference with phosphocreatinine metabolism at the muscular level.

Lead Encephalopathy It is said to be seen almost in all the cases of plumbism but is more common in poisoning by tetra-ethyl lead. It is frequently encountered in children. This may be due to inactivation of monoamine oxidase due to combination of lead with the SH radical of the enzyme. There may occur changes in personality, restlessness, fatigability and mental dullness. In some cases, there may be acute conditions like convulsion, delirium and coma. **Lead osteopathy:** In children, lead is deposited beyond the epiphysis of the growing long bones. The deposition is promoted by calcium and vitamin D and is detectable by radiological examination in the form of increased density at the metaphyseal plate of growing long bones.

Effects on Reproductive System Chronic exposure may cause sterility in both male and female patients. In males, there may be loss of libido. In females, there may be menstrual irregularities; in carrying women, there may be abortion due to chronic atrophy or spasmodic contraction of the uterus. Even a healthy woman, if impregnated by a man suffering from chronic lead poisoning, is likely to abort, due to blastophoric influence of the disease upon the spermatozoa.

Effects on Circulatory System There may be degenerative changes in the arteries with resultant hypertension and hypertensive cardiopathy.

Diagnosis

The decision to use chelation therapy is based not only on blood lead concentration but also on the symptoms present and if available, an estimate of the total body burden of lead using X-ray fluorescence.

- **Blood investigations**
  - Normal range of level of lead is 0–50 μg/dl. Chelation is recommended for the treatment of all children whose blood lead levels are >55 μg/dl. Chelation is recommended for adults, if blood lead levels exceed 80 mg/dl or if these levels exceed 60 mg/dl and symptoms have developed.
  - Peripheral blood picture may show anaemia and basophilic stippling.
  - Most of the patients may show fluorescence of RBCs when examined under ultraviolet light. This is probably due to increased FEPP (free erythrocyte protoporphyrin).
- **Urine investigations**
  - In chronic poisoning, there may be irregular excretion of lead in urine. Between 80 and 100 mg/L should be considered a borderline case. Above 100 mg/L, care must be taken for avoiding further exposure and preferably treatment should be administered. [The concentration of aminolaevulinic acid (ALA) in the urine is widely used as a measure of lead toxicity in workers who are exposed occupationally.]
  - Calcium EDTA mobilisation test: $CaNa_2EDTA$ is administered at a dose of 500 mg/m^2 in 5% dextrose infused over an hour. Urine volume over the next 8 hours should be measured for lead. The total urinary excretion of lead (mg) is divided by the amount of $CaNa_2EDTA$ given (mg) to obtain the lead-excretion ratio. An 8 hour $CaNa_2EDTA$ chelation provocative test is considered positive if the lead excretion ratio is more than 0.6.
  - Porphyrin excretion may be more than 500 mg/day in the urine.
- **X-ray examination**
  - An abdominal X-ray can demonstrate flecks of lead paint.
  - An X-ray of the long bones may show 'lead lines' that are bands of increased density and can occur within a matter of days following ingestion.

Management

- Precautionary steps to prevent toxicity in the workers of lead industries are must.

- Regular medical check-up of the workers and appropriate steps to be taken when they develop minor signs or when the amounts of lead in the urine and blood approach threshold levels.
- Chelating agents need to be administered depending upon the indications as written earlier.
- In addition, the patients' general condition should be looked after throughout the treatment with suitable measures.

Medicolegal Aspects

- Historical record shows that lead water pipes and food containers were popularly used by elites of the ancient Roman Empire, which became one of the causes for the disintegration and destruction of the Roman culture. Members of the upper class of society suffered from various physical and mental disabilities and their women, in addition, suffered from sterility due to chronic lead toxicity.
- Children often have a craving for eating inedible things such as mica and often lick lead-based flakes of lead paints or crumbling plaster or chew tooth-paste tubes and hence are more susceptible to lead poisoning than adults. (The condition **pica** refers to an abnormal craving for nonnutritive substances.)
- Infants may suffer from slow and progressive lead poisoning by imbibing lead secreted in the milk of the mother who is poisoned by face powders, skin cosmetics and hair dyes containing lead.
- Suicidal and homicidal uses are very rare. Suicides do not prefer it due to long painful sufferings before death. Homicides do not use it due to chance of detection from the taste of the poison as well as due to high fatal dose.
- Workmen who contract disease during the course of and by reason of their employment are entitled to compensation from their employer during such time as they are incapacitated from earning their livelihood and in the event of death, their dependants are entitled to such compensation.

Six persons who had worked for periods varying from 3 months to 2 years for long shifts in an enclosed space filled with a heavy concentration of fumes from petrol were reported suffering from chronic lead poisoning. The main symptoms pertained to the CNS, probably owing to addition of lead tetraethyl in the petrol. Of these, two died.

- A rare form of chronic poisoning may occur through absorption of lead from an unremoved bullet lodged somewhere in the body.
- Lead may be used criminally as an abortifacient. It acts by producing tonic contractions of the uterus and causing degeneration of the embryonic cells and the chorional epithelium. The paste used for anointing 'abortion stick' often contains red lead as the chief ingredient.
- Lead is normally present in almost all human tissues. The amount present in individual cases varies according to the difference in the lead content of food ingested. Lead is retained in the bones, teeth, hair and nails. The maximum amount is usually found in hair, especially the black hair of Indian women. Testicle contains fairly appreciable amount.

MERCURY (*PARA*, QUICKSILVER)

Mercury is the only metal that is liquid at room temperature. It exists in three forms—metallic (Hg^0), mercurous (Hg_2^{2+}) and mercuric (Hg^{2+}). Metallic mercury, also known as quicksilver, is a liquid metal having a bright silvery lustre. It exists in nature as the metal itself and as the sulphide (cinnabar or *ras sindoor*). Metallic mercury is not poisonous if taken by mouth because it is not absorbed. It vaporises even at room temperature to an extent sufficient to permit the inhalation to toxic amounts. Mercury depresses cellular enzymatic mechanisms by combining with sulphydryl groups.

Poisonous Compounds of Mercury and their Sources

- Mercuric chloride or **corrosive sublimate** is available as colourless prismatic crystals or as crystalline powder. It is odourless but has a burning metallic taste. It is used in medicine, in laboratories, as preservative and in industries. It is by far the most common cause of acute poisoning.
- Mercurous chloride or **calomel:** This is known as subchloride of mercury. It is heavy, amorphous, white and tasteless powder. It is insoluble in water and its insolubility is the greatest bar to its toxicity. It is used as purgative as it is nontoxic for human consumption in therapeutic dose.
- Other compounds include mercuric oxide or red precipitate (sipichand), mercuric ammonium chloride or white precipitate, mercuric potassium iodide, mercuric nitrate and mercuric cyanide.
- **Organic compounds of mercury:** Medicinal preparations of organic compounds of mercury were used as diuretics in the past. The volatile diethyl and dimethyl mercury compounds have proved valuable as fungicides. Mercury is methylated under sea water and certain sea fishes, particularly sword fish, are rich in methylmercury. Prolonged and excessive intake of these may cause chronic mercurial poisoning.

Absorption, Fate and Excretion

Mercuric chloride and some other mercurial salts, being soluble in gastric juice, are readily absorbed through GIT. Vapour of mercury and soluble mercury salts are also well-absorbed through the respiratory tract, through vaginal tract (douche) and urinary bladder (mercurial antiseptic washing agent). Skin ointment of mercury, when used for a long period, may cause chronic poisoning.

After absorption, mercury gets deposited in all tissues of the body, particularly in liver, kidneys, spleen and bones. When absorbed by way of inhalation, maximum concentration

occurs in brain tissue. In toxic deaths, the concentration in liver may go as high as 1 mg per 100 gm and the concentration in kidneys up to 2 mg per 100 gm.

Organic mercurial compounds pass placental barrier and foetus may have more concentration of methylmercury than the mother.

Mercury is mainly excreted through the kidneys, liver (bile) and large intestines.

Fatal Dose

Mercuric chloride: 0.5–1 gm/70 kg. Mercurous chloride: 1.5–2 gm/70 kg.

Fatal Period

Death may occur within a few hours but is usually delayed for 3–5 days.

Acute Poisoning

Poisoning may occur through **inhalation** while heating metal in a closed room, or following gold refining in an enclosed area. Symptoms include cough, dyspnoea, fever, headache, chills (metal fume fever), gastrointestinal disturbances including metallic taste and blurring of vision. In severe cases, there may be noncardiogenic pulmonary oedema, convulsions, etc.

The symptoms frequently assert themselves soon after the poison is **swallowed/ingested**. The taste is strikingly metallic, with a feeling of constriction in the throat, burning sensation from mouth to stomach and pain radiating over the abdomen. The mouth, tongue and fauces usually become corroded and the mucous membrane appears greyish white. Nausea with frequent vomiting is seen. The vomitus contains mucus and altered blood, and shreds of gastric mucosa. It may be followed by profuse purging, often bloody with painful tenesmus and with presence of necrosed mucus shreds from the colon. With development of renal damage, there occurs oliguria, albuminuria and haematuria. Subsequently, more generalised symptoms appear and collapse sets in, with cold clammy skin, pale anxious face, sunken eyes, rapid feeble pulse and sighing respirations. Convulsions and general insensibility usually precede death.

Subcutaneous or intramuscular injections of elemental mercury may cause abscess formation with ulceration, exuding tiny droplets of mercury. Intravenous injection can result in **mercurialism**, characterised by thrombophlebitis, granuloma formation, pulmonary embolism, etc. (Such an activity may be a deliberate suicidal gesture or the result of a misconception that such injections can enhance athletic or sexual performance. In fact the term 'quicksilver' has its origin in the false belief that mercury can quicken a boxer's punches.)

Diagnosis

- Mercury is not a constituent of human body. Its determination in tissues proves that it must have been introduced from outside. The amount of mercury entering the body through food stuffs varies from 5 to 20 μg per day. This is balanced by the daily excretion in faeces and urine.
- Levels of mercury in blood and urine should not exceed 3.6 μg/dl and 15 μg/L, respectively. Symptoms may develop when blood and urine mercury levels exceed 20 μg/dl and 60 μg/L, respectively. Levels in the hair may be used as a dosimeter for chronic organic mercury exposure.

Management

- If vomiting has not occurred, emetics should be given.
- Gastric wash may be carried out with 250 ml of 5% solution of sodium formaldehyde sulphoxylate. A further of 100 ml may be left in the stomach after lavage. This substance reduces the perchloride to a less soluble (less toxic) mercurous compound.
- Egg albumin, which forms an insoluble albuminate of mercury, is advocated.
- 100 ml of 5% sodium sulphate solution may be given intravenously to help anuria. Sodium bicarbonate may be given orally to combat acidosis.
- For the absorbed part of the poison, effective chelating agents are dimercaprol, succimer and penicillamine. Dimercaprol is given deep intramuscularly at a dose of 100 mg, every four hourly for 48 hours. Therapy is usually given in 5-day courses separated by several days of rest. [DMPS (2,3-dimercapto propane-1-sulphonate) or DMSA (succimer) meso-2,3-dimercapto succinic acid may be the effective alternative choice]. The N-acetyl form of penicillamine is also useful in a dose of 30 mg/kg per day in divided doses.
- Peritoneal or haemodialysis helps excretion of absorbed poison quite effectively in acute cases.

Postmortem Appearances

Appearances of corrosive poisoning will be present if the poison is taken in a concentrated form. Otherwise, the signs of irritant poisoning will be evident. The appearances are most marked in the stomach and bowel. **Externally,** the body may look emaciated due to loss of body fluid by way of vomiting and purging. **Internally,** mucous membrane of mouth including tongue appears necrotic. These areas generally have a diffuse greyish white escharotic appearance. The mucous membrane of oesophagus is usually corrugated and corroded. The stomach is swollen with evidence of desquamation, haemorrhage and ulceration. Necrosis of the mucous membrane of intestine with ulceration at places is quite common. Mercury appears to have a selective action on the caecum and large intestine, which often show intense inflammation, ulceration, etc. Kidneys almost always show toxic nephritis. Liver and heart may show fatty degeneration. The latter may also show subendocardial haemorrhages.

Chronic Poisoning (Hydrargyrism)

This may occur due to (*i*) after effects of acute attack, (*ii*) injudicious medical use and (*iii*) continuous accidental absorption in individuals working with the metal or a salt as in the manufacture of thermometer, barometers, fur felt and ultraviolet apparatus or in police personnel engaged in finger print detection work where the powder contains mercury.

Clinical Features

Excessive salivation with metallic taste in mouth with painful inflamed gums and occasionally a blue-black line on the gums as with lead poisoning. Nonspecific early symptoms may include anorexia, insomnia, abnormal sweating, headache and lassitude. **Mercuria lentis** may be the early symptom of chronic poisoning exhibiting in the form of discolouration of the anterior capsule of the lens of the eye due to deposition of mercury, as observed through the slit lamp.

Neurological toxicity is manifested by tremors. It is one of the most characteristic and consistent manifestations of chronic mercury poisoning and is sometimes referred to as **Danbury tremor**. It is coarse, intentional and affects the hands, arms, tongue and later the legs. The advanced condition is referred to as **Hatter's shake** (because it was common in workers of that industry). The tremor can be detected early in the writing of the patient, from his stammering speech or from his daily activities involving some delicacy of movements like shaving, holding a tumbler or spoon, etc. The most severe form of the condition is referred to as **concussio mercurialis** when literally no activity is possible. Tremor may persist even years after exposure to mercury has ceased. **Mercurial erethism** is a peculiar disturbance of the personality and comprises of constellation of findings including excitability, memory loss, insomnia, timidity and sometimes delirium that was described in workers with occupational exposure in the felt-hat industry hence the expression **'mad as a hatter'**. (It has been suggested that Lewis Carroll's Mad Hatter may really have been suffering from mercury poisoning, as did many hatters of the 1800s.)

Management consists of removal of patient from exposure and promoting elimination of mercury by bowels and kidneys. Dry extract of belladonna may be given to relieve the excessive salivation. Chronic inorganic mercury poisoning is best treated with N-acetyl penicillamine. Other symptoms may be treated as they arise.

Medicolegal Aspects

- Occupational exposure to inorganic mercury has occurred in mercury mines, chloroalkali plants, thermometer factories and in health service maintenance workers responsible for repairing broken sphygmomanometers.
- Nonoccupational exposure to mercury occurs principally from dietary intake and to a minor extent from dental amalgam. Many foodstuffs contain small amounts of inorganic mercury but organic mercury compounds bioaccumulate in the aquatic food chain so that certain fishes (e.g. trout, pike and tuna, etc.) contain significant amounts of methylmercury.
- Accidental poisoning may occur in children from (*i*) the use of ammoniated mercury in some bleaching creams and (*ii*) swallowing the sulphocyanide of mercury, the main constituent of **Pharoah's serpents**. It is mainly sold in the festive season, especially Diwali, for entertainment of children. When ignited, it gives out pungent smell and ash in the form of a long tortuous figure resembling a snake, hence the name.
- Mercurous compounds are less soluble, less corrosive and less toxic than mercuric salts. Ingestion of mercurous chloride in teething powder has led to **acrodynia** (Pink disease/Swift disease) in infants. This condition presents as fever with a pink-coloured rash, irritability, photophobia, painful and swollen extremities and hypersecretion of sweat glands. It is believed to be a hypersensitivity response to the mercurous chloride.
- **Fish contamination:** High in protein, low in calories, fat and cholesterol, and widely suspected to fight against heart disease and cancer—this highly praised health food can also be a site of contamination and spoilage. The fish readily soak up poisons and contaminants in water, concentrating heavy metals (such as methylmercury) in their organs. These fishes are eaten by larger fish, further concentrating the toxins. The early cases of contaminated fish poisoning occurred in Japan in 1956, called as **Minamata Bay disaster**. This resulted from methylmercury poisoning when mercury in a factory effluent was discharged into the bay. The mercury was concentrated up the food chain and affected members of the community living around the bay. Unlike inorganic mercury compounds, methylmercury is hard to detect in the blood. It does not readily break down in the body and can take months to be excreted. In addition, it can pass easily through the blood-brain barrier and can also cross the placenta and build up in the foetal brain and blood. Both DMSA and DMPS appear to be of value in the treatment of methylmercury poisoning.

THALLIUM

Thallium is a soft white heavy metal with a lustrous colour, which tarnishes on exposure to air owing to the formation of thallous oxide. The metal and its compounds are highly toxic. From the medicolegal point of view, important salts are thallium acetate and thallium sulphate. The salts are soluble, colourless and nearly tasteless, and therefore, can easily be administered by mouth in food or drink. Thallium acetate was used in the past as a depilatory and in the treatment of ringworm; thallium sulphate is used as a rodenticide.

Mechanism of Action

The exact mechanism of toxicity is unknown but may involve disruption of sulphydryl groups on the mitochondrial membrane

Table 34.3 Fatal Thallium Concentration in Various Organs and Tissues of Body

Specimen	Range	Average
Blood	0.5–11	4.0
Urine	1.7–11	5.2
Kidney	6–20	11.0
Liver	5–29	15.0

and interference with the function of sodium-potassium ATPase for which thallium has 10 times more affinity than that of potassium. In addition, it has been suggested that thallium may interfere with riboflavin homeostasis leading to dermatitis, alopecia and neuropathy. It has further been suggested that thallium is capable of breaking down all cells in the body, especially hair follicles and the central nervous system.

Thallium ion has a similar charge and ionic radius as the potassium ion and therefore, some of its toxic effects may result from interference with the biological functions of potassium (atomic weight 204; periodic table group IIIA; valence, +1 or +3; discovered in 1861). Acute cardiovascular effects of thallium probably result from competition with potassium for membrane transport systems, inhibition of mitochondrial oxidative phosphorylation, and disruption of protein synthesis. Heart is first stimulated followed by depression leading to death by cardiac failure. It also alters haeme metabolism.

It is absorbed through the gastrointestinal tract and skin. The half-life in humans has been reported to be in the range of 1–30 days and may be dose-dependent. It undergoes enterohepatic circulation (Prussian blue being used in the treatment of its poisoning is given orally to break the enterohepatic cycle by trapping thallium secreted in the bile and carrying it into the faeces). High concentrations are found in the kidney and liver (see Table 34.3). Blood and urine thallium concentrations above 100 µg/dl and 200 µg/L, respectively, are toxic.

Acute Poisoning

Symptoms and signs usually appear within a few hours of ingestion in the form of nausea, vomiting and abdominal pain. Less commonly, gastrointestinal bleeding occurs, which may be severe. In the first week, there is often a sudden onset of acne, which may be severe and complicated by necrotic lesions. Sweat glands and sebaceous glands are destroyed and skin becomes dry and scaly. Nail growth is impaired with the development of ridges, usually known as Mees lines [Mees lines/stripes need to be differentiated from (*i*) Beau lines—white lines across the nails because of trauma, skin disease, or hypercalcaemia; (*ii*) Muercke lines—white bands in the nail bed associated with hypoalbuminaemia; and (*iii*) Terry nails—white colouration of nails, except for distal portion (which may be pinkish), seen in cirrhosis.]

Characteristically, hair loss is one of the clinical signs that arouses suspicion of thallium poisoning. This effect begins about a week after the administration, but may not be noticeable for twice that period. Large tufts tend to come away, rather than a general thinning. (Medial parts of the eyebrows are usually spared, for some as yet unknown reason). The presence of alopecia, skin rash, and painful peripheral neuropathy with mental confusion are considered as pathognomonic for thallium poisoning (the so-called **thallium triad**). Later, there may be cardiac manifestations possibly due either to vagal denervation or increased secretion of catecholamines secondary to the effect of thallium on chromaffin cellular ATP.

Fatal Dose

Usual fatal dose is about 1 gm.

Fatal Period

Death may occur in 2 days to 2 weeks.

Chronic Poisoning

Following repeated small exposures, patient insidiously develops a distal neuropathy initially with sensory and then motor loss, which spreads proximally. Cranial nerves may become involved and respiratory paralysis is recognised.

Management

It is possible to sequester thallium ions and prevent reabsorption by the oral administration of colloidally soluble Prussian (Berlin) blue [potassium ferrihexacyanoferrate(II)], 250–300 mg/kg/day. Thallium ions are exchanged for potassium ions in the lattice of the Prussian blue molecule and are subsequently excreted in faeces. Faecal excretion of thallium is detected even when urine excretion of the metal has ceased and, therefore, administration of Prussian blue should be continued until thallium can no longer be detected in the faeces. Thallotoxicosis often causes intestinal stasis and severe constipation, and hence, 50 ml mannitol (15%) needs to be administered along with Prussian blue. Gastric wash with 1% potassium iodide and activated charcoal and a saline purgative to increase faecal excretion are also advocated.

Medicolegal Aspects

Thallium salts possess many properties of an ideal homicidal poison, viz., they are odourless, tasteless, freely soluble in water, and produce toxic manifestations resembling natural disease. The commonest salt is thallus sulphate, which can be mixed with tea, coffee or juice, etc., and can be administered without

arousing suspicion. Relatively long latent period before symptoms begin to manifest (12–48 hours) gives the culprit time to cover his/her designs. The first case of homicidal poisoning was reported in 1927 when a series of homicides were committed by using thallus sulphate meant for killing rats (Zelio paste). It was mixed with butter, spread on bread slices and administered to victims, of whom 7 died. Graham Young case, which occurred in Britain in 1971, is informative. In this case, two victims died and others narrowly avoided death. The case was forensically unique in the sense that it was probably the first murder wherein the poison was detected posthumously in the ashes of a cremated victim. It is also unusual in being detectable radiologically in the intestine and liver.

Accidental intoxication may result from its therapeutic use as a depilatory. Accidental poisoning may also occur as a result when cocaine mixed with thallium is used. Atmospheric pollution may occur as a result of coal-burning power plants and from smelting copper, lead or zinc. Use of 1% thallium sulphate to control ground squirrels has been reported to result in outbreaks of thallotoxicosis in humans. Because of its high cumulative toxicity, the use of thallium has been restricted with a resultant marked decline in its use as a rodenticide.

Autopsy appearances are nonspecific. Pallor and streaking of a pale, degenerating myocardium have been reported. Fatal thallium concentrations (mg/L or mg/kg) reported by Baselt (2002) are being furnished in Table 34.3.

35 CHAPTER

Irritants of Plant Origin

After going through this chapter, the reader will be able to describe: Features, mechanism of action, fatal dose, fatal period and medicolegal aspects of important plant irritants

They act as irritants, and their action is due to an active principle contained in them. **On external application**, inflammation, vesication, pustulation or even ulceration are produced. **On internal administration**, symptoms and signs of gastrointestinal irritation are produced. Therefore, the clinical features will be corresponding to these actions, and the postmortem appearances will be in accordance with the clinical picture. Management is advocated on general principles.

Ricinus communis (Castor Oil Plant, *Arandi*)

It grows all over India. Its seeds are oval, of a glossy brown colour and mottled. They are available in two sizes, the big and the small; the small ones resembling seeds of *Croton tiglium* in size and shape. They contain an active principle called ricin, a **toxalbumin**. (A toxalbumin or phytotoxin is a toxic protein and resembles a bacterial toxin in its action. It causes agglutination and lysis of red cells. It is antigenic in nature and is capable of producing antibody when administered into the body. Other toxalbumins are crotin and abrin.)

Entire plant is poisonous. The active principle is highest in the seeds. They are not poisonous when swallowed entire or after cooking. (Seeds must be chewed before the toxin is released. If the castor beans are swallowed whole, systemic toxicity is unlikely because the hard coat of the seed prevents absorption and, therefore, inhibits poisoning.) They are poisonous when eaten in the raw state. The residue (**press cake**) left after extraction of the oil from the seeds contains ricin and is poisonous to rats, cattle and human beings. The extracted oil (**castor oil**) does not contain ricin and is therefore not poisonous. However, it may cause purging in large doses.

SYMPTOMS AND SIGNS

Usually there is a latent period varying from 2 to 24 hours between the time of ingestion and the onset of toxic features. The early features are burning pain in mouth, throat and abdomen; nausea; severe vomiting and diarrhoea with or without blood. Fluid loss may lead to hypotension and shock. Dehydration and cramps are common. Consciousness is usually retained till the end. The late features include seizures, intravascular haemolysis and uraemia.

FATAL DOSE AND FATAL PERIOD

The unbroken and unmasticated seeds are nonpoisonous as outlined above. When eaten in raw state or when chewed, a single seed may produce alarming symptoms. The fatal dose is usually 6 mg of ricin, which is equivalent to about 10 seeds. Fatal period may extend from 2 to several days.

MEDICOLEGAL ASPECTS

Accidental cases may occur among children who eat the seeds out of curiosity or due to mistake. Workmen who come in contact with the toxin while separating the castor oil from the seeds may be poisoned. The powder of the seeds causes local irritation of the skin and mucous membrane of the nose and eyes. Ricin is more toxic when given by injection than when taken orally because it is destroyed by the digestive ferments.

The case of Georgi Markov (Bulgarian dissident Georgi Markov was the second to be attacked by agents of the Bulgarian Government): Georgi Markov was working as a broadcaster for BBC World Service in London. On 7th September, 1978, while standing on the Waterloo Bridge, he was waiting for a bus for going home. Suddenly, he turned surprised when he received a sharp jab in his right thigh from behind. This had occurred due to dropping of furled umbrella by a man standing behind Mr. Georgi. The tip of umbrella had apparently accidentally poked into Markov's thigh. The man apologised and moved away. Puzzled, Georgi caught his bus home. Late that night he developed fever, vomiting and was rushed to hospital the next morning. The puncture wound in thigh had become inflamed and painful by then. He was diagnosed as a case of septicaemia. However, over the weekend, Georgi passed into coma and died.

Considering the circumstances of death, an autopsy was carried out, which revealed presence of a tiny pellet (the size of a pinhead) in the thigh. Examination of the pellet through scanning electron microscopy at Metropolitan Police Forensic Laboratory revealed that it was made up of an alloy of platinum and iridium, carrying two small holes bored through it. Chemical analysis of the pellet did not reveal the presence of any poison. Identifying the poison became a process of elimination. Considering the minute size of the dose and its catastrophic effects, it was believed that the pellet must have been charged with **ricin**, a potential chemical warfare agent. The theory was tested by injecting a pig with a quantity of ricin similar to the quantity that could have been contained in the pellet. The animal died within 24 hours, and its organs showed damage similar to that found at Markov's autopsy. (Following a change of regime in Bulgaria in 1991, the new government admitted that assassination attempts had been made on a number of former citizens living in the West, including Markov and Rostov.)

Croton tiglium (Jamal Gota or Nepala)

This plant grows all over India. The seeds and the oil (croton oil) extracted from the seeds are poisonous. The seeds resemble small castor seeds in size and shape but differ in their appearance. They are oval, blackish-brown in colour with a white oily kernel and have longitudinal lines on them, while castor oil seeds are of a glossy brown colour and mottled.

The active principle is crotin, a **toxalbumin**, which is similar to ricin and possesses irritant and vesicant properties. The oil contains a powerful vesicating resin composed of crotonoleic acid, methyl crotonic acid and several other fatty acids.

SYMPTOMS AND SIGNS

These are similar to poisoning by ricin. The oil causes blistering externally and on ingestion causes severe gastrointestinal irritation with burning pain in the abdomen, salivation, vomiting, powerful purging, etc. In substantial doses, collapse precedes death.

FATAL DOSE AND FATAL PERIOD

Four seeds have produced death, and twenty drops of the oil have proved fatal to adults. Fatal period may be from a few hours to a few days.

MEDICOLEGAL ASPECTS

Croton oil has been used as an abortifacient. Suicide and homicide are rare. Accidental poisoning has occurred from the use of croton oil as a purgative or when the oil has been swallowed by mistake. It may also occur by chewing seeds or inhaling their dust. Soft parts of *Croton tiglium* have been used as an arrow poison. **Other arrow poisons** include abrin, *Calotropis*, aconite, strychnine and curare, etc.

Abrus precatorius (Indian Liquorice/ Jequirity/*Gunchi*/*Rati*)

The plant is found all over India. Though all parts of the plant are poisonous, the seeds are commonly used as poison. They are of the size of a small pea and have an average weight of 120 mg. They are egg-shaped, having bright scarlet colour with a large black spot at one pole. Seeds may be white with black spot, all black, yellow or blue. The seeds contain an active principle abrin, **a thermolabile toxalbumin** and its actions resemble those of viperine snake bite with intense local symptoms and haemorrhages followed by general symptoms. In addition to this, seeds contain poisonous proteins, fat-splitting enzymes, abrussic acid, haemagglutinin and a quantity of urease.

SYMPTOMS AND SIGNS

When the seeds are swallowed as such, they are not poisonous because the outer covering is so hard that it escapes disintegration in the gut. If chewed before swallowing, they cause toxicity. They are also not poisonous after cooking because the abrin (the active principle) loses its toxicity on boiling. If the extract is injected under the skin or into a wound, death may be produced depending upon the amount administered. Symptoms may be delayed from a few hours to 2–3 days when taken by mouth. They include abdominal pain, nausea, vomiting, diarrhoea followed by a circulatory collapse.

When the extract of the seeds is injected under the skin of an animal, inflammation, oedema, oozing of haemorrhagic fluid from the site of puncture and sometimes necrosis occurs surrounding the site of injection. The animal does not take food and drops down after 3–4 days and cannot move, becomes comatose and dies. Convulsions may precede death. These manifestations resemble those of viper snake bite, for which they may be mistaken. **In human**, painful swelling and ecchymosis develops with inflammation and necrosis at the site of injection. There is faintness, vertigo, vomiting and general prostration. Convulsions may precede death from cardiac failure.

FATAL DOSE AND FATAL PERIOD

The fatal dose is 1–2 seeds (90–120 mg by injection). Subcutaneously, abrin is manifold as toxic as by the oral route. Fatal period may extend from 3 to 5 days.

MEDICOLEGAL ASPECTS

The powdered seeds in the form of *sui* (needle) are employed as cattle poison by leather workers in order to produce cheap

hides or for revenge. For this purpose, the seeds are decorticated, and alone or mixed with *dhatura*, opium or onion are made into a paste with spirit and water. Small sharp-pointed spikes or needles, the so-called ***'suis'*** are prepared, which are then dried in the sun. Two needles are inserted by their base into holes in a wooden handle or bamboo stick. A blow is struck to the animal with great force, which drives the needle into the flesh. For homicidal purposes, the needle may be kept between two fingers and the person is slapped which drives the needle into the body. Powdered seeds may be used by malingerers to produce conjunctivitis. The seeds are sometimes used as an abortifacient and as arrow poison. (Abrus means graceful, which here refers to flowers, while 'precatorius' is derived from 'precor' which means 'to pray'. Seeds are often used as beads in rosaries.)

Colocynth (Bitter Apple/*Indrayani*)

Colocynth is obtained from the dried pulp of the fruit of *Citrullus colocynthis* or bitter apple. The plant grows widely throughout India. The pulp freed from the seeds is called colocynth and occurs as white spongy, light fragments having an intensely bitter taste. The active principle is a resinous glycoside, **colocynthin**, which is a proved irritant of the gastrointestinal tract. (**Glycosides** are substances found in plants and are composed of a sugar and a non-sugar compound, the latter having a toxic action.)

SYMPTOMS AND SIGNS

Nausea, yellow vomiting, yellow coloured watery stools and abdominal pain are often present. Stools may be blood stained. Occasionally, death may occur.

FATAL DOSE AND FATAL PERIOD

The fatal dose is usually 1–2 gm and the fatal period is usually within 24 hours but may be delayed for a few days.

MEDICOLEGAL ASPECTS

As it causes congestion of pelvic viscera, it may have an indirect effect on the uterus and has accordingly been used to procure abortion.

Capsicum (Chillies/Cayenne Pepper)

Capsicum fruits are powdered and universally employed in India as a condiment, the condiment being known as red pepper or ***lal mirch***. The active principle contained in capsicum is **capsicin**, an exceedingly acrid, volatile, nonalkaloidal substance. Capsicum has a pungent smell and is used in medicine as a stomachic and carminative.

SYMPTOMS AND SIGNS

Applied to the skin, it causes irritation and vesication. When taken internally, it acts as an irritant poison. When applied to the eyes, it causes burning and lacrimation. Because of volatility of the active principle, the fumes arising from burning capsicum are highly irritant.

MEDICOLEGAL ASPECTS

Chillies may be used for purpose of torture to extort money or a confession of some guilt by introducing them into the nostrils, eyes, vagina or urethra. They may be used by burning them under the nose, rubbing them on the breasts of the females and covering the head with a bag that contains chillies. The method of introducing chillies into the rectum for causing torture is sometimes termed as ***Hyderabadi goli*** (it is the colloquial expression denoting the heinous practice of extracting confessions by police people in certain parts of India). Well-powdered chillies may be thrown into the eyes to facilitate robbery. The fumes from burning chillies are quite irritating to the eyes and upper respiratory passage. **Hunan hand:** Severe, burning lesions of fingers in persons handling dried red chilly powder for prolonged periods. It is caused by volatile oils activating dermal pain fibres. Superstitious people may use them to scare away devils and ghosts. **The seeds**, about 0.30 cm long and wide, resemble dhatura seeds and may be differentiated from them as shown in Table 35.1.

Table 35.1 Differentiating Features of Chilli Seeds and Dhatura Seeds

Features	Chilli seeds	Dhatura seeds
Size	Smaller	Bigger
Colour	Yellow	Rather brown
Appearance	Rather round in shape and smooth	Kidney shaped, finely pitted and reticulated
Smell	Pungent	Odourless
Taste	Pungent	Bitter
Border	Convex border with single edge	Convex border with double edge
On section	Embryo embedded in fleshy albumin and curved inwards	Embryo embedded in white oily albumin and curved outwards

Semecarpus anacardium (Marking Nut)

The fruit of this plant is known as *Bhilawan* or the marking nut because its juice is used by washermen as marking ink on clothes. The main active principles contained in fruit juice are semecarpol and bhilawanol.

SYMPTOMS AND SIGNS

Applied to the skin, the juice causes irritation and a painful blister containing acrid serum, which causes an eczematous eruption. Scratching of the affected parts causes similar eczematous eruptions on the tips of the fingers. Apart from itching, the eruptions are usually accompanied by constitutional symptoms such as fever, painful micturition with urine of brown colour. When administered internally, the juice is much less irritant. Larger doses may cause blisters in the mouth and throat, and severe gastroenteritis. In occasional cases, there may be dyspnoea, tachycardia, hypotension, coma and death.

FATAL DOSE AND FATAL PERIOD

The fatal dose is usually 5–10 gm and the fatal period may be 12–24 hours.

MEDICOLEGAL ASPECTS

Accidental poisoning may result from the administration of juice internally by quacks. The juice may be used criminally as an abortifacient by application to the os uteri by means of an abortion stick. It may be applied to the genitals as a punishment for adultery. It may be used by malingerers to produce an **artificial bruise** to support a false charge. Its presence, however, can be detected by chemical analysis of the blister fluid.

Table 35.2 Differentiating Features of Artificial and True Bruise

Features	Artificial bruise	True bruise
Cause	Chemical	Trauma
Situation	Accessible parts	Anywhere
Appearance	Blister formation	No blister formation
Fingers	May show marks due to scratching	Not so
Itching	Present	Not so
Colour changes	Nil	Characteristic
Contents	Acrid serum	Extravasated blood
Analysis	Chemical detectable in the blister fluid	Not so

Other agents that can produce artificial bruise may include madar juice or *Plumbago* root. The juice, like vitriol, has been thrown on face with evil intention. Homicidal poisoning by internal administration of the juice is very rare. Table 35.2 summarises the differentiating features of an artificial bruise and true bruise.

Calotropis (Madar, Akdo)

The plant grows wild almost everywhere in India. Two varieties are there, namely, *Calotropis gigantea* (which has purple flowers) and *Calotropis procera* (which has white flowers). These two plants closely resemble each other and are known by the same vernacular names. They contain **three active principles**, namely, uscharin, calotoxin and calotropin. The leaves and the stem of the plant, when incised or crushed, yield an acrid milky juice. The juice is acidic in reaction, bitter in taste and if heated or allowed to stand, forms a white clot leaving a clear straw coloured serum. The serum contains an active principle gigantin, which is highly toxic, while the clot contains a resin and is less poisonous.

SYMPTOMS AND SIGNS

Milky juice, when applied externally, causes redness and vesication of the skin. When instilled into the eyes, it produces conjunctivitis, which may result in impairment of vision. When taken internally, the juice acts as a gastrointestinal and cerebrospinal poison. It produces an acrid bitter taste, vomiting, burning pain in the throat and stomach, salivation, stomatitis, diarrhoea, dilated pupils, tetanic convulsions and occasionally, collapse and death. Powdered madar root may sometimes be used as snuff.

FATAL DOSE AND FATAL PERIOD

Fatal dose is uncertain. The fatal period may extend to 12 hours.

MEDICOLEGAL ASPECTS

Madar juice has been used (*i*) for infanticide by mixing it with milk or water, (*ii*) for procuring abortion by internal administration and by local application by means of an abortion stick, (*iii*) as a cattle poison by smearing on a cloth and pushing into the rectum of the animal or is given with fodder, (*iv*) as arrow poison and occasionally (*v*) to produce artificial bruise. The root of *Calotropis procera* is highly poisonous to cobras and other poisonous snakes, which cannot even stand its smell. Therefore, snake charmers use it to control the newly caught snakes.

All parts of the plant may be useful, i.e. flowers as digestive stimulants, leaves as external poultice, the powdered root as an emetic and the milky juice as a vesicant, depilatory and for treatment of chronic skin conditions.

A case has been reported in the UP Ch Ex's Annual Report (1964) wherein a 17-year-old unmarried Muslim girl became pregnant because of an illicit intimacy with a boy in the neighbourhood. She was given some abortive drug by her father to save the honour of the family. However, the girl died. Autopsy on the exhumed dead body showed presence of a 3 month old fetus in the uterus and *Calotropis gigentea* in the viscera.

Plumbago rosea (*Lal Chitra*) and *Plumbago zeylanica* (*Chitra*)

The roots and possibly other portions of these plants contain an active principle, **plumbagin**, a highly acrid crystalline glycoside. When the crushed root or twigs are applied externally, redness and vesication occurs. Taken internally in small doses, it acts as a sudorific and stimulates the contraction of the muscular tissue of the heart, intestine and uterus. Taken internally in large doses, it acts as an irritant poison and produces same symptoms as *Calotropis*.

FATAL DOSE AND FATAL PERIOD

Fatal dose is uncertain and fatal period may extend to a few days.

MEDICOLEGAL ASPECTS

Plumbago is mainly used as an abortifacient, either using it by ingestion or by local application directly of the root or twig or the paste by means of an abortion stick to the os uteri. Malingerers have used *Plumbago* to produce artificial bruise. Artificial bruise can also be produced by application of marking nut juice or madar juice.

An interesting case has been reported in Bengal Ch Ex's Annual Report (1938) wherein one Jitan Ali Mir of Murshidabad reported to the police that some 20 men armed with lathis, lanterns and torches had entered his house on previous night and carried away his valuables after having beaten and branded him with torches. Examination showed about 27 trifling injuries on accessible parts of his body. These appeared to have been self-inflicted and caused by the application of Plumbago rosea to the skin. The stains/marks were of reddish-brown colour and without raised or inflamed margins. The hair were unsigned or uninjured. The man was found guilty of bringing a false charge of dacoity.

Claviceps purpurea (Ergot/Mother of Rye)

Ergot is the dried sclerotium (compact mycelium or spawn) of the parasitic fungus *Claviceps purpurea*, which grows on cereals like rye, barley, wheat, oats, etc. (principally on rye and barely in wet seasons and in ill-drained soils.) It gradually invades the interior of the cereal/grain, producing a curved, dark-purple or black compact mass called sclerotium. It contains about thirty alkaloids, but ergotoxin, ergotamine and ergometrine are important. Ergotoxin promotes contraction of uterus in later months of pregnancy. By contracting the arterioles, it eventually causes gangrene. It is the most important factor in ergot poisoning. Ergotamine has similar properties. Ergometrine is an active ecbolic and causes prolonged muscular contractions of the post-parturial uterus. Chronic overdose (ergotism) results either in a neurologic disorder (hallucination in the form of formication, ataxia, convulsions, etc.) or gangrene of extremities due to vasoconstriction. Treatment involves withdrawal of drug, administration of intravenous nitroprusside (in gangrene) for severe ischaemic changes or diazepam 0.1 mg/kg intravenously slowly for convulsions, depending upon the manifestations of poisoning.

FATAL DOSE AND FATAL PERIOD

The fatal dose of ergot may be as low as 1 gm. Death may occur in 24 hours or may be delayed for several days.

MEDICOLEGAL ASPECTS

Acute poisoning is rare, it may occur when preparation of ergot is taken in a large dose to procure abortion. Chronic poisoning results from ingestion of ergot-contaminated bread/cereals for a long time.

In August 1951 (usual days of rainy season), a case of **mass poisoning** occurred in the town of Pont-Saint-Esprit wherein poisoning of 230 people was reported in a single day by consuming bread or cake made from flour contaminated with ergot. All women sufferers aborted and most of them suffered from severe menorrhagia. **Stwart Mckay** reported the case of a married woman, aged about 30 years, who suffered from gangrene of the fingers following administration of liquid ergot for inducing abortion. About two bottles (twelve ounce each) of liquid ergot was consumed by her in about 2 weeks.

36 CHAPTER

Irritants of Animal Origin

After going through this chapter, the reader will be able to describe: Epidemiology and identification of snakes | Composition pharmacology and pathophysiology of snake venom | Toxicity resulting from bites of different types of snakes | Management of snake bite | Medicolegal aspects of snake bite | Features of irritant arthropods and attributes of their venom with medicolegal aspects

Irritants of animal origin mainly comprise envenomation by way of stings and bites of various reptiles, arthropods and arachnids. Because the incidence of serious bites and stings is relatively low, there remains a paucity of relevant clinical research and literature. Therapeutic decision making is often based on anecdotal information. Furthermore, the responses of different species to various toxins make it difficult to extrapolate data from animal studies to clinical application. The important representatives in each category are discussed in this chapter.

Snakes (Ophida)

EPIDEMIOLOGY

Most snake bites are inflicted on the lower limbs of farmers, plantation workers, herdsmen, and hunters. Usually, the snake is trodden on at night or in undergrowth. Snakes do not bite without provocation, but there may be an inadvertent tread or touch. **Seasonal peaks** in the incidence of snake bite are associated with agricultural activities, such as ploughing, or to fluctuations in the activity or population of venomous snakes. Severe flooding, by concentrating the human and snake populations, has given rise to epidemics of snake bite in Colombia, Pakistan, India, Bangladesh and Vietnam. Penetration of jungle areas during construction of new highways, and irrigation and hydroelectric schemes may also be the other cause.

There are more than 2000 species of snakes in the world and about 216 species in India; of which, about 50 are poisonous. Global estimates suggest that 30,000–40,000 persons die each year from venomous snake bite, but this range is likely an under-estimate because of incomplete reporting.

ANATOMY/IDENTIFICATION (Fig. 36.1)

Snakes have a characteristically elongated body, a proportionately short tail and no limbs. An opening, known as vent, is present in the rear part of the body. This serves as a common orifice for the intestinal as well as genito-urinary systems. The part behind the opening is called the tail, which is round in land snakes and flat in sea snakes.

The body is covered with scales. On the head there are two eyes, two nostrils and no external ear. The eye is covered by a transparent scale, has a round or vertical pupil but no eyelid. The lower jaw consists of two bones in front joined by an elastic ligament. It is not properly articulated with the upper jaw so that the mouth of the snake is widely distensible. This is an adaptation for the mode of feeding because a snake may swallow creatures as a whole. The upper marginal teeth are modified to form **fangs**. When a fang is broken, its place is taken by a new one, which gets developed out of the fang buds in 3–6 weeks. The fangs are solid in nonpoisonous snakes, whereas they bear a groove or channel in the poisonous ones. They are connected to the poison gland by means of a duct. The parotid salivary gland is modified in poisonous snakes to act as a venom gland. It is situated below and behind the eye (one on each side), and secretes toxic saliva, known as **venom**. The tongue of the snake is forked at the outer end and often projects out of the mouth even when it is closed. It is primarily a sense organ to help the snake in its search for food, opposite sex and helping to detect the enemies. As soon as a snake comes into the world, it moults its skin and continues to do so several times a year throughout life. Such moultings keep the snake active. All venomous snakes commonly encountered in India fall basically into two groups (families):

1. **Fam. Colubridae,** which has further three subfamilies:
   (a) **Elapidae:** Cobra, krait, coral snakes and Mamba (several African and Asian species, namely, rinkhals and spitting cobras, can eject their venom from the tips of the fangs as a fine spray for a distance of a few metres into the eyes of an enemy).
   (b) **Crotalidae:** Pit viper and rattle snake.
   (c) **Hydrophidae:** Sea snakes.
2. **Fam. Viperidae:** True vipers.

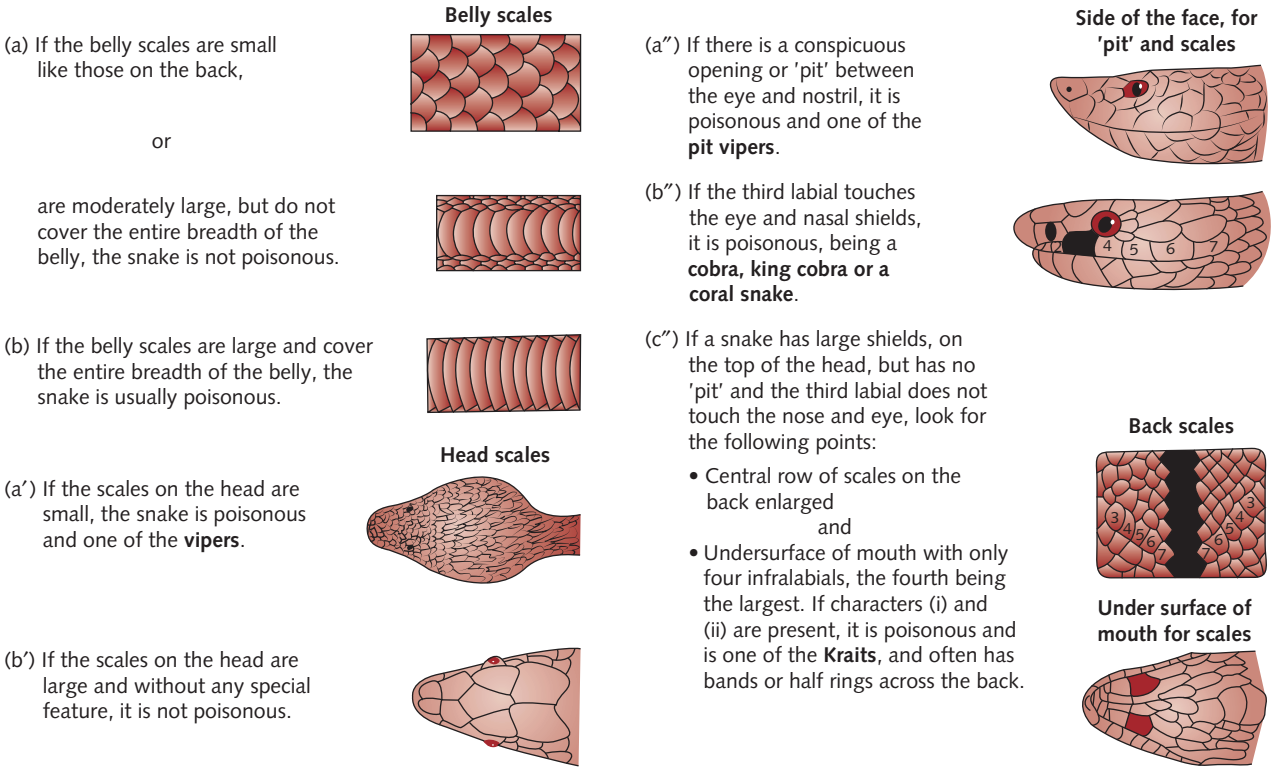


Fig. 36.1 Differentiating poisonous/nonpoisonous nature of a snake.

(The African and Middle Eastern burrowing asps or stiletto snakes, also known as burrowing or mole vipers or adders, strike sideways, impaling their victims on a long front fang protruding through the partially closed mouth.)

VENOM COMPOSITION, PHARMACOLOGY AND PATHOPHYSIOLOGY

In the fresh state, venom is a clear transparent, amber-tinted fluid and dries into a yellow granular mass, which retains its activity for many years. It is customary to classify venom as:

1. Neurotoxic (all elapids, i.e. cobra, krait, etc.)
2. Vasculotoxic (vipers)
3. Myotoxic (sea snakes)

} However, this may be an oversimplification, since mixed features may be commonly seen.

Snake venoms may contain 20 or more components. More than 90% of the dry weight is protein in the form of enzymes, nonenzymatic polypeptide toxins and nontoxic proteins. The role of enzymes in envenoming is most clearly seen in the case of venom procoagulants. Phospholipase A_2 **(lecithinase)** is the most widespread venom enzyme, which may contribute to myotoxicity, neurotoxicity, cardiotoxicity, haemolysis and increased vascular permeability. Hyaluronidase promotes spread of venom. L-Amino acid oxidase is responsible for the bright yellow colour of some viper venoms. Toxic phospholipases A_2 can block neuromuscular transmission by acting pre- or postsynaptically and can damage skeletal muscle. They include the presynaptic β-neurotoxins, β-bungarotoxin, crotoxin and taipoxin. Presynaptic neurotoxins block the release of acetylcholine at the neuromuscular junction. Postsynaptic neurotoxins such as α-bungarotoxin and cobrotoxin contain about 60 short-chain or 60–70 long-chain amino-acid residues and bind to the acetylcholine receptors at the motor end plate.

Neurotoxins of Elapidae and Hydrophidae are rapidly absorbed into the bloodstream, whereas the much larger molecules of Viperidae venoms are taken up more slowly through lymphatics. Venoms of the spitting cobras and rinkhals can be absorbed through the intact cornea, causing systemic envenoming and even death in animals. Most venoms are concentrated and bound in the kidney, and some components are eliminated in the urine. Crotaline venoms are selectively bound in the lungs, concentrated in the liver and excreted in bile, while neurotoxins such as bungarotoxin are tightly bound at neuromuscular junctions. Most venom components do not cross the blood–brain barrier.

Swelling and bruising of the bitten area result from increased vascular permeability induced by proteases, phospholipases, membrane-damaging polypeptide toxins and endogenous autacoids released by the venom such as histamine, 5-hydroxytryptamine and kinins. **Tissue necrosis** near

to the site of the bite is caused by myotoxic and cytolytic factors. In some cases, ischaemia resulting from thrombosis, intracompartmental syndrome or a tight tourniquet may contribute. **Hypotension and shock** may be attributed to hypovolaemia, vasodilatation and myocardial dysfunction. Snake venoms can cause **haemostatic defects** through procoagulants, which can activate the blood clotting cascade at various sites. Some Viperidae venoms contain fibrinogenases, which degrade fibrinogen directly and others activate endogenous plasminogen. Spontaneous systemic bleeding is caused by **haemorrhagins,** which damage vascular endothelium. The combination of defibrination, thrombocytopenia and vessel wall damage can result in massive and incontinent bleeding, a common cause of death after bites by Viperidae. **Acute renal tubular necrosis** may be caused by severe hypotension, disseminated intravascular coagulation, a direct nephrotoxic effect of the venom and myoglobinuria secondary to generalised rhabdomyolysis.

CLINICAL FEATURES OF SNAKE-BITE POISONING

The hallmark of attack by a venomous snake is the presence of fang marks, usually two but only one may be evident or sometimes, fang marks may be invisible. In contrast, bites by non-poisonous snakes may produce a characteristic U-shaped set of teeth marks. Venomous snakes can bite without injecting venom. Approximately 20% of pit viper bites and an even higher percentage of bites inflicted by some other snake families (up to 75% for sea snakes) are **'dry'**.

Degree of Toxicity Resulting From Snake Bite

It depends upon the age and size of the person bitten, the potency of the venom, the main toxic principles of the venom and the amount injected (which in turn depends upon the age, size, sex and species of the snake and whether it had recently had a prey). The season and the time of bite, the extent of anger or fear that motivates the snake and the length of time the snake holds-on and clothing or unclothing of the part bitten also influence the results. (Snakes that have recently emerged from hibernation have particularly potent venom. Nocturnal bites may be more serious than those that occur during the day.)

Inability for Envenomation

It may be due to factors given as follows: (*i*) A snake may not always inject the venom at the time of biting due to relative protection of the site. Envenomation may not occur in case of bites inflicted on shod feet or heavily clothed parts. (*ii*) Head-on bites often result in efficient introduction of venom, whereas side swipes may cause some (or entire) of the venom to escape outside the bite site. (*iii*) Human beings do not constitute usual prey for most of the snakes and therefore, snakes usually bite human being to defend themselves. In the process, snakes usually bite superficially so as to conserve their venom for their genuine prey.

Bites by Colubridae (Back-fanged Snakes)

Individuals with severe or fatal envenoming usually experience repeated vomiting, colicky abdominal pain and widespread systemic bleeding including extensive ecchymoses, incoagulable blood, intravascular haemolysis and renal failure. In some cases, local swelling and bruising may be the only features. Envenoming may develop slowly over several days.

Bites by Atractaspididae (Burrowing Asps or Stiletto Snakes)

Local effects include pain, swelling, blistering, necrosis and enlargement of local lymph nodes. Gastrointestinal symptoms include nausea, vomiting and diarrhoea. Anaphylaxis (dyspnoea, respiratory failure) and ECG changes have also been described.

Bites of Elapidae (Cobras, Kraits, Mambas, Coral Snakes, etc.)

They produce minimum local effects, which may be in the form of swelling, blistering and superficial necrosis. However, elapid venoms are **best known for their neurotoxic effects**. Early symptoms usually include vomiting, hypersalivation, headache, heaviness of the eyelids, blurred vision, paraesthesiae around the mouth, hyperacusis, dizziness, vertigo and 'gooseflesh'. Paralysis is first detectable as ptosis and external ophthalmoplegia appearing as early as a few minutes after the bite, but sometimes may be delayed for some hours. Later, the face, palate, jaws, tongue, neck muscles and muscles of deglutition may become paralysed. Respiratory failure may be precipitated by airway obstruction at this stage or later after paralysis of intercostal muscles and diaphragm. Neurotoxic effects are completely reversible either in response to antivenom or anticholinesterases, or they may wear-off spontaneously in about a week.

'Spitting' by Elapids may produce venom ophthalmia. There occurs intense pain in the eye, blepharospasm, palpebral oedema and leucorrhoea. Rarely, venom is absorbed into anterior chamber causing hypopyon and anterior uveitis.

Bites by Hydrophidae (Sea Snakes and Sea Kraits)

Persons bitten by sea snakes usually complain of headache, a thick feeling of the tongue, thirst, sweating and vomiting. Generalised aching, stiffness and tenderness of the muscles may follow. Trismus is common. Later, there is generalised flaccid paralysis as in elapid neurotoxicity. Myoglobinuria appears usually after some hours of the bite. Myoglobin and potassium released from damaged skeletal muscles can cause renal failure, while hyperkalaemia may precipitate cardiac arrest.

Bites by Viperidae (Vipers, Adders, Rattle Snakes and Pit Vipers, etc.)

Bites by Viperidae usually produce more **severe local effects** than other snake venoms. Swelling may become detectable within 15 minutes, but sometimes may be delayed for several hours. It spreads rapidly and involves the whole limb and adjacent trunk. Bruising, blistering and necrosis may appear during the next few days. If there is no swelling for about a couple of hours after a viper bite, it is usually safe to assume that there has been no envenoming. However, fatal envenoming by a few species can occur in the absence of local signs. **Haemostatic abnormalities are characteristic of bite by Viperidae.** Persistent bleeding from fang punctures, venepuncture or injection sites, etc. suggests that blood is incoagulable. Spontaneous systemic haemorrhage is most often detected in the gingival sulci. Epistaxis, haematemesis, haemoptysis, subconjunctival, retroperitoneal and intracranial haemorrhages have been reported. Direct myocardial involvement is suggested by an abnormal ECG or cardiac arrhythmia. Renal failure is the major cause of death in victims envenomed by some species of Viperidae. Victims of Russell's viper may become oliguric within a few hours of the bite and have loin pain suggesting renal ischaemia.

MANAGEMENT OF SNAKE BITE

First Aid Measures

Allaying anxiety and freight of the victim should form a part of the treatment. The victim should be reassured about the fact that all snakes are not poisonous, and even the poisonous snakes may not always inject a lethal dose. Arrangement needs to be made for delivering the victim to the hospital for medical care while keeping him/her as inactive as possible to limit systemic spread of venom.

After viperid bites, local mechanical suction applied to the site within a few minutes may remove some percentage of deposited venom. Mouth suction should be avoided as it inoculates the wound with oral flora and theoretically can also result in the absorption of venom by the rescuer through lesions in the upper digestive tract. A proximal lympho-occlusive constriction band may limit the spread of venom when applied shortly after the bite. To avoid worsening tissue damage, however, the band should not interrupt arterial blood flow. The bitten extremity should be splinted and kept at approximately heart level.

For elapid or sea snake bites, local incisions and suction may be useful. The entire bitten extremity may be wrapped with an elastic or crepe bandage and then splinted. The bandage is applied with good snugness. This technique usually restricts absorption and circulation of venom significantly.

Hospital Treatment

In most cases of snake bite, there are uncertainties about the species, quantity and composition of venom injected that can be resolved only by admitting and observing the patient for at least 24 hours.

Antivenom Therapy

The most important decision in the management of a patient bitten by a snake is whether or not to give antivenom. Most commercial antivenoms are of equine origin and carry a risk of anaphylactic, anaphylactoid and delayed hypersensitivity reactions. Skin testing does not reliably predict which patients will have an allergic reaction to equine antivenom. False-negative and false-positive results may be there. Therefore, appropriate loading doses of intravenous antihistamines and cimetidine may be given in an effort to limit acute reactions. Epinephrine should be immediately available. It is almost never too late to give antivenom while signs of systemic envenoming persist, but ideally, it should be given as soon as it is indicated. Antivenom has proved effective up to 2 days after Hydrophid bites and in patients still defibrinated, weeks after bites by Viperidae. In contrast, local envenoming is probably not reversible unless antivenom is given within a few hours of the bite. **Intravenous route is the most effective,** and it should be administered after dilution (approximately 5 ml of isotonic fluid/kg body weight) rather than intravenous 'push'.

Initially, the infusion may be regulated at 15–20 drops per minute. The rate can be progressively increased so that the infusion is completed in 1–2 hours. Begin with 8–10 vials and check for improvement. It is advisable to wait for 6 hours and perform investigations. If there is no significant improvement, additional 5–8 vials can be administered, and a final round of 5–8 vials can be given in the continuing absence of improvement. (The accompanying package insert usually outlines techniques for administration and dosage.)

In India, antivenins (antivenene) are prepared from horses. Monospecific (monovalent) serum (not available in India) is prepared using the venom of a single snake. Polyspecific (polyvalent) serum is prepared by using venom of more than one snake. This is prepared at Haffkine Institute, Mumbai and Central Research Institute, Kasauli. It is in the form of a lyophilised powder of horse serum produced by immunisation of horses with the venom of four snakes, namely, common cobra, common krait, Russell's viper and saw-scaled viper. (The serum is lyophilised by drying it from the frozen state under high vacuum.)

Indications for antivenom therapy may include:

- Haemostatic abnormalities such as spontaneous systemic bleeding, incoagulable blood or thrombocytopenia.
- Hypotension and shock, abnormal ECG or other evidence of cardiovascular dysfunction.
- Neurotoxicity and generalised rhabdomyolysis.
- Supporting evidence of severe envenoming includes neutrophilic leucocytosis, elevated serum enzymes like creatine phosphokinase and aminotransferases, haemoconcentration, myoglobinuria, haemoglobinuria, methaemoglobinuria, hypoxaemia and acidosis.

- Tender enlargement of the local lymph nodes usually indicates a systemic spread. (The apparent serum elimination half-lives of antivenoms in envenomed patients range from 26 to 95 hours. Envenomed patients should therefore be assessed for at least 3 or 4 days.)

Other Measures

- IV fluids, vasopressors, as indications arise.
- Whole blood or fresh frozen plasma, if there are clotting abnormalities and anomalous bleeding.
- Use cholinergics to reverse the neurotoxic features of elapid bites. Neostigmine (0.25–0.5 mg IV half hourly) needs to be given if there are signs of neuroparalysis. Give atropine 0.6 mg IV before every injection of neostigmine to block its muscarinic side effects.
- Renal failure to be tackled on appropriate lines, if need arises.
- Oxygen, assisted ventilation if there is respiratory failure.
- A fasciotomy may be required in very rare cases. Before performing fasciotomy, an objective assessment of impaired blood flow using Doppler ultrasound may be conducted.

POSTMORTEM APPEARANCES

Two bite marks about 1 cm deep in cases of elapid and 2 cm deep in cases of viper may be present. These should be searched and if need be, magnifying lens may be used. Sometimes, the bite marks may not be visible. **In case of viper bite,** there is swelling and cellulitis about the bitten part. Local appearances are more striking due to considerable oozing of blood from the site of puncture. The blood is generally fluid and haemolysed causing early staining of the blood vessels. As the venom is predominantly vasculotoxic, there may be haemorrhages in the lungs and also extravasation of blood in the serous membranes such as pleura and pericardium. Endocardial haemorrhages are characteristically found in the left ventricle, septum and papillary muscles. Haemorrhages may also be found in other organs. The regional lymph nodes are swollen and haemorrhagic. **In case of elapids,** where the venom is predominantly neurotoxic, there may not be definite local appearances and the cause of death may be difficult to approach except for the signs of asphyxia.

Snake specific venom antigens have been detected in wound swabs, aspirates or biopsies, serum, urine, CSF and other body fluids. Therefore, skin and underlying tissue surrounding the fang punctures, wound and blister aspirate, serum and urine should be collected and sent to the laboratory. Of the various techniques for the detection of snake specific venoms, radioimmunoassay (RIA) is probably the most sensitive and specific. However, enzyme immunoassay (EIA) has been most commonly used as it is cheap and simple. The RIA can detect venom levels as low as 0.4 µg/L, whereas EIA can detect venom levels as low as 5 µg/L.

MEDICOLEGAL ASPECTS

The vast majority of snake bites are accidental in nature. Homicide may rarely be committed (for instance by throwing a venomous snake on a sleeping victim or slipping it under the bathroom door or through a window). Suicides are virtually unreported (with the famous exception of Queen Cleopatra who is reputed to have committed suicide by getting herself bitten by a venomous snake, an asp which is an exotic variety of viper).

Pursuing and killing the snake is not recommended but if the snake has been killed, it may be taken to the hospital and may provide a clue towards treatment. However, the snake must not be handled negligently as even a severed head can inject venom. This may be possible up to an hour or so after death due to the reflex action.

Some nonvenomous snakes kill humans without injecting venom. They inflict death by squeezing the victim within their coiled body, e.g. python, boa constrictor, etc. Sometimes, killing by some other means may be disguised as death due to snake bite.

Sometimes, snake venom is used to kill cattle by a peculiar method for the sake of hides. For this purpose, a cobra is placed in an earthen vessel with a banana. The cobra is irritated by applying heat to the vessel. It bites the fruit, the pulp of which is then smeared on a rag and the rag thrust into the animal's rectum with the help of a bamboo stick. *Sui* poisoning of cattle resembles viperine snake bite.

Snake venom is poisonous only when injected and has no ill-effects when taken by mouth, as the venom is not absorbed from the gastric mucous membrane. The bodies of animals killed by snake poisoning may be eaten without ill-effects, but their blood is poisonous and is fatal if injected into the human body.

Arthropods

Phylum Arthropoda is the largest of the phyla in the animal kingdom and comprises six major groups (classes). However, class Insecta and Arachnida will be discussed here.

CLASS INSECTA

Bees

Of the various types of bees, the bumble bee (large in size and makes a buzzing sound) is the only harmless one. **Honey bees** can sting, but do so when threatened or harmed. They have a barbed stinger, which contains two lancets. When exercising sting, these lancets become firmly anchored to human skin resulting in disengagement of the whole apparatus from the abdomen. The eviscerated bee soon dies, but the reflex action of the attached muscles may continue to inject venom for some time.

Wasps

Of the various types of wasps, the yellow jackets are most ill-tempered and may sting without provocation. The wasps and hornets are usually nonaggressive unless their hive is disturbed. The stinger of the wasp does not have barbs and hence can easily be withdrawn and reinserted. (Wasps and hornets, unlike bees, are scavenging insects and can transmit infection during a sting.)

Venom Composition

- Direct toxic effects are mediated by mixtures of low-molecular weight compounds such as serotonin, histamine, acetylcholine and several kinins.
- Polypeptide toxins in honey bee venom include mellitin, which damages cell membranes; mast cell-degranulating protein, which causes histamine release; apamin (a neurotoxin) and adolapin, which has anti-inflammatory action.
- Enzymes in venom include hyaluronidase, which allows the spread of other venom components and phospholipases.

Symptoms and Signs

- Uncomplicated stings cause immediate pain, a wheal and flare reaction, and local oedema and swelling that subside in a few hours.
- Multiple stings can lead to vomiting, diarrhoea, generalised oedema, dyspnoea, hypotension and collapse. Rhabdomyolysis and intravascular haemolysis may cause renal failure.

Diagnosis of Venom Hypersensitivity

Type I hypersensitivity is confirmed by detecting venom specific IgE in the serum using radioimmunosorbent testing (RAST). Intradermal skin tests using dialysed freeze dried pure specific venoms are also diagnostic. The RAST test has been used for postmortem diagnosis of Hymenoptera sting anaphylaxis. [Insects that sting to defend their colonies or subdue their prey belong to the order Hymenoptera, which includes aphids (bees and bumble bees), vespids (wasps, hornets and yellow jackets), and ants.]

Treatment

In case of bee stings, stingers embedded in the skin should be scraped or brushed off with a blade or a fingernail but not removed with forceps, which may squeeze more venom out of the venom sac. The site should be cleansed and disinfected, and cold compresses may be applied to slow down the spread of venom.

- Anaphylactic reaction necessitates immediate administration of epinephrine hydrochloride (0.3–0.5 ml of 1:1000 solution subcutaneously, repeated if necessary).
- Intravenous epinephrine (2–5 ml of a 1:10,000 solution) may be administered by slow push in profound shock.
- Antihistaminics are beneficial. Large local reactions may require a short course of oral therapy with glucocorticoids.
- Fluid resuscitation, oxygen, intubation and vasopressors may be required. The patient should be observed for 24 hours for recurrent anaphylaxis.

Spanish Fly (Cantharis vesicatoria or Blister Beetle)

A special type of insect-related poisoning is associated with this creature. The active principle is a highly irritant substance called **cantharidin**. The active principle is insoluble in water and soluble in alcohol, fats, ether and chloroform. Cantharis may be administered in the form of powdered beetles, or the tincture, or the active principle. The powder cannot be easily administered as it floats for sometime in any liquid with which it is mixed and attracts attention due to shining green particles.

On external application, it causes a blister or vesicle. Therefore, the Spanish fly is also known as blister beetle or cantharis vesicatoria. **On internal administration,** it causes severe inflammation of the gastrointestinal and genito-urinary tract. There is burning sensation in the throat and stomach, difficulty in swallowing, nausea, abdominal pain, vomiting of blood stained material and diarrhoea with blood and mucus. As time passes, nephrotoxic effects become evident. There occurs a dull heavy pain in the loins and constant desire to micturate but only small amount of blood stained urine is passed **(strangury)**. In the male, persistent and painful erection of the penis (priapism) may occur and there may be frequent seminal emissions. In females, there may be engorgement of the vulva and abortion may occur. In fatal cases, coma with convulsions usually precedes death.

Fatal Dose and Fatal Period

Cantharidin is readily absorbed from all surfaces including the skin, and it is possible that 10 mg of active principle (cantharidin) may cause death. Ordinarily, 1.5 gm of powdered cantharides is regarded as a fatal dose. Death usually occurs within 24 hours.

Treatment

Stomach wash may be carried out with warm water. Demulcents may be given in any quantity. Renal damage should be treated on appropriate lines. Alkalies need to be given to allay the irritation of the genito-urinary tract.

Postmortem Appearances

The whole alimentary tract may show intense inflammation. Parts of the powdered beetle may be found as shining elytra. The shining wings of the beetle resist putrefaction and may provide a valuable clue as to the identity of the poison. The kidneys may show acute nephritis and the genito-urinary tract, severe inflammation.

Medicolegal Aspects

Most of the cases of poisoning result from accidental overdose, arising out of the mistaken belief in its aphrodisiac properties. Contact with skin or eyes can result in intense irritation with blister formation.

It is said that infamous Marquis de Sade (the perversion 'sadism' is named after him), poisoned a number of prostitutes in a brothel with cantharidin in an attempt to arouse them. However, many of them died.

CLASS ARACHNIDA

Scorpion and spider are important representatives of this class.

Scorpion

It carries a cephalothorax, an abdomen and a six-segmented tail, which terminates in a bulbous enlargement called telson. The telson contains the stinger and venom apparatus. In addition, the scorpion also has two claws, which help to grasp its prey. The scorpions feed at night and remain hidden during the day in crevices or burrows or under wood, loose bark or rocks. Scorpions sting human beings only when disturbed.

The venom usually carries haemotoxic and neurotoxic actions. (It is predominantly neurotoxic.) It is a potent autonomic stimulator resulting in the release of massive amounts of catecholamines from the adrenal glands and nerve endings into the circulation. It also has some direct effect on the myocardium. Both these actions result in cardiac arrhythmias, hypertension and systolic dysfunction. Later, due to depletion of catecholamines, hypotension, bradycardia, etc. occur.

Symptoms and Signs

Local irritation is characterised by redness and burning pain radiating from the site. In most cases, the pain grows within a few hours of being stung. The victim may not be able to localise the pain due to its radiation along the dermatomes involved. However, the presence of local swelling and a punctated haemorrhagic spot may help in localising the site of the sting. There may be headache, giddiness, nausea, profuse perspiration, chest discomfort, paraesthesias, hypersalivation, cold extremities and sometimes, priapism. Hypertension may occur within 6 hours of sting, while pulmonary oedema takes longer time. Later, features suggestive of myocarditis may develop. Neurologic manifestations may persist for up to a week or so. While the mortality in adults is negligible, children may succumb from pulmonary oedema.

Treatment

Stings of nonlethal species require at most ice packs, analgesics or antihistamines. Antivenin therapy can reduce or eliminate mortality from more severe envenomations. Keeping the patient calm and applying pressure dressings and cold packs to the sting site decreases the absorption of venom. Intravenous infusion of midazolam controls the agitation, flailing and involuntary muscle movements produced by scorpion stings. Hypertension and pulmonary oedema respond to nifedipine, nitroprusside, hydralazine or prazosin, and bradyarrhythmias can be controlled with atropine. Currently, treatment of accelerated hypertension with prazosin (post-synaptic alpha-blocker) and nifedipine (calcium channel blocker) has been advocated. Simultaneously, correction of fluid loss due to sweating and vomiting needs to be taken care of by administering intravenous fluids.

Spider

The vast majority of spiders are harmless in the sense that they do not attack unless provoked. Two varieties are capable of producing severe toxicity.

Latrodectus mactans (Black Widow or Hourglass Spider)

This is found all over the world. The female of the species is larger and more vicious. There is usually a red hourglass spot on the dorsal surface of its shiny black body. **The venom is predominantly neurotoxic** and therefore does not produce much local necrosis and some persons experience no other symptoms except a sharp pinprick. However, α-latrotoxin, the most active component of the venom, binds irreversibly to nerves and causes release and eventual depletion of acetylcholine, norepinephrine and other neurotransmitters from presynaptic terminals. Within an hour or so, painful cramps spread from the bite site to large muscles of the extremities and the trunk. Other features may include salivation, diaphoresis, vomiting, hypertension, tachycardia, laboured breathing, anxiety, headache, fasciculations, paraesthesia, hyper-reflexia, urinary retention, uterine contractions and premature labour. Rhabdomyolysis and renal failure have been reported.

Loxosceles reclusa (Violin Spider or Brown Recluse)

It has a violin-shaped mark on its brown coloured back, and the female (as in case of black widow) is more dangerous. **The venom is mainly cytotoxic.** It contains an esterase, alkaline phosphatase, protease and other enzymes that produce tissue necrosis and haemolysis. Sphingomyelinase B, the most important dermonecrotic factor binds cell membranes and promotes chemotaxis of neutrophils, leading to vascular thrombosis. **Initially,** the bite is painless or produces a stinging sensation. Within the next few hours, the site becomes painful and pruritic, with central induration surrounded by a pale zone of ischaemia and a zone of erythema. **In severe cases,** the erythema spreads and centre of the lesion becomes haemorrhagic and necrotic. A black eschar forms and sloughs several weeks later, leaving an ulcer.

Treatment

Initial management includes local cleansing, application of sterile dressings and cold compresses. Elevation of the effected limb is helpful in preventing the spread of venom. Analgesics, antihistamines and antibiotics may be administered, if indicated. Within 48–72 hours, administration of dapsone (a leucocyte inhibitor) may halt the progression of lesions that are becoming necrotic. Usually, the dose is 50–100 mg twice daily after glucose-6-phosphate dehydrogenase deficiency has been ruled out.

Debridement and later skin grafting may be necessary after signs of acute inflammation have subsided. The victims should be monitored closely for signs of haemolysis, renal failure and other systemic complications.

37 CHAPTER
Somniferous Group

After going through this chapter, the reader will be able to describe: Source, extraction and characteristics of opium | Alkaloids of opium | Mechanism of action and metabolism | Acute and chronic morphine poisoning | Features of 'heroin' poisoning with medicolegal aspects | Medicolegal aspects of 'pethidine' and 'methadone' poisoning

This group of poisons is known as somniferous or narcotic poisons, because their preparations are used therapeutically to lessen pain and induce sleep. Though there are numerous examples of drugs that produce such effects, this group includes only those that are derived from opium, i.e. the **opiates**. Those that have a similar action but are not derived from opium are often designated as the **opioids**.

Opium (*Afim*)

The word 'opium' is derived from the Greek name for 'juice', related to the milky exudate of the poppy capsule. The first authentic reference to poppy juice is found in the writings of Theophrastus, who was a Roman physician. He referred to opium as 'meconium', which is Greek name of 'poppy'. In 1806, Serturner isolated an alkaloid from opium, which he named 'morphine' after Morpheus, the Greek God of dreams.

Opium is the coagulated air-dried latex juice obtained by longitudinal (vertical) incisions of the unripe capsules of the white poppy plant, *Papaver somniferum*, which grows in India. The milky exude from the incisions on the unripe poppy capsule is air-dried to obtain dark brown opium.

Ripe and dry poppy capsules (*post ka doda*) contain only a trace of opium and are used for their sedative and narcotic action. Their warm decoction is used locally as a sedative fomentation and poultice. The **poppy seeds** (*khas khas/posto*) are creamish in colour, do not contain any opium and, therefore, harmless. They are used as food and occasionally sprinkled over sweets. They yield a bland oil, known as poppy seed oil (*khas khas ka tel*), which is largely used for culinary and lighting purposes.

Opium occurs in more or less rounded, irregularly formed or flattened masses. It has a characteristic smell and a bitter taste. **When fresh,** it is plastic and internally moist, coarsely granular or nearly smooth and reddish or chestnut brown, but becomes hard, brittle and dark brown **on storage**. Concentration of morphine varies with season, geographic origin and the strain of the plant. In the variety from Turkey, concentration of morphine is about 20%. In the Indian variety, the concentration of morphine is about 10%. (In India, cultivation of opium is permitted only under a license. The morphine content of opium is standardised in the Govt. Opium Factory at Ghazipur, to contain about 10% morphine. The total alkaloids of this opium usually go up to 40%). The list of naturally occurring alkaloids and the preparations derived from them is given in Table 37.1.

Table 37.1 Various Naturally Occurring Alkaloids and Preparations Derived from Them

Naturally occurring alkaloids	Derived/semi-synthetic	Synthetic
Phenanthrene group Morphine (about 10%) Codeine (about 0.5%) Thebaine (about 0.3%) **Isoquinoline group** Papaverine (about 1%) Narcotin (noscapine) (~6%) Narceine	Diacetyl morphine (heroin) Benzyl morphine Oxymorphone Hydromorphone Dehydromorphine N-Allyl-normorphine (nalorphine) Hydrocodone bitartrate	Pethidine Hydroxypethidine Methadon Isomethadon Normethadon α-Methadol Acetyl methadol Levorphanol tartrate

MECHANISM OF ACTION AND METABOLISM

The existence of opiate receptors was first suggested by two chemists Beckett and Casy in 1954, which was subsequently experimentally demonstrated in 1973. Presently, it is considered that there are three major classes of opiate receptors to which different opiates bind with different affinity:

- Mu (μ) receptor, also known as OP_3. Most of the clinically used opiates are relatively selective for μ receptors. They mediate euphoria, supra-spinal and peripheral analgesia, respiratory depression, gastrointestinal dismotility and physical dependence.
- Kappa (κ) receptor, also known as OP_2. They mediate miosis, spinal analgesia, central nervous system depression, supraspinal analgesia, etc.
- Delta (δ) receptor, also known as OP_1. It is said to be important in spinal and supraspinal analgesia.

  [Sigma (σ) receptor, originally thought to be an opiate receptor, is no longer considered to be so since it is insensitive to naloxone, which is most important character of such receptors. However, some opiates like pentazocine and dextromethorphan are sigma receptor agonists. Stimulation of sigma receptor produces psychotomimetic effects and disorders of movements, both of which have been reported with these agents.]

Substances capable of antagonising one or more of these actions include nalorphine, levallorphan, cyclazocine, butorphanol and pentazocine; each of which has mixed agonist and antagonist properties. However, naloxone, nalmefene and naltrexone are pure opiate antagonists.

Opioid drugs are absorbed from the gastrointestinal system, the lungs and/or the muscles. The most rapid and pronounced effects occur following intravenous administration, with only slightly less efficient absorption after smoking or inhaling the vapour, and the least intense actions are seen after absorption from the digestive tract. Most of the metabolism of opioids occurs in the liver, primarily through conjugation with glucuronic acid and only small amounts are excreted directly in the urine or faeces. The plasma half-lives of these drugs range from 2.5 to 3 hours for morphine to more than 22 hours for methadone and even longer for levomethadyl acetate as reported.

ACUTE POISONING

The symptoms of opium poisoning are practically those of morphine poisoning on account of the high morphine content. They usually appear in from half an hour to about an hour after the poison has been swallowed. However, when the poison is injected, effects may appear within a few minutes. The opioids produce their effects by binding to different types of opioid receptors throughout the body including the central nervous system as mentioned above. The effects may be studied under the following stages.

Stage of Excitement

This stage may be absent if a large dose is taken. In adults, a euphoric feeling of well-being usually comes early. There may be certain pleasurable mental excitement usually of short duration due to effects on limbic system. The breath may smell of opium. In children, convulsions are usually the marked feature of this stage.

Stage of Stupor

The stage of excitement is soon succeeded by weariness, headache, giddiness, a sense of weight in limbs, diminution of sensibility and an intense tendency to sleep from which the patient can be aroused by applying external stimuli. The pupils are constricted, the face and lips cyanosed. The pulse and respiration are almost normal.

Stage of Narcosis

Here, the victim passes into deep coma from which he cannot be aroused. The muscles are relaxed and reflexes abolished. The pupils are constricted to pinpoint and show no reaction to light. (This state of pupils, probably, is due to the depression of supranuclear inhibition of the pupillary constrictor tone.) They may dilate terminally when anoxia ensues. The conjunctivae are injected. The blood pressure falls. The pulse is small and weak in the early stages but later becomes slow and full as coma develops. The breathing is slow, sighing and irregular; the rate may fall from 2 to 4 per minute. This results from a decreased response of the brainstem to carbon dioxide tension and is known as *Cheyne Stokes Breathing*. The temperature is subnormal, and the skin cold and bathed in sweat. Coma deepens and death usually results from asphyxia due to respiratory paralysis, which may be preceded by convulsions.

(Acute changes in the gastrointestinal system are the result of decreased motility with resulting constipation and anorexia. Emptying of stomach is delayed. Hence, even in delayed cases, gastric lavage may be done. Further, it is also important due to excretion of morphine in the stomach, i.e. after absorption in the blood, morphine is excreted into the stomach, from which it is again liable to be reabsorbed.)

Fatal Dose and Fatal Period

The toxic dose may be variable because a considerable tolerance can be acquired. In a nonaddict, 200 mg of morphine and its equivalent of opium (2 gm) is a fatal dose. Usual fatal period may extend from a few hours to 2 days (Table 37.2).

Treatment

It needs to be regulated by the circumstances of each case. The first step in managing overdose is to provide any needed respiratory or cardiovascular support including intubation for airway protection.

Table 37.2 Therapeutic and Fatal Dose of Various Compounds

Compound	Therapeutic dose (mg)	Usual fatal dose (mg)
Crude opium	–	500
Morphine	10–15	200
Pethidine	50–150	1000
Codeine	10–60 (oral)	800 (oral)
Methadone	5–10	100
Heroin	–	50
Pentazocine	30–60	300
Propoxyphene	100–150 (oral)	1000 (oral)
Diphenoxylate	10–20 (oral)	200 (oral)

Doses mentioned are for parenteral route unless mentioned otherwise.

- As the vomiting centre soon gets depressed, **gastric lavage** first with tepid water (the returning fluid kept for analysis) and then with the solution (1:5000) of potassium permanganate needs to be performed. This tends to oxidise opium into harmless substances. Lavage should be continued till washed water returns with its original pink colour. (Even when the drug has been administered through injection, the permanganate treatment should be adopted since morphine is excreted in the stomach. Some solution should be left in the stomach with the aim of oxidising the alkaloid that might be so excreted.)
- Purgative such as magnesium sulphate (15 gm) orally to clear the gastro-intestinal tract.
- Antibiotics to prevent pulmonary infection.
- The kidneys and skin need to be stimulated to excrete the poison by giving intravenous fluids. Correction of fluid and electrolyte imbalance.
- **The narcotic antagonists:** The opiate antagonists compete with heroin and other opioids for receptors, reducing the effects of the opioid agonists. Currently, the drug of choice is **naloxone**. It is pure opioid antagonist and competes with opioids at receptor sites. It can reverse not only the respiratory depressant, analgesic and euphoric effects of opioids but also the dysphoric, delusional and hallucinatory properties of the synthetic opioids. It is given in a dose of 0.4–2 mg intravenously and can be repeated every 10–15 minutes up to a dose of 10 mg. (It is important to titrate the dose relative to the patient's symptoms. The goal is to ameliorate the respiratory depression but not to provoke a withdrawal state.) The most widely used antagonist in rehabilitation is **naltrexone**. It is free from agonist properties, produces no known withdrawal symptoms when stopped and its side effects tend to be mild. To avoid precipitating a withdrawal syndrome, patients should be free of opioids for a minimum of 5 days before beginning treatment with this medication. In addition, they should first be tested with 0.4–0.8 mg or shorter-acting agent naloxone to be certain that they are able to tolerate the long-acting antagonist.

Postmortem Appearances

Intense postmortem lividity/staining, deeply cyanosed face, extreme cyanosis over the fingertips, lips and ear lobules, frothing from mouth and nose are the usual **external findings**.

Internally, the stomach may show the presence of small, soft, brownish lumps of opium and the smell of the drug may be perceived, which disappears with the onset of decomposition. The lungs are congested and oedematous with petechial haemorrhagic spots in the under surface of the pleura. In addition, trachea and bronchi may be covered with froth. The blood is usually dark and fluid. Organs are congested. Combination of oedema of the lungs and intense lividity of the face almost approaching to blackness are the **'hallmark'** of diagnostic findings. It is probably responsible for the old belief that the face of a poisoned person turns black after death.

Medicolegal Aspects

Opium has been used **for suicidal purposes** as it lessens pain and induces sleep. For this purpose, it is usually mixed with mustard oil or ginger oil when its action starts quickly and in such cases it is difficult to remove opium by washing the stomach. Sometimes, morphine may be taken by injection to commit suicide.

On account of its dark brown colour, characteristic odour and bitter taste, it is **usually not selected for homicidal purposes**. However, it is sometimes used as an infanticidal agent to get rid of illegitimate children. Death of an infant has resulted from the breastfeeding of a woman who had smeared her nipple with tincture of opium with evil motive.

Accidental poisoning may occur in addicts, in children or due to therapeutic misadventure. Infants and children may suffer poisoning due to accidental swallowing of crude opium or opium pills meant for their parents or grandparents, who are in the habit of using the drug. They may also be sometime poisoned by an accidental overdose, as they are usually drugged with opium by their parents, especially the labour class with a view to lulling them to unnatural sleep. Elimination of opium through breast milk is known by occurrence of fatal poisoning in infants sucking their mothers, who have been poisoned by opium. It is said to be an aphrodisiac drug but its chronic use actually diminishes performance. Poisoning may result from an overdose when thus used.

Crude opium is sometimes used on special festive occasions, *kasoomba*, its decoction, is offered to the guests. Opium is also smoked in the form of madak, chandu or opium dross. An infusion of poppy capsules is often drunk by some people in certain districts in Punjab and parts of Rajasthan. A sweet, called *halwa,* prepared from the juice extracted from green poppy capsules, is also used.

Opium is believed to increase the duration of the sexual act. Hence, it is often taken by young men, who get accustomed to the drug by its constant use. It is also used to steady the nerves for doing some bold act. For instance, in the ancient times, the Rajputs used to take the drug before they took part in battles.

CHRONIC POISONING (MORPHINO-MANIA/ MORPHINISM)

The symptoms and signs in such cases may include gastrointestinal disturbances with anorexia, constipation, furred tongue, irritability, disturbed sleep (insomnia with inadequate dose) and fatigue. Impotence in males may occur as the drug decreases levels of luteinising hormone, with a subsequent reduction in testosterone, which might contribute to the decreased sex drive reported by most opioid-dependent individuals. Females may show frigidity. Mental depression, moral degradation, loss of self-respect and morality, a desire to procure drug by any means may be the other features. For those who use morphine parenterally (known as 'skin popping' when used subcutaneously and 'main lining' when injected intravenously), pigmentation and scar formation may be there, which are often masked by artificial tattooing. **Treatment** consists of gradual withdrawal of drug, maintenance of food, nutrition, vitamins, etc., physical restrain (if necessary) and good nursing care. At the same time as the drug of addiction is being withdrawn, a less potent drug needs to be given as a substitute to take care of the withdrawal symptoms that are likely to develop. The drug of choice for this purpose is **methadone**, which needs to be given at a dose of 30–40 mg/day and then gradually tapered off. A **beta-adrenergic blocker** like propranolol is said to be effective in relieving anxiety and caving associated with opiate addiction, but has no effect on physical symptoms. Tranquilisers may be given if necessary. Psychiatric counselling is frequently necessary after the acute phase of de-addiction is over.

Symptoms of Withdrawal

Withdrawal symptoms (also referred to as withdrawal reaction or cold turkey), usually the opposite of the acute effects of the drug include nausea and diarrhoea, coughing, lacrimation, mydriasis, rhinorrhoea, profuse sweating, muscle twitching, piloerection (goose bumps) as well as mild elevation in body temperature, respiratory rate and blood pressure. In addition, diffuse body pain, insomnia and yawning occur along with intense drug craving. The intensity of symptoms usually reaches peak within 36–72 hours after discontinuation of the drug, and the acute syndrome disappears within 5–8 days. However, a protracted abstinence phase of mild symptoms may persist for 6 or more months. **Treatment of withdrawal symptoms** requires administration of sufficient opioid medication on the first day to decrease symptoms followed by a gradual withdrawal of the drug. Methadone is preferred for this purpose. After several days of a stabilised drug dose, the opioid is then decreased by 10–20% of the original day's dose each day. (To estimate the first day's dose from the patient's history, 1–2 mg of methadone can be considered approximately equivalent to 3 mg of morphine, 1 mg of heroin or 20 mg of meperidine.)

Heroin

Opiates have been used for at least 3500 years, mostly in the form of crude opium or in alcoholic solutions of opium. Morphine was first isolated in 1806. The first semisynthetic opium derivative diacetylmorphine **(heroin)** was introduced into medicine in 1898. The first purely synthetic drugs with morphine-like opioids, meperidine (Demerol) and methadone (Dolophine) were introduced into medical practice in the 1940s.

Heroin was created in an attempt to find a safer type of morphine and was named, presumably due to drug's 'heroic' ability to mimic the effects of morphine without causing addiction. Developers hoped that the new drug would be used to cure morphine addiction. Unfortunately, heroin is in fact highly addictive. Heroin is preferred by the addicts due to its more intense action as compared to morphine. It is a white or brown powder (depending on where it has been processed) that can be smoked, sniffed or dissolved in water and injected. It can be smoked when the end of a cigarette is dipped in heroin powder and lighted (this is called ack-ack); 'chasing the dragon' or 'playing the organ'. The drug is lighted, and smoke inhaled. Subcutaneous injection is called skin-popping, intravenous as mainlining. Heroin is not taken orally because it is rapidly hydrolysed in the stomach.

POSTMORTEM FINDINGS

Autopsy findings are relatively nonspecific. However, certain features can be useful pointers. Presence of injection marks (needle marks) commonly in the antecubital fossa on the front of the elbow, or into one of the prominent veins of the forearms or dorsum of the hand. In habitual users, sclerosis of the veins may lead to the arms being used randomly. The veins of the dorsum of the foot may be used when the hands and arms have become unusable because of thrombosis and scarring. Less common sites are thighs, abdominal wall; where the injections may be subcutaneous, which can lead to areas of subcutaneous sclerosis, fat necrosis and abscesses, etc. Tattooing is common among addicts, sometimes to conceal old scars or fresh injection sites, but often manifesting psychiatric significance.

Internal abnormalities due to narcotism are not prominent at autopsy. Examination of the needle scars will reveal perivenous fibrosis in the intravenous addict and acute or chronic abscesses or diffuse subcutaneous scarring in the skin popper. Long-term use of heroin itself causes no damage to the body organs that can be identified at autopsy. There may be subtle chronic changes in the reticuloendothelial system,

which probably result from persistent antigenic stimulation by unsterile injection of foreign material. These changes include enlargement of lymph nodes near the liver and pancreas, prominence of thymus, mononuclear cell infiltrates in the portal triads of the liver. The most striking change is severe congestion and oedema of the lungs with abundant froth filling the bronchi, and trachea, and protruding from the nose and mouth. This is the so-called **heroin lung**.

Heroin, either alone or mixed with cocaine, is numerically the most likely way of transmitting hepatitis, HIV and pyogenic infections. In relation to AIDS and HIV positivity, opinions differ. It seems obvious that HIV virus is not in the same category of infectivity as hepatitis and no case is yet known of postmortem contagion. It has been reported that HIV may remain viable for many days after death.

CAUSE OF DEATH

Heroin is rapidly metabolised in the body by blood esterases to 6-mono-acetyl morphine, so that taking of either heroin or morphine results in the finding of morphine toxicologically. Using an immunochemical method, excretion of morphine or morphine equivalents may be detected for 2 weeks or more after the last dose, depending upon the sensitivity of the testing method. Blood, urine and bile are good specimens for morphine recovery. In the absence of urine and bile, kidney and liver need to be retained for analysis. The conclusion that the death is due to narcotism is based on an examination of the scene, investigation of circumstances, history obtained from relatives and friends, autopsy examination that demonstrates drug use and excludes other causes of death and toxicologic analysis. Thus, the analysis is not the final arbiter of the cause of death, although it is a highly important component. Further, the interpretation of laboratory analytical results may present considerable difficulties as outlined in the first introductory chapter. The unknown factor is usually of 'tolerance', and there is no method of assessing this at autopsy. The absolute concentration of morphine in the organs, and possibly bile, might provide a clue to previous use of the drug with the implication of tolerance but is not proof of this. **As far as the manner of death is concerned,** there may be different views—death may not be considered accidental in the sense of being unexpected and by unforeseen chance, since the addict deliberately took the drug knowing that he risked death. It may be considered accidental as being inadvertent death due to self-administration of the drug for purposes of euphoria. It may be considered suicidal because of inherent self-destructive nature of the drug taking and known risks involved.

SOME ADDITIONAL POINTS OF MEDICOLEGAL INTEREST

- A new type of Mexican heroin called 'black tar' is becoming increasingly popular in the USA, because it is 40 times stronger and 10 times cheaper than pure heroin.
- The illicit heroin found on the streets of India is frequently adulterated with 'diluent powders' to increase the quantity and reduce the price. These include mannitol, lactose, dextrose, talc, quinine, caffeine, strychnine, etc. This sort of adulteration is sometimes referred to as 'cutting'.
- Brompton's cocktail is a mixture of morphine, cocaine, chlorpromazine and alcohol used to alleviate severe and intractable pain (as in certain types of cancer). Brompton is the name of the chest hospital in England.
- 'Drug abuser's elbow': Myositis ossificans resulting due to repeated needle puncture near the elbow in the IV drug abuser.

Pethidine (Meperidine—USA)

Pethidine (phenylpiperidine) is a colourless, crystalline powder with a bitter taste. It is synthetic analgesic having a morphine-like (analgesic-narcotic) action. Pethidine usually produces miosis like all other opiates and not mydriasis. However, sometimes pethidine is more excitant than hypnotic. This is, probably, due to the action exerted by one of its metabolites, norpethidine (normeperidine). In such an eventuality, there may be mydriasis. It inhibits heart musculature. Monoamine oxidase inhibitors and phenothiazines can produce severe reactions and even death, when taken in conjunction with pethidine. (In general, 100 mg of pethidine given parenterally is approximately equal to 10 mg of morphine.)

Pethidine abuse is not common. When seen, it is mostly restricted to those having access to the drug such as medical and para-medical professionals, since it is not widely available as an illicit drug.

Methadone

Methadone, a narcotic synthesised by the Germans during World War II because of unavailability of morphine and named Dolophine after Adolph Hitler (hence the street name dollys). It was later used to minimise the discomforts of heroin withdrawal by being administered in doses for about ten days—a process termed detoxification. Methadone is a long-acting opioid that possesses almost all the physiologic properties of heroin. The plasma half-life for morphine ranges from 2.5 to 3 hours and to more than 22 hours for methadone. Maintenance programmes with methadone and even longer acting levomethadyl acetate (LAAM) should be used in combination with education and counselling. Unfortunately, it has appeared to be quite addictive itself, albeit somewhat less lethal as compared to heroin. Its clinical uses are as an analgesic more powerful than morphine, with the advantage that it is almost as potent by oral administration as by injection.

Many deaths occur from its misuse. Experience in Scotland (1995) has shown that deaths from methadone may exceed those from heroin, for which it was intended to replace. The findings at autopsy in case of acute methadone intoxication will be those associated with respiratory depression. It has been reported that toxicological data for autopsy specimens showed that the unchanged drug and major metabolite are found in the bile and urine in higher concentrations than the blood and that the relationship between liver, lung and kidney concentrations reflect the time interval between administration of the drug and death. (The urinary excretion of the unchanged drug is well-documented. It is the major pathway that is influenced by urinary pH value and is dose-related.)

38 CHAPTER

Alcohol and Alcoholism

After going through this chapter, the reader will be able to describe: Source and consideration in various preparations | Consumption, absorption and elimination of alcohol with medicolegal aspects | Stages of alcohol intoxication with fatal dose and fatal period | Alcohol withdrawal syndrome | Drunkenness and its medicolegal aspects | Death in acute alcoholic poisoning | Collection and preservation of blood and urine samples | Alcoholism and drug dependency | Medicolegal examination in a case of alcohol intoxication

Alcohol

The word 'alcohol' comes from the Arabic 'alkohl', which is a fine metallic powder used in the East to stain the face and eyelids. The name was later extended to mean any powder produced by titration and sublimation, and then to a fluid obtained by distillation. The word 'whisky' was derived from the Gaelic 'uisgebeatha' meaning **'water of life'**. The transition from ancient medicine to biological toxin did not occur until when the great anatomist first described the association of hepatic injury and alcohol in 1543.

From the point of view of chemist, the word alcohol refers to a large group of chemical compounds characterised by the possession of a functional –OH group. In common usage, however, the word alcohol has been accepted as indicating a specific compound, i.e. ethanol, having chemical formula C_2H_5OH.

From the medicolegal viewpoint, there is perhaps no other chemical substance more frequently involved as a contributory or causative factor in violent or natural deaths and also in many nonfatal incidents. It acts as an adjuvant to many other toxic substances, combining to result a fatal outcome where often the other drug alone would not have caused death. Because of this ubiquity, it is wise to consider various angles of this wonder substance separately, with particular emphasis on medicolegal issues encountered in usual practical experience. Here, it may be mentioned that the terms 'alcoholism', 'alcoholic', etc. are not pleasant terms to describe a person's drinking habits, as it implies stigma to the affected person. It is preferable to use the term **'alcohol misuse'** to indicate excessive or repeated drinking, which may lead to social, psychological or physical problems. **The most sensitive markers of alcohol misuse** are the serum concentration of the enzyme gamma glutamyl transferase (gamma GT) and the red cell mean corpuscular volume (MCV). Measurement of gamma GT activity is the best screening test available. The activity of gamma GT returns rapidly to normal after abstaining from alcohol and therefore may be misleading if measured after about 48 hours after the last drink. The MCV (high-normal or slightly elevated value, for example, ≥91 fL) on the other hand, takes up to about 3 months to return to normal after abstinence, reflecting red cell turnover by bone marrow. Other indicators may include serum uric acid (more than 7 mg/dL), carbohydrate-deficient transferrin (CDT ≥20 gm/L) and triglycerides (≥180 mg/dL).

Absolute alcohol (alcohol dehydratum) contains 99.95% of alcohol. Rectified spirit contains 90% by volume of alcohol, and industrial methylated spirit or denatured alcohol is a mixture of alcohol 95% and 5% of wood naptha.

ALCOHOL CONCENTRATIONS (UNITS AND VARIOUS DRINKS)

The concentration of alcohol in blood, urine and breath is expressed by a variety of metric units. The most widely used for blood, urine and other body fluids is the weight of alcohol per volume of diluent (milligram per hundred millilitres, i.e. mg/100 ml). The expression 'decilitre' may be used instead of 100 ml (mg/dl). In the United States, percentage system is common and usually assumed to be a weight/volume. Breath is almost universally measured as micrograms per hundred millilitres (μg/100 ml). In alcoholic drinks, the manufacturer's description and labelling is almost always 'volume by volume' (v/v), but physiological calculations are usually made via the weight of alcohol in a given volume of body fluid (w/v).

Approximate strengths of common drinks are (v/v): 1/2 pint beer (3–8%), 1 measure of spirits (brandy, gin, whisky, rum, vodkas, etc.) (40–55%), 1 glass of wine (8–14%), 1 glass of sherry (17–23%). [A 'unit' is a common measure of alcohol and approximately contains 8–10 gm of alcohol, which is equivalent to half a pint of beer, a single measure (30 ml) of spirits, or a glass of table wine.]

Arrack is an eastern name for any country liquor, especially that distilled from the coco palm or from rice and sugar or from jaggery. Its strength may be as high as that of whisky. In addition, it may be fortified with powerful knockout agents like potassium bromide, chloral hydrate, methyl alcohol, *dhatura* or *bhang*, etc.

CONSUMPTION, ABSORPTION AND ELIMINATION WITH THEIR MEDICOLEGAL IMPLICATIONS

Alcohol almost invariably enters the body by ingestion of an alcoholic beverage. Alcoholic beverages are primarily a mixture of alcohol and water with small amount of other substances, which impart characteristic tastes and odours to the various beverages. These substances are referred to as congeners, since they are simultaneously produced during the process of fermentation. The odour may persist in the tissues for several hours after all alcohol has been metabolised. Hence, the conclusion of presence of alcohol based upon odour may be erroneous.

Alcohol requires no preliminary digestion and is capable of being absorbed by any part of the gastrointestinal tract. The upper small intestines, the duodenum and the jejunum have the maximum capacity for absorption, followed by the gastric mucosa. A fatty meal will slow the process of absorption and a milk feed too has a marked delaying effect. A full stomach will retard absorption by mixing with the alcohol and physically reducing its access to the gastric lining where transit into the blood takes place.

Another factor in influencing the speed of absorption is the concentration of alcohol. Strength of about 20% is the optimum for rapid absorption. Carbonated drinks (those containing dissolved carbon dioxide, such as champagne, beer and whisky with soda or lemonade) hastens absorption, possibly because the bubbles greatly increase the surface area carrying alcohol. Dilute drinks like beer are absorbed slowly, probably the large volume hinders access of the alcohol molecules to the stomach lining.

Quality and extent of aqueous compartment of the body also matters as the ethanol is a small molecule which is easily miscible with water and quickly diffuses through the whole of aqueous compartment of body. The fact that ethanol is almost insoluble in fat has an important practical bearing, particularly in case of women, who have large fat deposits, they may develop blood alcohol concentrations at least 25% higher than men of the same body weight after similar drinks. It is due to this factor that the aqueous humour of eye is in equilibrium with blood, as is the cerebrospinal fluid.

Taking the stomach to be empty and concentration of alcohol to be optimum, maximum blood alcohol concentrations are usually reached within 30–90 minutes. Rates vary greatly among different persons and even in the same person at different times under different circumstances, with the majority of the individuals reaching their maximum about an hour after ingestion.

Absorbed alcohol is carried from the gastrointestinal tract via the portal vein to the liver. This is why the alcohol concentration in the portal vein blood exceeds the concentration of blood elsewhere in the body during the phase of active absorption. Later, the portal vein blood alcohol level decreases as the alcohol is lost to the liver for metabolism and mixing of the alcohol with the blood from the hepatic artery. Ultimately, the blood containing alcohol approaches the heart through inferior vena cava and gets circulated through the body.

As the arterial blood passes through the lungs, some alcohol is lost by diffusion into the alveolar air. The basis for breath analytical methods is that there is a constant partition factor between the alcohol in the blood in the pulmonary circulation and the alcohol in the alveolar air. However, during the phase of active absorption, there may be considerable difference between the concentration of alcohol in the arterial blood and that in the venous blood. This is responsible for the difference sometimes observed in the determination of blood alcohol level by analysis of alveolar breath compared with direct analysis of a sample of venous blood.

When equilibrium has been reached, the distribution of alcohol in the various tissues and body fluids is as follows: whole blood (1.00), plasma or serum (1.12–1.20), brain (0.85), spinal fluid (1.10–1.27), urine (ureteral) (1.3), alveolar air (1/2100) and liver (0.85).

More than 90% of alcohol that has been absorbed is eliminated from the body as a result of oxidation effected by liver. In addition to oxidation, alcohol may be eliminated unchanged through kidneys, lungs, sweat glands and colon. Usually, excretion by way of faeces is relatively insignificant and by way of sweat glands, at about one half the rates of elimination by way of urine. However, in excessively hot climates or excessive perspiration resulting from heavy exercise, loss of body water by way of the skin may increase enormously. This would lead to corresponding increase in the loss of alcohol through the skin. The ethanol in glomerular filtrate is in equilibrium with plasma, but as water is absorbed in the renal tubules, the urine concentration is higher than the blood level at the time of filtration, the ratio being approximately 1.23 to 1.00.

Alcohol is eliminated from the body mainly by oxidation, and only a small proportion (not more than 10%) is eliminated by excretion, chiefly in the urine and breath, and to a lesser extent in sweat and saliva. Two most important pathways of metabolism are discussed here.

The first major pathway involves two steps. The first step in the process of oxidation is the breakdown of ethyl alcohol to acetaldehyde by the enzyme alcohol dehydrogenase (ADH), the coenzyme nicotinamide dinucleotide (NAD) acting as a hydrogen acceptor: $C_2H_2OH + NAD^+ \rightarrow CH_3CHO^- + NADH + H^+$

[ADH, which is present in the body from birth in the liver, kidney and retina, has a high affinity for higher alcohols whose presence, even in small amounts, will retard oxidation and elimination of ethyl alcohol. Acetaldehyde, which is a product of this first stage in oxidation, is a toxic substance, but it is rapidly destroyed by aldehyde dehydrogenase (ALDH). Each of these steps requires nicotinamide adenine dinucleotide (NAD) as a cofactor, and it is the increased ratio of the reduced cofactor (NADH) to NAD, i.e. NADH:NAD, that is responsible for many of the metabolic derangements observed after drinking.]

The next step is the conversion of acetaldehyde into acetyl coenzyme A and acetate, which is then passed from the liver into the blood. The greater part of the acetate enters the Kreb's tricarboxylic acid cycle and is broken down, chiefly in the muscles, to carbon dioxide and water with the release of energy, whilst the remainder is incorporated in the body as lipids.

Alcohol will replace other substances in energy production and will diminish the breakdown of fats and carbohydrates without affecting the breakdown of proteins. This results in serious metabolic disturbances, such as hypoglycaemia, which is usually seen after having drinking episodes. Further, it is reported that 1 gram of ethanol has approximately 7.1 kcal of energy, and a drink contains between 70 and 100 kcal from ethanol and other carbohydrates. However, these are 'empty' of nutrients such as minerals, proteins and vitamins. In addition, alcohol interferes with absorption of vitamins in the small intestine and decreases their storage in the liver. These actions affect folate (folic acid), pyridoxine (B_6), thiamine (B_1), nicotinic acid (niacin, B_3) and Vitamin A.

The second pathway occurs in the microsomes of smooth endoplasmic reticulum (the microsomal ethanol-oxidising system, or MEOS), which is responsible for 10% or more of ethanol oxidation at high blood alcohol concentrations.

Practically speaking, blood alcohol concentration is almost never static and is either rising or falling, so the amount of alcohol in the glomerular filtrate. It is being mixed in the bladder, with previous filtered urine and will also have that which is filtered later and added to it until the bladder is emptied. Therefore, it can only provide an average concentration for the time between two urinations. Another caution to be kept in mind is that the urine produced before drinking began (which was consequently alcohol free) might have already been in the bladder and will dilute the alcoholic urine. This is the ground for advising collection of two urine samples. The collection of second sample of urine is reasonable in the sense that it is the sample of urine that was most recently in equilibrium with the current blood concentration and, moreover, taking of two urine samples ideally removes any possibility of there being any factor operating against the accused. If the UAC of the first specimen is higher than the second, then the post-absorptive phase is probably well-established. However, if the ratio of UAC/BAC is less than or close to unity, this supports the contention of recent consumption of alcohol. In forensic practice, the magnitude of UAC/BAC ratio is a useful parameter to evaluate before a retrograde extrapolation is attempted.

While the rate of absorption is variable and is affected by a host of factors, **the speed of detoxification** in the liver is fairly constant and relatively independent of external influences and is therefore capable of reasonable approximation. (This is called as zero-order kinetic of metabolism. Most of the drugs are metabolised by first-order kinetic where a certain proportion of the drug is metabolised and the absolute quantity metabolised will go on decreasing as the blood level decreases.) As a generalisation, rate of metabolism of alcohol by the liver is about 9–15 ml per hour, which is equal to about half a peg of whisky. The result is lowering of alcohol in the blood by about 12–15 mg per hour. This applies to healthy adults, not habituated to drinking and includes light to moderate drinkers. However, about 10% of this is deposited in the tissues as lipids, while 10% is excreted in the urine, lungs and sweat, as already mentioned.

STAGES OF ALCOHOL INTOXICATION

From the medicolegal angle, three stages of alcoholic intoxication may be the following.

Stage of Excitement (Blood Alcohol Concentration 50–150 mg%)

There is a feeling of well-being, pleasure and some excitation. The actions, speech and emotions are less restrained due to lowering of the inhibition normally exercised by the higher centres of the brain. Accordingly, the social value of alcohol rests upon its property of knocking-out of inhibitions with the result that the individual converses well, laughs and smiles more readily or becomes angry more easily. This stage, therefore, is sometimes called as **flippant stage**. The face is flushed, conjunctivae injected, pupils dilated and sluggishly reacting to light and accommodation. Development of nystagmus may be there. (When jerking movement is in the direction of the gaze and independent of the position of the head, it is known as **alcohol gaze nystagmus** and may appear at blood levels of 50–100 mg%. It is not a constant or common sign.) The breath smells of alcohol and the pulse is accelerated. Mental concentration is poor and judgement impaired. Recall memory is often disturbed.

Stage of Incoordination (Blood Alcohol Concentration 150–300 mg%)

There occurs incoordination of thought, speech and action. Incoordination of thoughts leads to confusion and hence, this stage is sometimes called the stage of confusion. Speech becomes slurred and incoherent, and there occurs difficulty in pronouncing consonants. Incoordination of muscles leads to 'staggering gait'. Skilled movements are impaired, and the reaction time is increased. The eyes are suffused, the pupils usually dilated and sluggishly reacting to light and accommodation. The vision is blurred, or transitory double vision may be there. The mouth is dry, tongue furred and breath strongly smells of alcohol. Nausea and vomiting are common. The individual may suffer from hiccups and is untidy in his appearance (due to loss of tone of facial muscles, individual may give 'owlish' facial look). He may become morose, gay or irritable depending upon his inherent emotions. **Medicolegally, this is an important stage.** A person in charge of a vehicle may commit an accident (driving capacity deteriorates due to lack of visual acuity, tactile perception, reaction or reflex, judgement and due to over confidence). An ordinary moral individual may plunge into sexual excesses.

Stage of Narcosis (Blood Alcohol Concentration >300 mg%)

This is also known as stage of coma. The individual passes into deep sleep and responds only to strong stimuli. Dryness of mouth and tongue are frequently noted. Sometimes, there is excessive salivation. The pulse is rapid, temperature subnormal and the pupils may be contracted. However, on stimulation of the subject (e.g., by pinching his neck or face), pupils dilate initially and slowly return to their original size. This is known as **Macewan sign** and is helpful in differentiating alcoholic coma from other comatose conditions. A fine lateral nystagmus is usually present. Progressing medullary paralysis supervenes and is shown by symptoms like slow strenuous respiration, cold clammy cyanotic skin, dilated pupils, abolished reflexes, etc. Death usually occurs from paralysis of respiratory centre.

Death from acute intoxication is rare, unless large quantities have been absorbed in a short time. Recovery usually takes place after some hours of sleep with some leftover symptoms like headache, nausea, dizziness and mental irritation, etc. Most of the leftover or hangover symptoms are due to resultant hypoglycaemia and cerebral oedema. The so-called **Saturday night paralysis** occurs in this stage. (A worker who has received his salary at a weekend goes on drinking and he might assume an abnormal posture in the drunken state. This may lead to pressure on the radial nerve or sometimes pressure may be exerted on the nerve trunk when an arm hangs over a chair.) Prolonged coma due to alcohol may cause irreversible hypoxic brain damage and death. In such cases, blood alcohol level is usually low as some or even all of the alcohol in the body may have been oxidised and excreted. Low levels are also seen if the person survives for several hours after excessive drinking.

FATAL DOSE AND FATAL PERIOD

This will depend upon the age and habits of the individual and strength of the alcohol ingested. A concentration of 400–500 mg% and above of alcohol in blood is generally sufficient to cause death. The usual fatal period is 12–24 hours, though death may be delayed for some days. [The various factors concerned in affecting the time of maximum concentration and the quantity of alcohol found in the blood are (*i*) the weight of the person, (*ii*) the amount and concentration of alcohol taken, (*iii*) whether taken slowly at intervals or all in one gulp, (*iv*) the presence or absence of food (its quality and quantity), (*v*) previous addiction to alcohol and (*vi*) the amount of rest or exercise taken after consumption.]

DIAGNOSIS

Subjective measures such as odour of breath, dilatation and sluggish pupillary reaction, incoordination, slurred speech and a history of alcohol intake are helpful in documenting alcohol intoxication. [However, the smell of alcoholic drink can persist in the breath for several hours after the whole alcohol has been metabolised in the body, as it is due to nonalcoholic constituents (congeners) in the drink, which are simultaneously produced during the process of fermentation].

Blood levels of alcohol correlate fairly with symptoms in acute cases though variations are common. Alcohol concentration may be assayed in the expired breath, which correlates roughly with blood levels.

TREATMENT

- The first priority is to be certain that the vital signs are relatively stable without evidence of respiratory depression, cardiac arrhythmia, etc. Life-threatening problems require appropriate emergency care and hospitalisation.
- Gastric lavage is usually not indicated as it retrieves only a small amount of alcohol from the gut. However, if necessitated it may be performed with plain warm water or soda bicarb solution.
- Respiration is safeguarded by clearing the passage. If need be, analeptic like Nikethamide is given. If analeptic does not work, 50–100 ml of 50% dextrose may be given by slow IV infusion, along with 15 units insulin subcutaneously.
- Thiamine 100 mg IV.
- Intravenous fluids as indicated.
- A variety of drugs have been tried to hasten the elimination of ethanol or reverse its intoxication effects. Recently, flumazenil (3 mg IV) has been shown to be effective (in experimental studies) in reversing the respiratory depression.

Alcohol Withdrawal Syndrome

Once the brain has been repeatedly exposed to high doses of alcohol, any sudden stoppage/decrease in intake can produce symptoms of withdrawal. As with all CNS depressants, the symptoms are generally the opposite of those produced by intoxication. Features may include tremor of the hands **(shakes or jitters)**, agitation and anxiety, autonomic nervous system overactivity such as an increase in pulse, respiratory rate and body temperature. Insomnia and gastrointestinal upset may also be there. Anxiety, insomnia and mild levels of autonomic dysfunction may persist at decreasing levels for 6 months or so as a protracted abstinence syndrome, which may contribute to the tendency to return to drinking.

At some point of time, some alcoholics may experience withdrawal seizures, the so-called **rum fits**. These are usually generalised and generally return to normal within several days. The term **delirium tremens** (DTs) refers to delirium (mental confusion with fluctuating levels of consciousness) along with a tremor, severe agitation and autonomic overactivity, e.g. marked increases in pulse, blood pressure and respirations. DTs are most likely to develop in individuals with concomitant severe medical disorders or evidence of underlying brain damage. **Alcoholic hallucinosis** may be another manifestation wherein objects appear distorted, shadows seem to move, shouting or snatches of music may be heard. They may consist of instructions to the sufferer, which can lead to some form of a bizarre behaviour.

TREATMENT

- The most important step is to perform a thorough physical examination in all alcoholics who are considering stopping drinking. It is necessary to evaluate organ systems likely to be impaired by chronic alcohol intake.
- All individuals should be given oral multiple B vitamins, including 50–100 mg of thiamine daily for a week or more.
- Individuals can be weaned by administering benzodiazepines. The drugs with short half-lives are especially for patients with serious liver impairment or evidence of pre-existing encephalopathy or brain damage. However, as such short half-life benzodiazepines (e.g. oxazepam or lorazepam) result in rapidly changing blood levels and, therefore, need to be given every 4 hours to avoid abrupt fluctuations in blood levels that may increase the risk for seizures. Therefore, drugs with longer half-lives, such as diazepam or chlordiazepoxide are preferred.

Aversion Therapy

Many methods have been tried in this connection and one of the more successful ways is to administer a drug called **disulfiram** (available in India as Antadict/Esperal in tablets of 250 mg strength). It is disulfide molecule (tetraethylthiuram) that interferes with the oxidative metabolism of ethanol at the acetaldehyde stage, as a result of which acetaldehyde accumulates producing unpleasant symptoms in the form of flushing, headache, palpitations, vertigo, vomiting, abdominal and chest pain, hypotension, etc. Some advocate that these symptoms are actually produced by a metabolite of disulfiram (carbon disulfide) and not by acetaldehyde accumulation. Whatever may be the mechanism, the individual avoids taking alcohol as long as he is on disulfiram. Some guidelines for disulfiram therapy include the following:

- Ensure that the individual is off alcohol for a minimum period of 12 hours before starting medication and administer the drug only by oral route (250 mg per day).
- Warn the individual in clear terms that during medication, alcohol must not be consumed even in small quantity since it can provoke a severe (and sometimes fatal) reaction. (This is especially so in the individuals with heart disease, diabetes and hypertension.)
- There are side effects of the disulfiram also, like halitosis (rotten egg odour due to sulphide metabolites), pruritis, headache, drowsiness, impotence, peripheral neuropathy, depression, psychosis and hepatotoxicity.
- The drug needs to be administered under medical supervision, especially during discrete periods representing high-risk drinking situations.

Supportive Psychotherapy

Supportive psychotherapy is an integral part of the treatment. More than individualised psychotherapy, it is group therapy that is effective in the long-term management of abstinence.

Drunkenness

ETHANOL AND VEHICULAR ACCIDENTS

It is well-known that consumption of ethanol and consequent intoxication has adverse effects on the driver of a vehicle in the form of visual blurring, reduction in visual acuity (stronger illumination is often required to distinguish objects, and dimly lit objects may not be distinguished at all), decreased awareness and reaction to stimuli (differences in intensity of various stimuli are less easily distinguished, especially in case of light and touch), motor incoordination, impairment of judgement and increased reaction time. This has given rise to the adage 'Drinking and driving don't mix'. Driving a vehicle on a public thoroughfare under the influence of alcohol (or any other intoxicating drug) is an offence in almost every country of the world. In India, it is an offence punishable under Section 185 of the Motor Vehicle Act.

The Section reads as: Whoever, while driving, or attempting to drive a motor vehicle (*a*) has in his blood alcohol exceeding 30 mg per 100 ml of blood detected in a test by a breath analyser, or (*b*) is under the influence of a drug to such an extent as to be incapable of exercising proper control over the vehicle shall be punishable for the first offence with imprisonment for a term that may extend to 6 months or with fine that may extend to ₹ 2000, or with both; and for a second or subsequent offence, if committed within 3 years of commission of the previous similar offence, with imprisonment for a term that may extend to 2 years, or with fine that may extend to 3000 rupees, or with both.

BMW Hit and run/hit and skip case (as gathered from news reports): The incidence occurred at about 4.30 am early morning of January 10, 1999. The accused was returning from some late night party along with his friends. On the way, there was some police check point. However, the car was going so fast that it ran out of control mowing six people, three of them being constables. The trial of the case saw many twists and turns because of witnesses' flip-flop. Scientific evidence helping the court to arrive at the outcome were as under:

- Blood stains on the steering of the car matching the blood sample of the accused, which suggested that the accused not only got himself injured but also was driving the car.
- Broken pieces of the headlight on the right side of the car seized from the spot matching with the head lights of the car, which were found to be parts of same headlights.
- CD comprising of a video recording of the petrol marks (left by the leak in the car petrol tank) right from the scene of the accident to the house of a friend of the accused.

And finally, the accused was brought to book under Section 304 (Part II) of the IPC. The blood alcohol level of the accused determined after about 8 hours of the incident was reported to be 115 mg%.

BREATHALYSER (ALCOMETER/INTOXIMETER/ DRUNKOMETER)

In many countries including India, traffic police carry special equipment in the form of breathalysers to detect alcohol in the breath of a suspect driver. It serves as an 'on the spot test'. Presently, more sophisticated versions based on fuel-cell sensing, electrochemical oxidation, infrared photometry and microprocessors have come into use. It is now established that there is a fair correlation between the breath and the blood level, and the ratio is generally 2100:1. This is based on **Henry's law**, which states that when a volatile chemical (ethanol) is dissolved in a liquid (blood) and is brought to equilibrium with air (alveolar breath), there is a fixed ratio between the concentration of the volatile compound (ethanol) in air (alveolar breath) and its concentration in the liquid (blood), and the ratio is constant at a given temperature. However, some may argue that the sample obtained for testing is of tidal air at mouth temperature and not of alveolar air and that, according to Henry's law, the ratio of blood-alcohol to that of breath-alcohol will vary with the temperature of the sample. It has been reported that every increase of 1° in temperature of breath between 34° C and 37° C would tend to increase the estimated blood alcohol concentration by 6.5%. Therefore, 'Proper Breath Sampling' is essential to the correct analysis of the breath alcohol. This invites the collection of the end-portion of a prolonged forced expiration. This is necessary to avoid too much mixing with deadspace air, i.e. the air which is not totally in equilibrium with the blood alcohol concentration.

ETHANOL AND CRIME

Brawls, assaults (sexual and nonsexual), homicides and suicides are commonly associated with intoxication. **Section 85** (IPC) gives the same immunity to a person intoxicated involuntarily as **Section 84** (IPC) gives to a person of unsound mind, provided the person was intoxicated to such an extent as to be incapable of knowing the nature and consequences of the act. **Section 86** (IPC), dealing with voluntary drunkenness, imputes the same knowledge to such a man as he would have had, had he not been intoxicated. No one can be permitted to wear the cloak of immunity by getting drunk, and so voluntary drunkenness is never an answer to criminal charge.

The usual form of alcoholic intoxication occurs in three stages, viz., the stage of euphoria and excitement, stage of incoordination and the stage of narcosis. Small to moderate doses of alcohol in no way affect the responsibility of the consumer as he will still retain command over his faculties. The law makes an allowance for the possibility of the mind of a person so affected by drink as to render him incapable of knowing the consequences of his act. This, no doubt, is so but such a state approaches at a late stage of intoxication when the stimulation of the central nervous system is being replaced by depression and the likelihood of a wrongful act being committed is well nigh crossed. Acts contrary to the law are usually committed during the prior two stages, particularly when the stimulation of brain is at or about its peak. During this period, the individual is often capable of appreciating the consequences of the act, though the power of resisting the impulse towards the performance of the act may not be equitable to that of a sober man. (Alcohol is a depressant of central nervous system. Its apparent stimulant effect results from depression of the higher centres of the brain, thereby releasing the lower centres from cortical control, i.e. releasing inhibitions. During this stage, the subject may overestimate his capacity to carry out physical and mental work, and may underestimate any mistake(s) made in its performance.)

The frequency of crimes committed by persons under the influence of alcohol is not due to failure to realise the nature and consequences of the acts, but to the repression of those inhibitory influences that in sober persons prevent the commission of such acts. The crimes most frequently indulged in by the drunk are those actuated by passions. Most actions actuated by passion are not naturally repugnant to man, but they are preeminently those that the normal man has learnt to restrain. Removal of the controlling forces increases the probability of their commission.

Sometimes, the issue regarding the quantity of liquor consumed by the individual surfaces in the medicolegal field. A rule of thumb is that every ounce of an 80° proof liquor will raise the BAC by 25 mg%, i.e. one peg of distilled spirit, or one glass of wine, or a quarter to half bottle of beer. Average elimination rate is 12–15 mg% per hour. As an alternative or supplement to BAC, the level of alcohol can also be estimated in the urine (UAC). To calculate the BAC from the UAC, multiply the latter by 0.66, which gives a rough estimate. For example, if the UAC is 100 mg%, the BAC would be $100 \times 0.66 = 66$ mg%. Precautions to be taken in collecting urine samples have already been stressed.

Death in Acute Alcoholic Poisoning

Death due to toxic effects of acute over-ingestion of ethanol is essentially due to respiratory and central nervous system depression, and usually involves blood ethanol levels of 400–500 mg% or high. However, one must be reminded here that a blood ethanol concentration of around 400 mg%, and a novice, nontolerant individual may die from a blood ethanol concentration as low as 200–300 mg% or lower [Jones AW, Holmgren P. *J Forensic Sci* 2003;48(4):874–9]. Further, if ethanol is combined with an opiate or other type of respiratory depressant, death can occur even in the presence of lower ethanol concentration. Still further, ethanol levels recorded in the blood may not necessarily be the highest level that the individual had achieved because he/she might have metabolised the agent to some degree during his/her comatose state, before dying. Alcohol often indirectly lead to death, some of the circumstances may be as follows:

- Alcohol is often indirectly involved in trauma, which may be of many types. The majority of the homicides are catalysed

by the aggressive behaviour generated by alcohol. Falls associated with drunkenness are frequent, and may be fatal. Vehicular accidents are the other hazards.

- Death from burns or carbon monoxide poisoning may occur in drunken persons while smoking during intoxication without exercising due care when the cigarette may ignite the bed clothes. Occasionally, gas, stove or electric heater may be knocked down during drunken staggering, leading on to a fatal fire.
- Drowning is seen rarely. Death is sometimes not caused by drowning, but by sudden vagal cardiac arrest from the shock generated by hitting of cold water against the chest/abdomen or having cold water suddenly flooded the pharynx and larynx. The drunken state appears to have sensitised the victim possibly because of marked cutaneous vasodilatation produced by alcohol.
- The individual may turn on the gas and forget to light the burners, which may lead to fatality due to vicious effects of the gas. He may electrocute himself when fumbling with a plug or a defective electrical circuit.
- Death may occur due to consumption of some other additive or synergistic drug along with alcohol, and further, it may also occur due to consumption of adulterated drink.
- A person may be killed by some poison, for which the alcohol has been used as a vehicle to mask the smell and taste of the poison. (A few cases have been conducted by the author where organophosphates were reported along with alcohol.)
- A person may be killed by inflicting injury or by way of drowning, after making him unconscious/semiconscious by giving alcohol.

Collection and Preservation of Blood and Urine Samples

COLLECTION

Here at the onset, it may be borne in mind that the normal concentration of alcohol in human tissue and blood does not exceed 0.001%. In most cases, blood is the sample selected for analysis. However, the method of obtaining and the site from where it is obtained has been the subject of discussion. Swabbing of the arm with alcohol prior to collection of sample from the antecubital fossa is not advisable. Use of swabs containing isopropyl alcohol or dry swabbing may be advocated. During autopsy, blood should preferably be collected from some peripheral vein such as femoral vein. (After death, alcohol defuses through the intact stomach wall into the surrounding blood and tissues, including the pericardial and pleural fluid.)

During autopsy, if the blood is to be collected from heart, it is preferable to use dry, clean syringe and needle, and to massage the heart gently before taking the samples so as to promote mixing of the blood in the chambers. This will prevent obtaining a sample with either a high or low haematocrit, which could lead to false high or low alcohol results, as there may be grounds for assuming that the alcohol determination in plasma is not the same as that in the whole blood.

In occasional cases, death due to trauma may be as a result of production of subdural haematoma, and the individual may survive for a varying period after sustaining the fatal injury. In order to achieve an indication as to the state of intoxication at the time of the injury, subdural blood clot will likely to represent alcohol concentration essentially the same as that in the blood at the time of fatal injury.

In putrefied bodies, caution must be exercised in interpreting blood or tissue alcohol levels, because the alcohol may be produced during the putrefactive process. Levels of 'reported alcohol' generally are in the range of 20–30 mg/100 ml of blood. Blackmore has shown that alcohol can be produced from a wide range of endogenous molecules in the body, and the supply of glucose is not necessary. Therefore, in order to decide whether the deceased was drinking or not, when the blood level is found to be low, i.e. less than 40 mg/100 ml of blood, it cannot be decided on blood level alone. In such cases, urine sample, if available, should also be obtained because urine is a far more reliable biological fluid for deciding whether a small amount of alcohol has been consumed or not. If possible, vitreous humor may be obtained as a sample of analysis. Felby and Olsen observed that the alcohol concentration in vitreous humor remains quite constant, even over a postmortem interval up to 99 hours, while Scott et al. showed that the vitreous humor alcohol remains essentially unchanged after embalming. Coe and Sherman state that the ratio of blood alcohol concentration to that of vitreous humor is 0.89 ± 0.02.

For collecting samples, a screw-capped glass bottle should be used. The container should be tightly clamped and sealed to prevent loss of alcohol by evaporation and duly labelled with particulars of the case. Rubber stoppers should be avoided as they may contaminate the sample with oxidisable substances.

PRESERVATION

Two preservatives sodium fluoride and phenyl mercuric nitrate have been used in blood and urine, respectively, as alcohol stabilisers. The sodium fluoride performs the function of enzyme inhibition and prevention of clotting of the sample. Phenyl mercuric nitrate has the property of bacterial inhibition. It is preferable to use a combination of sodium fluoride (10 mg/ml) and potassium oxalate (5 mg/ml). The concentration of ethanol in plasma and serum is higher than in whole blood. Samples preserved in this manner will maintain their alcohol concentration for several weeks even at room temperature. Refrigeration or freezing, coupled with such fluoride treatment, will maintain the integrity of the alcoholic contents in the blood for prolonged periods.

Table 38.1 Differentiating Features amongst Substance Dependence, Addiction and Abuse

Substance dependence	Substance addiction	Substance abuse
In the medicolegal context, substance may be denoted as a chemical or drug with potential for abuse		
It is considered as a state, psychological and/or physical, wherein a person has the compulsion to take a drug on a continuous or periodic basis, either to experience its pleasurable effects or to avoid discomfort of its absence. A higher priority is given to the drug than to other activities, obligations, etc. Tendency to increase the dose (due to development of tolerance) and occurrence of withdrawal phenomenon on stopping a drug are also features under this entity.	It is considered as a state of periodic or chronic intoxication, harmful to the individual and to the society. Some important features are (*i*) craving or actual need (compulsive drive) for taking the drug and to obtain it by any means, (*ii*) a tendency to increase the dose, (*iii*) psychological (and sometimes physical) dependence upon the effects of the drug and (*iv*) occurrence of withdrawal symptoms when the drug/chemical is withdrawn.	It is considered as the improper use of therapeutic or nontherapeutic drug to affect the body and mind for nonmedical reasons. This may cover (*i*) using drugs/chemicals that were never intended to be put into the body (e.g., glue sniffing) and (*ii*) using drug at a dose level and in the circumstances/settings that can augment their potential for harm and physical dependence.

POST-SAMPLING FORMATION/ELIMINATION OF ALCOHOLS

The possibility of various alcohols being produced in the blood specimen after sampling is sometimes put forward. In theory, this may occur if the specimen was infected with bacteria and the preservative was inadequate. Microbial fermentation is a more significant problem with postmortem specimens. The presence of 1-butanol in the blood is a useful marker for postmortem synthesis of alcohols including ethanol. Conversely speaking, loss of alcohol from the blood can also occur especially during storage of specimens at ambient temperatures.

Alcoholism and Drug Dependency

The older terminologies like 'drug addiction' and 'drug habituation' have been replaced by the term 'drug dependence' by World Health Organisation (WHO). However, the older terms are still in use, especially the 'drug addiction', and the person falling prey to it is often labelled as 'drug addict'. This necessitates clarifying such terms so that one gets conversant with medicolegal implications likely to be encountered in the realm of drug jargon (Tables 38.1, 38.2 and 38.3).

Wernicke encephalopathy is a known neuropathological feature of alcoholism. Most of the features associated with the Wernicke–Korsakoff syndrome, including ophthalmoplegia, ataxia, nystagmus and mental features like confusion, disorientation, etc., can be related to damaged systems in the hypothalamus, midbrain and cerebellum. As reported, about 80% of alcoholic patients recovering from Wernicke encephalopathy develop **Korsakoff syndrome**. Korsakoff described this condition as resulting from alcohol abuse or from a number of other causes. However, these days, by far the most common cause is the alcohol abuse leading to nutritional depletion, notably thiamine. This syndrome is characterised by marked deficits in anterograde and retrograde memory (learning over more prolonged periods is severely impaired and there is usually a

Table 38.2 Differentiating Characteristics of Drug Addiction and Drug Habituation

Characteristics	Drug addiction	Drug habituation
Compulsion	Present	Only desire, no compulsion
Dependence	Both psychological and physical	Psychological (mild)
Dose	Tendency to increase	Not so
Harm	To both victim and society	If any, to victim only
Withdrawal symptoms	Characteristic	None/mild

Note: Although the differences are shown in tabular columns, it is extremely difficult to evaluate that when the habituation can get transformed into addiction, imparting both entities the same status, i.e. drug dependence as advocated by WHO.

Table 38.3 Differentiating Characteristics between Physical and Psychological Dependence

Physical dependence	Psychological dependence
Body needing the drug to function properly.	Mind needing the drug to feel normal.
Physiology of the body is altered to such an extent that the cells can function satisfactorily when such drugs are continued.	Functioning of the mind is altered to such an extent that it leads to compulsive craving for the drug to satisfy emotional or personality needs.
Occurrence of withdrawal symptoms (especially physical pain) on stoppage of drug.	Disturbance in personality, feelings and comforts, etc. are manifested when the drug is discontinued.

retrograde memory loss, which characteristically extends back many years), an intact sensorium, apathy and relative preservation of other intellectual abilities. Confusion in remembering the temporal sequence of events associated with severe disorientation in time was a conspicuous finding in Korsakoff's patients. The typical pathology in such cases resided in neuronal loss, microhaemorrhages, and gliosis in paraventricular and periaqueductal grey matter.

Genetic factors in drug dependence have also been studied. Some genes confer a reduced risk for alcoholism. Approximately half of all individuals in Asian populations carry an allele of aldehyde dehydrogenase that encodes an isozyme with reduced enzymatic activity. A recent multicentre study of 105 families of alcoholics revealed evidence for susceptibility loci for alcohol dependence on chromosomes 1, 7 and possibly 2. An additional study of a Southwestern Native American population found evidence for genetic linkage to alcohol dependence on chromosomes 4 and 11.

Methanol (Methyl Alcohol/Wood Spirit/ Wood Naphtha/Colonial Spirit)

Methanol or methyl alcohol is a colourless liquid with a faint spirituous odour and a burning taste. It mixes with water in all proportions. It is obtained by destructive distillation of wood or molasses. It is used for denaturing rectified spirit so as to render it nondrinkable. Such rectified spirit mixed with 5% methyl alcohol is known as methylated spirit, and is used in arts. In spite of its nauseating taste, such spirit is not infrequently consumed by poor sections of society on account of its cheapness and easy availability. Surgical spirit consists of 95% ethyl alcohol and 5% methyl alcohol, in which oil of wintergreen is added to give it a sweetish flavour, for easy detection and pleasant taste.

MODE OF ACTION

Methanol as such is not a toxic agent. In the liver, methanol is metabolised to formaldehyde (by alcohol dehydrogenase) and then to formic acid (by aldehyde dehydrogenase), and these metabolites of methanol are responsible for its retinal toxicity as well as metabolic acidosis. Though its action resembles that of ethyl alcohol to a great extent, its rate of oxidation is one-fifth that of ethanol, and with repeated small doses, it tends to accumulate in the blood. Dangerous level is considered to be 80 mg/100 ml of blood. As it is metabolised and excreted slowly, it remains in the body for several days.

CLINICAL FEATURES

Symptoms may appear within an hour but are commonly delayed for several hours. The earliest manifestations include vertigo, headache with stiff neck (meningismus), nausea, vomiting and pain or cramps in the abdomen. (Finding of intestinal contraction is considered to be an important finding of methanol poisoning, it being described as resembling a thick pipe of narrow lumen.) There occurs marked muscular weakness and depressed cardiac action.

There may be an apparent relief for 12–24 hours, after which condition worsens. Most signs and symptoms return. By this time, blurring of vision or misty vision **(snowfield vision)** develops. Visual disturbances like photophobia, concentric diminution of visual fields for colour and form, followed by partial or total blindness due to retrobulbar neuritis resulting from specific toxic effect of formaldehyde on retinal cells are the peculiar findings. Ophthalmological examination usually reveals dilated pupils with sluggish reaction to light. There is usually restlessness, cyanosis, low BP, hypothermia and dehydration. Urine is scanty and contains acetone and formates. Death is due to respiratory failure. Terminal convulsions may precede death. Peculiarly, some persons can stand high dose of methanol without any significantly damaging effects.

MANAGEMENT

- Gastric lavage if the patient is seen within 2–4 hours after ingestion. (It needs be carried out with 5% solution of sodium bicarbonate in warm water. Activated charcoal reduces the mortality. It acts by reducing the absorption of alcohol from the digestive tract, and by creating a concentration gradient in favour of movement of alcohol and its metabolites back into the gut.)
- Combat acidosis by administering sodium bicarbonate. Bicarbonate is essential if the pH <7.35 or bicarbonate <15 meq/L.
- Ethanol is the specific antidote since it preferentially competes for the same enzyme (alcohol dehydrogenase) and effectively shuts off the conversion of methanol to formate and permits the remaining methanol to be eliminated intact via the kidneys and lungs. It may be administered as 10% ethanol at a dose of 10 ml/kg IV over 30 minutes followed by 1.5 ml/kg/hr, so as to produce and maintain a blood ethanol level of 100 mg/100 ml. Alternatively, 1 ml/kg of 95% ethanol in fruit juice can be given orally over 30 minutes.
- The eyes should be kept covered to protect them from strong light. Opinion of an ophthalmologist should be sought.
- Haemodialysis is very effective in removing methanol, formaldehyde and formic acid.
- Maintenance of respiratory and cardiac functions as warranted.

FATAL DOSE AND FATAL PERIOD

Usual fatal dose is 70–140 ml. Fatal period is usually between 24 and 36 hours. However, it may be delayed for 3–5 days.

POSTMORTEM APPEARANCES

Cyanosis is marked and blood may fail to clot. Prominent postmortem staining is the usual feature. There may be frothing from the mouth and/or nostrils. Mucous membrane of

stomach and duodenum is congested and inflamed. Lungs and brain are congested and oedematous. The mucosa of the urinary bladder is often congested. The liver may show fatty change and kidney, tubular degeneration. Retina and optic disc usually show degenerative changes. (Viscera need to be preserved in saturated solution of common salt. In addition to routine viscera, it is advisable to preserve one cerebral hemisphere also.)

MEDICOLEGAL ASPECTS

Most of the cases of methanol poisoning are accidental arising out of taking contaminated liquor containing methanol or its products. Sometimes, intentional adulteration of ethanol (especially *arrack*) has been responsible for mass deaths.

Medicolegal Examination in Case of Alcohol Intoxication

Report No. _____
Date and Time of Examination _____
D.D.R./FIR no. _____ U/S _____
Police Station _____
Name _____
S/D/W of _____
Resident of _____
Age _____ Sex _____ Caste _____
Occupation _____
Brought by _____
Consent: I, _____ S/D/W/of _____
R/o _____

give my full, free and voluntary consent for my medical examination and necessary investigations. The nature and consequences of the examination have been explained to me.

Date, Time and Place

Signature/Thumb
Impression of the Patient

Note:

- If the subject is too drunk to understand or give consent, then only the results of the observations can be provided to the police after giving a due 'note' as to his condition.
- If the patient refuses consent, examination may still be conducted provided (*i*) he has been arrested for some offence and (*ii*) the request is from an official not below the rank of a Sub-inspector of the Police (Section 53 of CrPC).

Identification Marks: At least two marks to be recorded with full details.

History: History of relevant incident as stated by the person or the accompanying relative/friend/attendant. Time, place, amount, kind of food, etc. relating to the incident. History of any disease/medicine intake.

EXAMINATION PROPER

- Skin: dry/moist/dirty-stained, etc.
- Clothes: decently dressed/disordered/soiled/torn.
- General disposition: calm/talkative/abusive/aggressive.
- Smell of alcohol from breath/mouth: present/absent.
- Speech: normal/thick/slurred/over precise/incoherent.
- Gait: steady/controlled/unsteady/staggering.

Eyes and Visual Acuity

Conjunctivae (suffused or not), nystagmus (presence/absence of involuntary rapid movement of the eyeball, which may be horizontal, vertical, rotatory or mixed), strabismus/squinting (presence/absence of deviation of the eye that the patient cannot overcome. The visual axes assume a position relative to each other different from that required by the physiological conditions. The various forms of strabismus are spoken of as 'tropias', their direction being indicated by the appropriate prefix, as cyclotropia, exotropia, hypertropia and hypotropia, etc.), pupils (dilated/constricted), pupillary reaction to light (prompt/sluggish).

Vital Parameters

Pulse, blood pressure, temperature and respiration.

Reflexes

Normal/brisk/sluggish, reaction time whether normal or delayed (time taken to react to a question or to other stimuli, ask the person to walk across the room with the instructions that he should turn about in response to a signal/verbal command).

Muscular Coordination Tests

- **Buttoning/unbuttoning of the shirt:** can do/cannot do.
- **Finger nose test:** can perform/cannot perform (stretch your left hand in front of you. With the right hand middle finger, touch the middle finger of left hand first and then the tip of your nose, asking the person to follow you. Repeat as quickly as possible, while observing the person).
- **Picking up of a coin from the floor:** can pick up/cannot pick up.
- **Walking on a straight line:** can walk/cannot walk.
- **Ramberg Test** (Stance: Presence or absence of swaying when standing erect with his feet together and eyes closed.)
- **Handwriting/copying simple geometric figures** (in assessing the ability to write, the doctor should allow for apparent social and educative standard of the person. Many may be unable to write well or at all, even when sober).

Mental Examination

Orientation to time and space (oriented/disoriented), cooperative/non-cooperative, memory of recent/remote events (normal/impaired), etc.

Table 38.4 8 Ds of Acute Alcoholic Intoxication based on Blood Alcohol Concentration

Blood alcohol concentration (mg/100 ml)	Behaviour	Detailed features
0–50	Dry and decent	Usually not noticeable. Behaviour almost normal.
50–150	Delighted and excited	Talkativeness, sociability, increased self-confidence, lowering of inhibitions, etc. Smiles more readily or becomes angry more easily (flippant behaviour). Impaired attention, judgement and control, etc.
100–200	Disoriented and disturbed	Emotional instability, decreased inhibitions, etc. Incoordination, mild ataxia. Impaired memory, reaction time, critical judgement, etc. Dilated sluggish pupils.
150–250	Dizzy and confused	Dizziness, confusion. Much impaired memory, reaction time, critical judgement, etc. Aggravated ataxia. Dilated sluggish pupils.
200–300	Dazed and confused	Disoriented, confused. Dilated much sluggish pupils. Incoordination of thought, speech, action, etc. Dysarthria.
250–350	Dazed and dejected	Dazed, dejected. Disturbed perception, sensation, etc. Pupils inactive (contracted or dilated). Marked ataxia. Dysarthria. Marked incoordination of thought, speech, action, etc.
300–400	Stupor to coma (Deadly drunk)	Apathy. Unconsciousness. Incontinence. Inability to stand and walk. Incoordination approaching paralysis. Depressed or abolished reflexes. Subnormal temperature. Embarrassment of circulation and respiration, etc.
400–500 and above	Comatose to Death	Unconsciousness, anaesthesia, abolished reflexes, hypothermia, hypotension, hypoventilation. (Progressive danger of death due to respiratory failure)

Note: The Table is intended to provide a general guideline of clinical manifestations in relation to blood alcohol levels. Overlapping ranges suggest that alcohol acts differently on different individuals and so also on the same individual at different timings/settings. Circumstances attending each case need consideration/evaluation.

Systemic Examination

All systems to exclude any co-existing disease condition.

Examination of Injuries

If any (especially look for head injury).

Collection of Samples

Blood (preservative: sodium fluoride 10 mg/ml and potassium oxalate 5 mg/ml), urine (preservative: phenyl mercuric nitrate). (Precautions to be exercised while collecting samples have already been explained under the relevant description.)

OPINION

The term 'under the influence' indicates that a person's behaviour is reflecting the disorganising functional changes produced by the alcohol on his central nervous system. The term 'under intoxication' may be avoided for its stigmatising gesture.

- Smell of alcohol, congestion of conjunctivae, dilatation of pupils, normal reaction of the eyes and normal muscular coordination. The person has consumed alcohol but is not under its influence.
- Smell of alcohol, definite signs of muscular incoordination, dilated pupils with sluggish reaction to light, slurred incoherent speech, staggering gait and inability to perform other tests as described above, impaired mental tests, etc. The person has consumed alcohol and is under its influence.

[Alcohol acts differently on different individuals and so also on the same individual at different times. Laboratory Tests being objective provide some safeguards. The tests will also obviate any argument relating to the so-called psychological and physiological alarm-reaction of the patient's predicament tending to have some influence upon his clinical performance. This reaction may effect either way, i.e. the reaction as that caused by being arrested or by being mixed up in an accident may have a sobering effect on a person not too deeply under the influence of alcohol so that he may sometimes be able to so pull himself as to pose better as compared to his true state and, on the other hand, an irritable, short-tempered individual, though sober, may be so incoherent with indignation and excitement at being suspected of drunkenness that his manner and behaviour may well be suggestive of that condition (Table 38.4).]

39 CHAPTER

Non-narcotic Drug Abuse

After going through this chapter, the reader will be able to describe: Methods and hazards of non-narcotic drug abuse | Toxicology and medicolegal aspects of barbiturates, amphetamines, tricyclic antidepressants, benzodiazepines, hallucinogens, cocaine and Cannabis, etc. | Drug abuse in sports

Non-narcotic Drugs

Abuse of non-narcotic drugs, often referred to by the somewhat misleading term **soft drugs** to distinguish them from the narcotics (the so-called **hard drugs**), is much more extensive than opiate abuse. These drugs or substances of abuse can be classified into **three main groups:** those that alter perception, those that stimulate the brain and those that depress it. Inevitably these groups overlap to some extent. Substances that predominantly depress the CNS often arouse and disinhibit behaviour before exerting their main action (e.g. ethanol and barbiturates) while, conversely, drugs that initially stimulate the brain can impair consciousness if taken in sufficient quantity. For simplification, the drugs/substances may be studied in the following groups:

- **Hypnotics (downers):** barbiturates.
- **Stimulants (uppers):** amphetamines.
- **Antidepressants:** tricyclic antidepressants.
- **Hypnotics, sedatives and tranquilisers:** benzodiazepines.
- **Hallucinogens:** lysergic acid diethylamide (LSD), Cannabis, cocaine, phencyclidine (PCP) and various hydrocarbon preparations, etc.

METHODS OF ABUSE

Virtually every possible route of drug absorption has been used for substances that are abused. The method of choice depends to a large extent on the nature of the substance and the desired intensity of effect on the brain of the user. However, absorption from the gut is relatively slow, and the impact of the drug on the brain correspondingly muted unless large quantities are taken. The speed of absorption can be increased by inhaling the substance, but this is possible with only a small range of materials. Nicotine from tobacco is the most common example. Absorption through the skin or mucous membranes is not a popular route except in the case of cocaine. Cocaine is commonly dissolved and the solution sniffed up into the nostrils where part is absorbed, a technique commonly referred to as **snorting**. Injection is the fastest method of getting drugs to the brain in high concentration. Subcutaneous injection is known as **skin popping** and that into veins as **mainlining**. The latter is the preferred route for the most potent opioid analgesics.

HAZARDS OF DRUG ABUSE

Accidental Overdosage

Drug abusers seek intoxication with the substance of their choice, but the amount taken is usually arbitrary and the potency may vary considerably from one time to another and from one source to another. Accidental overdosage is a constant risk, less with some routes of administration than others. Not surprisingly, intravenous injection carries the greatest risk of accidental overdosage and once done, the drug cannot be retrieved. Drug pushers are likely to 'cut' (dilute) what is sold on the streets when supplies of high quality drug fall, and during this period, users may increase the amounts they inject so that they can continue to experience a 'rush'.

Delayed Presentation

Since abuse is often illegal and the intoxication is the objective, drug users are usually anxious not to attract attention to them. Mild to moderate poisoning is therefore unlikely to come to medical notice; on occasions, even individuals who become unconscious or seriously ill in other ways may not be referred for medical help immediately.

Contaminants

Some of the apparent applications of drug misuse may not be due to the primary drug but to the substances such as talc, which are often used to cut its price before it is sold to the user. In other cases, contaminants are the result of the way in which the drug is prepared for injection. These are the causes for long-term, progressive, granulomatous pulmonary lesions, while others, particularly quinine, may be responsible for some of the more acute toxic phenomena and possibly, even deaths.

Non-drug Hazards

The non-drug related hazards of drug abuse are well-known. They include infections with hepatitis B and HIV; bacterial infections causing skin abscesses, thrombophlebitis and endocarditis, moniliasis and inadvertent intra-arterial injection leading to gangrene.

Body Packing and Stuffing (Table 39.1)

A potential source of poisoning and death unique to illicit drugs is the phenomenon of body packing and stuffing. This refers to the practice of smuggling these drugs by making them up into small packets, which are then swallowed for later retrieval from vomitus or faeces. The individuals involving into such practice are called **swallowers**. The packets may be inserted into the vagina or rectum, and the individuals in that case are called **stuffers**. Such practices are usually reserved for the financially most lucrative preparations such as high quality Cannabis, cocaine, heroin and morphine. There exists a grave risk of overdosage should one or more packets burst allowing absorption through the intestinal, vaginal or rectal mucosae. The material used for wrapping is, therefore, of considerable importance. Condoms, foil, cellophane and other wrappings are used. In addition to the hazard of leaching out of the drug while traversing the gut, the body packer is at risk of acute intestinal obstruction. **Abdominal radiography** may yield information about the physical characteristics of the wrappings which, together with the nature of the drug contained therein, is crucial in deciding whether a conservative or a surgical approach needs be adopted.

Table 39.1 Differentiating Features of Body Packer and Body Stuffer

Features	Body packer	Body stuffer
History	Specifically hired to smuggle drugs	User or seller on verge of arrest swallows the evidence
Packing	Carefully packed in plastic or latex packages	Not so, usually due to non-availability of time
Co-ingestants	Usually not present	Usually present
Toxicity	Rarely occurs (due to bursting of packages)	Relatively common
Radiography	Usually helpful in detection	Not always helpful
Treatment	Observation: rarely surgical removal. Emergency intervention as needed	Aggressive treatment often required

Body Packing—An Unusual Case

Couriers agree to swallow packets of drugs in one country and transport the same to the other country. (For this purpose, the drug is usually compressed into cylinders, sealed in plastic film, and wrapped in multiple layers of latex—perhaps condoms, balloons, or even the rubber gloves). At the end of journey, laxatives are consumed and the packets retrieved. However, the danger of rupture of the packet always exists wherein the large amount of drug will find its way into the circulation (even if packets do not rupture, osmotic seepage across the latex wrapping may allow some amounts of drug to appear in the circulation). Cases of intestinal obstruction have also been described. **In living individuals,** diagnosis of 'body packing' may be difficult to make as the radiodensity of heroin and cocaine is very close to that of stool and therefore, packets may not be immediately evident on X-ray. However, they can be easily demonstrable through CT scanning or barium contrast studies. **In cases where the courier dies** during the journey or after reaching the target, a thorough investigation of the scene plus detailed autopsy will be rewarding. **An informative case** has been reported in the literature wherein a naked body of a young man was found at an illegal dump-site. Nearby were found blood stained sheets and a large black garbage bag. The bag was found to contain loops of small bowel, some blades, and the ruptured latex packets. A gaping longitudinal midline wound was noticed running through the entire length of the abdomen. Autopsy disclosed lack of a vital reaction along the edges of the wound, indicating its postmortem origin. Circumstantial and other evidence revealed that the young man was a body packer or drug mule who had swallowed an unknown number of latex-wrapped packets of heroin in Colombia before flying to the United States. However, he died before he could pass all of the packets from his gastrointestinal tract. Those concerned with receiving of packets cut open the abdomen, extracted the loops of small intestine and milked whatever packets were palpable out of the bowel. Toxicology showed heroin toxicity.

Barbiturates

Barbiturates are sleep-inducing drugs. Their use as sleeping tablets and general soporific sedative agents led to widespread abuse, so that at one time they were easily the most common agents of drug addiction. The development of nonbarbiturate hypnotics such as benzodiazepines helped to remove the need for the older and more lethal compounds. Unfortunately, barbiturates

are still available and either alone or in combination with other substances can be the source of intoxication. Depending upon the duration of action, barbiturates may be divided into four groups:

Long-acting (onset of action about 2 hours and duration 6–12 hours): barbital, mephobarbitone, phenobarbitone, and primidone.

Intermediate-acting (onset of action half to 1 hour and duration 3–6 hours): amobarbitone, aprobarbitone, butobarbitone.

Short-acting (duration of action <3 hours): hexobarbitone, pentobarbitone, secobarbitone.

Ultrashort-acting (onset of action immediate and duration <15–20 minutes): thiopentone, methohexitone.

ABSORPTION, DISTRIBUTION AND ELIMINATION

They are rapidly absorbed from the gastrointestinal tract including the rectum. They are concentrated in the liver for a short time and then distributed into the body tissues and fluids. They are partly destroyed in the liver and excreted in the urine. The excretion is slow and may remain up to a week. With alcohol, there is an additive action. With chlorpromazine, there is potentiation that may be very dangerous. All sedatives, tranquilisers, anticonvulsants, hypnotics and analgesics are synergists of barbiturates.

FATAL DOSE

Long-acting, 4–7 gm; intermediate-acting, 2–3 gm; short- and ultrashort-acting, 1.5–2 gm. (Plasma levels of 3.5 mg/dl for short-acting and 10 mg/dl for long-acting barbiturates are indicative of serious toxicity. It may be mentioned that most of the quantitative assays just measure barbiturate moiety and do not differentiate between the various barbiturates. Deterioration of symptoms may sometimes occur due to delayed absorption caused by the formation of concretions of the drug in the gut. Much lower levels may be found in fatal poisonings by short-acting barbiturates as the death may occur more quickly from the usual mode of action, a central depression of the respiratory centres).

SYMPTOMS AND SIGNS

Impairment of consciousness, respiratory depression, hypotension and hypothermia are typical of barbiturate poisoning and, in common with all forms of hypnotic overdose, are potentiated by alcohol and benzodiazepines. Hypotonia and hyporeflexia are the rule and the planter responses are either flexor or absent. Hypotension is due not only to depression of medullary centres but also to peripheral venous pooling and myocardial depression. Most deaths result from respiratory complications. However, death may occur from respiratory failure or ventricular fibrillation in early stages.

TREATMENT

- Respiration has to be safeguarded by keeping the airways clean and if need be, by using endotracheal tube.
- There is no specific antidote. Analeptics stimulate central nervous system, especially the respiratory centre: (*i*) amphetamine sulphate 20 mg IV may be tried, (*ii*) cardiazol 5 ml IV initially and the dose may be increased depending upon the circumstances, (*iii*) 15 mg of amiphenazole in saline and 50 mg bemegride are added to the drip as indications arise. Analeptic therapy should be avoided unless a clear and compelling need is warranted.
- If the patient is not in coma, stomach wash should be carried out.
- In long-acting barbiturate poisoning, purgatives may be given for elimination from the intestine.
- Forced alkaline diuresis is most useful in poisoning by barbiturates, which are not protein bound like phenobarbitone, allobarbitone and barbitone. Forced diuresis is brought about by mannitol (100–200 ml of 25% solution) followed by an infusion of half litre of 5% solution during the next 3 hours. It can be continued alternative with 5% dextrose for the next 24 hours so as to maintain a urine volume of 10–20 litres in that period.
- Haemodialysis is advised for severely poisoned patients (blood levels >10 mg/dl for phenobarbital and >5 mg/dl for short-acting barbiturates).
- In patients with prolonged coma, miniheparinisation, elastic stockings, etc., are useful in preventing deep vein thrombosis and thromboembolism.

POSTMORTEM APPEARANCES

They are not characteristic, but are mainly those of asphyxia. Cyanosis is usually present. Postmortem staining may be prominent. In a few cases, there may be skin blisters, the so-called **barbiturate blisters**. They are commonly found at the sites where pressure has been exerted between the skin surfaces, such as buttocks, backs of thighs, calves and forearms. They are the result of cutaneous oedema and may be found in any deep coma where there has been immobility and lack of venous return from muscle movement. Some of the blisters may burst leaving a red, raw surface which later dries to a brown parchment-like area. **Internally,** some white particles of ingested barbiturate may be seen in the stomach. Gastric mucosa may be eroded. The cardiac end and lower oesophagus may be eroded from regurgitation. The lungs are congested, oedematous and may show petechial haemorrhages on the pleura. The organs in acute barbiturate poisoning are quite intensely congested and may be almost black, and the whole venous system may be engorged with dark, deoxygenated blood. Kidneys may show tubular degeneration. Brain is congested, oedematous with punctate haemorrhages. **In delayed deaths,** there may be necrosis of globus pallidus and corpus callosum, focal areas of necrosis in the cerebrum and cerebellum. Putrefaction causes decrease in blood barbiturate levels. Barbiturates, alcohol and CO produce irreversible brain damage and yet the patient may survive for a sufficiently long period so that they are completely metabolised or excreted before death occurs.

MEDICOLEGAL ASPECTS

Poisoning is mostly suicidal. Due to large size of fatal dose and prolonged unconsciousness, they are rarely used for homicide. Accidental poisoning may occur due to overdose (particularly due to automatism or due to mixed therapy with other additive or synergistic drug). The so-called **barbiturate automatism** may occur when the patient after taking dose of barbiturate confuses and thinks he has not taken the drug. That way he repeats the dose and ultimately consumes a toxic dose in total. Intravenous thiopentone has been used as **truth serum** to extract confessions during interrogation by inducing a state of drowsy disorientation in the course of which the person may reveal the truth.

Amphetamines

Amphetamines were employed for the abolition of fatigue and for the suppression of appetite. It was first synthesised in 1887, but began to be therapeutically used only since the 1930s. Because of its abuse potential, its therapeutic administration is greatly restricted today. Amphetamines stimulate the central nervous system causing increased alertness and self-confidence. (**Ecstasy,** i.e. methylenedioxymethamphetamine, was developed in 1914 and for some time had a role in psychotherapy as a consciousness-altering agent.) Initial euphoria, more extrovert behaviour, increased talkativeness with rapid speech, lack of desire to eat or sleep, tremor, dilated pupils, tachycardia and hypertension are the common features. More severe intoxication is associated with excitability, agitation, paranoid delusions and hallucinations with violent behaviour. Convulsions, rhabdomyolysis, hyperthermia and cardiac arrhythmias may develop in the worst cases.

Death is uncommon from overuse of the amphetamines alone. At autopsy, there are no specific findings apart from the rare possibility of a cerebral or subarachnoid haemorrhage from the induced hypertension.

TREATMENT

It includes supportive measures as per the circumstances. Sedation may well be required and chlorpromazine has been recommended. The peripheral sympathomimetic actions of amphetamines may be antagonised by β-adrenergic blocking drugs. Acidification of the urine has been reported to increase the renal elimination of methamphetamine.

MEDICOLEGAL ASPECTS

Other derivatives of amphetamines include methamphetamine, dextroamphetamine, fenfluramine, phentermine, mephentermine, methyl phenidate and synthetic amphetamines, i.e. **designer drugs** methylenedioxyamphetamine (MDA or Love Drug), methylenedioxymethamphetamine (MDMA or Ecstacy), and methylenedioxyethamphetamine (MDEA or Eve).

- Acute overdosage with amphetamines is uncommon in India.
- Intermittent amphetamine abuse is characteristically seen in athletes who wish to enhance their endurance and performance.
- Presently, 'designer drugs' are abused by youngsters during the course of rave parties, for dancing all the night long, particularly in the foreign countries.

Tricyclic Antidepressants

Tricyclic antidepressants and benzodiazepines (the ubiquitous hypnotics and sedatives) are the major causes of clinical problems with drugs that depress the central nervous system. In many countries, barbiturates have disappeared and are seldom encountered in overdosage. Tricyclic antidepressants have complex actions, which account for the diverse nature of the features seen after overdosage. They block the re-uptake of noradrenaline into peripheral and intracerebral neurons thereby increasing the concentration of monoamines in these areas. They also have anticholinergic actions and antiarrhythmic activity.

CLINICAL FEATURES

Features of poisoning usually appear within an hour or so after ingestion of an overdose and usually reach maximum intensity in 4–12 hours. Drowsiness, sinus tachycardia, dry mouth, dilated pupils, urinary retention, increased reflexes and extensor planter responses are the most common features of mild poisoning. Severe intoxication leads to coma. Sinus tachycardia, secondary to loss of vagal tone, is very common and the dose-related quinidine like action decreases myocardial contractility and delays conduction particularly in the Bundle of His and the more peripheral ventricular conduction system. The blood pressure and cardiac output fall due to combination of arrhythmias, negative inotropic effects and the relative hypovolemia. The combined effect is to produce metabolic acidosis and cardiorespiratory depression, which are the major causes of death.

TREATMENT

It consists of gastric lavage depending upon the condition of the patient. Absorption of the drug from the gut may also be reduced by the oral administration of activated charcoal. Attention to supportive measures, particularly adequate oxygenation, control of convulsions and correction of acidosis will be rewarding. Sodium bicarbonate should be given intravenously. Only a small proportion of the body load of tricyclic antidepressants circulates in the vascular compartment, and forced diuresis and haemodialysis are therefore of no value. Delirium with auditory and visual hallucinations is a frequent and troublesome complication during the recovery phase. Sedation with oral or intravenous diazepam may be required.

Benzodiazepines

The benzodiazepines comprise one of the most important groups of psychotropic drugs in present day medical practice. They are widely used as tranquilisers, hypnotics and sedatives. Chlordiazepoxide and diazepam are still commonly prescribed, but other early members of the group have largely been displaced by newer, short-acting compounds such as temazepam, lorazepam and triazolam. However, there is no reason to believe that the toxicity of the latter group in overdosage will be any different. Bromazepam, clonazepam, desmethyldiazepam, medazepam and prazepam are amongst the most recent additions to this extensive family of drugs.

CLINICAL FEATURES

Although many benzodiazepines have active metabolites that account for their prolonged sedative effects, they all share a remarkable safety when taken alone in overdosage. It has been reported that as many as 70 tablets of any of them are unlikely to produce anything more than mild effects in most adults. However, this is not always the case, and it cannot now be said that overdosage with these drugs is never without harm. More important, benzodiazepines potentiate the effects of other CNS depressants, particularly alcohol, tricyclic antidepressants and barbiturates. Dizziness, drowsiness, ataxia and slurred speech are the usual features, while coma, respiratory depression and hypotension are uncommon. Of all the benzodiazepines, flurazepam is most likely to cause significant CNS depression.

TREATMENT

Supportive measures are the important consideration. Gastric lavage is of doubtful value. In rare cases of severe poisoning, the use of the specific benzodiazepine antagonist, flumazenil, may be indicated. The dose is 0.5 mg intravenously over 30 seconds and if necessary, a further 0.5 mg over 30 seconds.

Hallucinogens

Hallucinogens are also referred to as **psychedelics.** 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. Some drugs, however, may have direct toxic effects.

LSD (LYSERGIC ACID DIETHYLAMIDE)

Common names: Acid/microdot/purple haze/white lightning etc.

The well-known LSD, which takes its acronym from the German *Lyserg Saure Diethylamide* and *psilocin*, respectively, is a powerful hallucinogenic that is not fatal in itself. It is an indole alkaloid derivative, of which other members are psilocybin and psilocin, contained in the Mexican mushroom (*Psilocybe mexicana*).

The discovery of psychedelic effects of LSD in 1947 led to an epidemic of LSD abuse during the 1960s. LSD is a very potent drug; oral dose as low as 20 μg may induce profound psychological and physiological effects. Tachycardia, hypertension, pupillary dilation, tremor and hyperpyrexia occur within minutes following oral administration. A variety of bizarre and often conflicting perceptual and mood changes, including visual illusions, synthesis and extreme lability of mood, usually occur within about half an hour after LSD intake. Time seems to pass very slowly and behaviour may become disturbed with paranoid delusions, necessitating sedation or physical restraint. **Flashbacks** in which the effects of LSD may be re-experienced without further exposure to the drug may occur in some cases for several years and have not been explained. 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 or symptoms. The most frequent medical emergency associated with LSD use is the panic episode (**'bad trip'**), which may persist for up to 24 hours. Management of this problem is best accomplished by supportive reassurance and, if necessary, administration of small doses of anxiolytic drugs.

LSD is the most powerful hallucinogen known to man. As with 'Cannabis', individuals intoxicated with LSD rarely present for medical help. It was popular among the Hippies in the West in the 1960s. Suicidal and accidental deaths arising out of bizarre behaviour induced by the drug have been reported.

PHENCYCLIDINE

Common names: Angel dust/PCP/Peace pill/Hog/Goon/Rocket fuel/Cadillac/Super grass, etc.

Phencyclidine (PCP) was developed for use as an anaesthetic in the late 1970s, but quickly abandoned mainly because of an unacceptably high incidence of postoperative psychotic reactions. PCP [l-(-phenylcyclohexyl piperidine)] is easily synthesised and several variants exist. It has a number of pseudonyms as written above. PCP is usually smoked in combination with tobacco and less frequently with marijuana, but it may also be ingested or injected. PCP binds to ionotropic 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.

Clinical Features

Pleasurable effects of phencyclidine include initial euphoria, a feeling of dissociation, numbness, perceptual distortion, and hallucinations (usually of visual type). Users may show horizontal or vertical nystagmus, flushing, diaphoresis and hyperacusis. Behavioural changes may include distortions of body image, disorganisation of thinking and feelings of estrangement. Higher doses of PCP (5–10 mg) may be complicated by hypersalivation,

profuse sweating, generalised seizures, prolonged psychotic reactions, dystonias and hypoglycaemia. Rhabdomyolysis and acute renal failure are common. Diagnosis may be achieved by determination of PCP levels in the serum or urine. PCP is excreted in the urine for a long time after ingestion, and autopsy samples may be positive for up to a week. CSF concentrations may be three or four times higher than those in the serum and take much longer to clear.

Treatment

PCP overdose requires life-support measures, including treatment of coma, convulsions and respiratory depression. There is no specific antidote or antagonist. PCP excretion from the body can be enhanced by gastric lavage and acidification of urine. Oral diazepam may suffice but chlorpromazine may be preferred, particularly if hypertension is a problem. Diaphenhydramine by intramuscular injection has been reported to abolish acute dystonic reactions induced by PCP. Acidification of urine enhances renal elimination and continuous gastric suction may be of some value by removing PCP secreted in gastric juice, thereby preventing reabsorption further down the gut.

Medicolegal Aspects

PCP has been a popular drug of abuse in the West. A common mode of intake involves sprinkling the drug on parsley or marijuana leaves, and smoking the same. It is possible that the poisoning can be caused by inhalation as intoxication has occurred in children in the same room as adults smoking the drug. It is sometimes used as an adulterant in expensive drugs of abuse such as cocaine. Addiction leads to violent tendency, psychosis and suicidal as well as homicidal behaviour.

Cocaine (Coke/Snow/Cadillac/ White Lady, etc.)

Cocaine is an alkaloid obtained from the leaves of coca tree, of which there are two important species *Erythroxylon coca* and *E. novogranatense*. They grow well in South America, Indonesia and India. Purified cocaine first became commercially available in 1884. Cocaine was an ingredient of Coca Cola until 1900. By 1985, owing to its increasing availability and declining price, 20 million people had tried cocaine in the United States. Increasing number of drug users, overdose deaths, crime and the images of 'crack babies' damaged in utero by cocaine-using pregnant women gave national visibility to the drug problem, particularly to cocaine use.

COMMON ROUTES OF ADMINISTRATION

Cocaine can be taken orally, by injection, by absorption via nasal and buccal membranes or by inhalation. **Cocaine hydrochloride,** the water-soluble form typically used for snorting or injection, is largely destroyed by the heat of burning and so is not well-suited for smoking. The hydrochloride salt can be converted to the freebase form by treatment with alkali and extraction with organic solvents. Inhalation of **freebase cocaine** produces almost immediate absorption and a rapid onset of effects. 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. In cocaine-producing countries, some users may smoke a crude intermediate product, cocaine sulphate (coca paste, pasta basica, basuca), which is usually contaminated with solvents. Other drugs such as ethanol, Cannabis and conventional hypnotics and sedatives are frequently taken with cocaine to reduce its less pleasant effects.

CNS AND PNS

Initial stimulation of brain occurs in a rostral-to-caudal fashion, i.e. cortex is stimulated first leading to excitement, restlessness and enhanced motor activity. Subsequently, lower motor centres get stimulated producing tonic-clonic convulsions. The medulla is initially stimulated causing an initial increase in respiratory rate, followed by depression with resultant respiratory depression. Such stimulant effects of cocaine are attributed to inhibition of dopamine reuptake in the nucleus accumbens.

- It is also suggested that cocaine also inhibits reuptake of noradrenaline (norepinephrine) and serotonin. Increase in the concentration of the former plays an important role in the production of toxic effects of cocaine.
- It stabilises the axonal membrane through blockade of sodium channels. This action of cocaine is responsible for production of local anaesthetic effects.

CVS

Cardiostimulatory effects of cocaine are largely due to sensitisation to adrenaline and noradrenaline, preventing neuronal reuptake of these catecholamines. Of these effects, prominent one is the production of tachycardia.

Arrhythmias are usually produced due to blockade of fast sodium channels in myocardial tissue.

METABOLISM

The half-life of a single dose of cocaine in the blood is only about 30–90 minutes. It is hydrolysed by butyrylcholinesterase (plasma pseudocholinesterase) and liver esterase into inactive metabolites, mostly benzoylecgonine and ecgonine methyl ester. 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 urine for 24–72 hours after brief periods of use. With repeated high dosages, cocaine or its metabolites may accumulate in

body compartments (e.g., fat and CNS), from where it is then slowly released. Consequently, using sensitive techniques, cocaine may be detectable in 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.

Cocaine abusers frequently use sedatives or opioids to modulate the stimulant and toxic effects of the cocaine. Sometimes an opiate, such as heroin, and cocaine are injected intravenously simultaneously, the mixture (**speedball**) is reportedly especially euphorigenic. Alcohol is probably the substance, most commonly used in conjunction with cocaine. (Alcohol is used to modulate both the 'cocaine high' and 'the dysphoria' associated with the abrupt disappearance of cocaine's effects.) The concurrent use of cocaine and alcohol may result in the accumulation of a distinct metabolite, cocaethylene. (It induces changes in the cardiovascular function similar to those of cocaine alone.) This metabolite is active and longer lasting than cocaine itself and may account for the enhancement of subjective effects and toxicity when the two are used simultaneously.

ACUTE INTOXICATION

Cocaine users commonly develop the symptoms of cocaine intoxication during the course of a single binge. The **euphoria** may be accompanied by increasing suspiciousness, hypervigilance, anxiety, hyperactivity, talkativeness and grandiosity. Other signs and symptoms of central stimulation usually follow in the form of tachycardia, cardiac arrhythmias, blood pressure changes, pupillary dilatation, perspiration or chills. Hallucinations may occur, including tactile hallucinations. Judgement is impaired but insight into the drug induced nature of the hallucinations is usually retained. Ultimately, **stage of depression** may supervene leading to loss of reflexes, coma, paralysis of muscles, circulatory and respiratory failure. Sudden death may occur following IV injection, smoking and also by snorting, due to cardiac arrhythmias and cardiopulmonary arrest. A number of rare complications of method of use of cocaine have also been reported. These include pulmonary oedema after intravenous injection of freebase cocaine and pneumomediastinum and pneumothorax after sniffing it.

CHRONIC INTOXICATION (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. Immediately upon intake, there is euphoria (**rush**) as written above, followed about an hour or so later by rebound depression (**crash**). To avoid unpleasant effects of the latter, the individual feels compelled to take the drug again. Cocaine can also induce a **toxic delirium** and a more persistent toxic psychotic disorder characterised by suspiciousness, paranoia, visual and tactile hallucinations and loss of insight. The hallucination of bugs (**cocaine bugs**) or vermin crawling under the skin (**formication/Magnan symptom**) is sometimes reported and is often associated with excoriation of the skin.

Chronic cocaine use causes significant loss of libido and adversely affects reproductive function. Impotence and gynaecomastia have been observed in male cocaine abusers. Women who abuse cocaine have reported major derangements in menstrual cycle function including galactorrhoea, amenorrhoea and infertility. Cocaine abuse by pregnant women (particularly the smoking of crack, producing the so-called **crack babies**) has been associated with both an increased risk of congenital malformations in the foetus and perinatal cardiovascular and cerebrovascular disease in the mother.

TREATMENT

Cocaine toxicity produces a hyperadrenergic state characterised by hypertension, tachycardia, tonic-clonic seizures, dyspnoea and ventricular arrhythmias. Intravenous diazepam in doses up to 0.5 mg/kg may be given for controlling seizures. For ventricular arrhythmias, 0.5–1.0 mg of propranolol intravenously has been advocated. Haloperidol for psychosis may be given. Treatment of chronic cocaine abuse requires combined efforts by primary care physicians, psychiatrists and psychosocial care providers.

AUTOPSY

Usually, there are no specific features at autopsy. The pulmonary oedema often seen in heroin deaths is not seen with cocaine, though the death in either case is due to dysrhythmia. The diluent used in 'cutting' the drug for street sale may be found in the injection sites, the regional lymph nodes, in the lungs and in other organs. Adulterants may be similar to those used with heroin and may include talc, starch, quinine, lactose and dextrose. (In former years, in New York and California, transmission of malaria was a known problem amongst those who shared syringes and needles. Use of quinine as a diluent was probably in the light of treatment for the plasmodium.) Cerebral haemorrhage may be a possible complication of acute hypertension produced by cocaine. Autopsy may reveal complications of the septic methods used for injections. The more serious sequelae in this context may include endocarditis. This can affect any heart valve, including those on the right side, which are not usually affected in postrheumatic endocarditis.

Any drug used intravenously, if it has particulate matter admixed, such as starch or talc, may cause foreign body granulomata in the lungs when the undissolved components are filtered out in the pulmonary capillary bed.

As cocaine is commonly used by sniffing, swabs should always be taken from each nostril using a plain cotton-wool swab. (Chronic 'snorting' has resulted in perforation of the nasal septum and CSF rhinorrhoea due to thinning of the cribriform plate and pulmonary granulomatosis.) An unused swab should be sent to the laboratory as a control. Blood levels in the fatal

cases vary widely, but typical ranges may extend from 1 to 21 mg/L, with a mean of 5.2 mg/L, according to Baselt.

Cocaine can be recovered by sampling from recent injection sites, or by swabs from the nasal mucosa. Brain is an excellent source for its detection where cocaine may be found not only in dopamine-rich areas such as caudate, putamen, and nucleus accumbens, but also in other extrastriatal regions.

CIRCUMSTANCES OF POISONING

It is rarely used for homicide or suicide. Accidental cases occur from addiction, hypodermic injection and from urethral, vesical and rectal injection. It is believed to be an aphrodisiac and to increase the duration of sexual act by desensitising sensory nerves of glans penis, when used locally. Prostitutes sometimes inject cocaine solution into vagina to produce local constriction. It causes mental derangement in chronic users who may get involved in crimes. Three major causal categories may be described: psychopharmacological effects, economic compulsion (violent crimes committed to obtain money for drugs) and systemic violence (associated with the business methods and lifestyle of drug dealers). Cocaine can induce states of paranoid and aggressive behaviour. Protracted cocaine abuse may cause paranoid ideation, and visual and auditory hallucinations, a state that resembles alcoholic hallucinosis. (Also see 'Body Packing and Body Stuffing' described in the beginning of this Chapter).

Cannabis

Known in Central Asia and China for at least 4000 years, the Indian hemp plant, ***Cannabis sativa/Cannabis indica***, is a hardy, aromatic annual herb, known as **Dagga** in South and Central Africa and **Hashish** in Egypt. (The female plant is taller, about 4–6 m, and has darker and more luxuriant foliage than the male.) The bioactive substances derived from the plant are collectively referred to as **Cannabis**. By most estimates, Cannabis remains the world's most commonly used illicit drug. It occupies fourth place in worldwide popularity amongst psychoactive drugs, after caffeine, nicotine and alcohol. *Cannabis sativa* is widely cultivated for its fibre, which is used to make rope and cloth. Seeds are used to make oil. The resin contains over 60 structurally similar compounds called **cannabinoids**, of which delta-9-tetrahydrocannabinol (THC) is responsible for most of its psychoactive effects. Various preparations of abuse are as follows:

Marijuana (marihuana/mary jane/pot/weed/grass): The most commonly abused form of Cannabis is marijuana, which is dried plant material including cut leaves, flowering tops, stems and seeds. The term marijuana is used in America to refer to Cannabis. Marijuana is a Mexican term meaning 'pleasurable feeling'. The form of Cannabis generally used in America is **similar to *ganja***. Marijuana is eaten alone or as a part of confection, or drunk in beer or some other beverage, or smoked in pipes or rolled in cigarettes and then smoked. These cigarettes contain between 350 and 650 mg of Indian hemp or its equivalent of the Mexican Cannabis plant, and are known as **reefers or weed.** Very potent forms of marijuana **(sinsemilla)** are now available in many countries, and concurrent use of marijuana with crack/cocaine and phencyclidine is increasing.

Hashish (*charas*): It is dark black-brown resinous exudate from the leaves and stems of the plant. It is smoked with tobacco in a pipe or *hukka*. It has 8–12% THC. (Hashish oil, a concentrated distillate of hashish, has been assayed at up to 25–60% THC and may be added to marijuana or hashish to enhance its THC concentration.)

Ganja: It is the resinous extract of leaves and bracts of female plant. It is mixed with a little tobacco and is usually smoked in a pipe (*chilam*). It contains the active principle in concentration of about 25%. The resin have a rusty green colour and a characteristic odour.

Majun: It is a sort of confection prepared from bhang after treating it with sugar, flour, milk and butter. It has a sweet taste. It is also sold as small lozenge-shaped pieces. Sometimes, *dhatura* is mixed with 'majun'.

Bhang (*Siddhi/patti/sabji*): It is also a form of Cannabis, which is ingested. It consists of dried leaves and fruiting shoots, and is prepared by rubbing black pepper, sugar and dried leaves of Cannabis on a stone slab. It is taken in the form of a bolus or pills or is mixed with water and strained through a muslin cloth before it is drunk. It may also be used as an infusion in the form of a beverage.

(THC content and concentration of different parts of the plant and between species varies greatly. THC produced by the plant is related to environmental conditions such as the amount of sunlight received, humidity and soil condition.)

METABOLISM

The THC dose needed to produce pharmacological effects in humans from smoking range from 2 to 22 mg. THC is lipid soluble and rapidly absorbed after inhalation. It is highly protein bound and quickly redistributed from blood into other tissues. About 1% penetrates the blood–brain barrier. About two-thirds of the drug is excreted via the enterohepatic circulation into the faeces and the remaining one-third is removed through the kidney. Most of the metabolites of THC are produced by the liver. Among these compounds, 11-norcarboxy-THC has the highest concentration in urine and is the metabolite usually screened for in routine toxicological analyses. This and other cannabinoid metabolites can be detected in the urine for 2–3 days after casual use. For daily heavy users, detectable level can persist for up to 4 weeks.

Orally ingested marijuana requires about three times as much THC as smoked marijuana to produce equivalent effects, because only 3–6% of ingested THC is absorbed. (During

pyrolysis, more than 150 compounds in addition to THC are released in the smoke. Although most of these compounds do not have psychoactive properties, they do have potential physiologic effects.)

MECHANISM OF ACTION

Demonstration of cannabinoid receptor sites and density in the animal experiments has been correlated with clinical effects in human beings. THC acts on widely distributed specific receptor regions of brain concerned with pain perception, cognition, memory, reward and motor coordination. The distribution appears as follows:

- Basal ganglia and cerebellum showing highest density of receptors, which reflects its interference with motor coordination (Cannabis produces dose-related impairment in cognitive and behavioural functions that may potentially impair driving automobile or operating machinery. About 11–33% of motor vehicle fatalities, especially in the age group of 15–30 years, have reportedly been attributed to Cannabis abuse in the developed countries).
- Cortex, hippocampus and dentate gyrus showing intermediate levels of binding, which are consistent with effects on cognition and short-term memory.
- Brain stem regions (which control cardiovascular and respiratory functions) showing low receptor density, correlating with the 'lack of lethality' of the cannabinoids.

Cannabis is often used in combination with other drugs. It may alter the effects of amphetamines, atropine, barbiturates, clomipramine, cocaine, ethanol, nicotine, opiates and phencyclidine. Because of shared hepatic metabolic systems, ethanol and phenobarbital can inhibit metabolism of THC.

FATAL DOSE AND FATAL PERIOD

Charas 2 gm, *ganja* 8 gm, *bhang* 10 gm/kg body weight. Death may occur in 12–24 hours in case of acute poisoning. [Fatalities are uncommon because fatal dose is very high. Death may occur due to hazards like inhalation of vomitus or due to some accident (injury, drowning or electrocution, etc.).]

ACUTE INTOXICATION

The effects of THC depend upon dosage, frequency, route of administration and the experience and expectations of the individual. Other factors that affect the amount of THC actually consumed are smoking technique, the amount destroyed by pyrolysis and how quickly the drug is used, because THC deteriorates by about 5% per month at room temperature. Although the effects of acute intoxication are relatively benign in normal users, the drug can precipitate severe emotional disorders in individuals who have antecedent psychotic or neurotic problems. As with other psychoactive compounds, both **the set** (user's expectations) and **the setting** (environmental context) are important determinants of the type and severity of behavioural intoxication.

The effects are somewhat similar to those of alcohol causing excitement/euphoria at first followed by narcosis. The individual experiences a feeling of **cheerfulness and well-being** and tends to become talkative, though rarely **running amok**. Appetite is increased and he enjoys food. Even modest doses of Cannabis impair memory, reaction time, perception, motor coordination and attention. Large doses may produce nausea, anxiety, confusion, hallucinations and delusions. Hallucinations are often of sexual character. Persons with excitable temperament may become violent if interfered with. This stage is followed by **stage of narcosis** characterised by giddiness, confusion, drowsiness and dilated pupils. Tingling and numbness of the extremities is often present. Fatality is unusual. The individual may pass into deep sleep and wake up without depression. Rarely, drowsiness may be followed by coma, collapse, and death may occur from respiratory paralysis.

Intravenous injection of Cannabis infusions leads to an altogether more serious effects. Within a few minutes, there occurs nausea, vomiting and chills, followed after an interval of an hour or so, by profuse watery diarrhoea, tachycardia, hypotension, etc. A marked neutrophil leucocytosis is often present.

TREATMENT

Most patients will settle with reassurance and comforting. Sedation with IV diazepam may be required. Those who have injected Cannabis infusions should be treated supportively. Cannabis body packers and stuffers should be managed as described earlier in the beginning of this chapter.

CHRONIC INTOXICATION

It results from continued use of the drug in any form and is characterised by anorexia, loss of weight, weakness, tremors, impotence and moral deterioration. Chronic abusers may lose interest in common socially desirable goal and steadily devote more time to drug acquisition and use. However, THC does not cause a specific and unique 'amotivational syndrome'. Conjunctival injection and tachycardia are the most frequent immediate physical concomitants of smoking marijuana. Cannabis-induced **delirium** is characterised by marked impairment of cognition and performance tasks. High doses also impair the level of consciousness along with affect on the cognitive functions. Delusions and hallucinations have been reported. Cannabis-induced **psychotic disorder** is characterised by persecutory delusions or auditory and visual hallucinations, especially in persons with underlying psychiatric disorders. The psychotic episodes are sometimes referred to as **hashish insanity/hemp insanity**. The victim may **run amok**. This condition is characterised by a frenzied desire on the part of the victim to commit murders (impulse to murder). He first kills a person against whom he may have real or imaginary enmity and then kills anyone that comes in his way until the homicidal tendency lasts.

Then he may commit suicide or may surrender himself. The question of criminal responsibility for the acts, done while in a state of intoxication, is not affected by the nature of the intoxicating agent. Hence, Sections 85 and 86 of IPC apply with the same vigour as they do to the alcoholic intoxication.

Withdrawal signs and symptoms have been reported in chronic Cannabis users. These include tremor, nystagmus, sweating, nausea, vomiting, diarrhoea, irritability, anorexia and sleep disturbances. The symptoms are usually relatively mild in comparison to those observed in heavy opiate or alcohol users and rarely require medical or pharmacological intervention.

LABORATORY INVESTIGATIONS

Most laboratories use the enzyme-multiplied immunoassay (EMIT), although a radioimmuno assay is also commonly used. Confirmation by gas chromatography mass spectrometry is routinely done. Cannabis and its metabolites may be detected in urine at the usual cut-off level of 100 ng/ml for 42–72 hours after the psychological effects subside. Passive inhalation that occurs under unusually crowded conditions may also reveal Cannabis metabolites in the urine but only if the cut-off level used in the urine test is decreased to 20–25 ng/ml. However, this may increase the frequency of false-positive results. Urine that contains Cannabis metabolites only implies that Cannabis exposure occurred at an indeterminate time prior to testing. Identifications of very low levels of metabolites in the urine (such as can occur with passive inhalation) may be carried out using cut-off point of 100 ng/ml or above.

THE CIRCUMSTANCES OF POISONING

Indian hemp in one form or the other is widely used in India as an intoxicant by the less reputable members of the community. *Sadhus* and *faqirs* use it to get into a religious mood and to overcome hunger and thirst. Majun and *charas* have been used by road-poisoners to stupefy persons to facilitate robbery. *Charas* and *ganja* are sometimes used to dope cigarettes. It is said that the victim can recall things forgotten since long, and this may be the reason for the extensive use of the reefer cigarettes in Western countries. Like opium, ganja has been used to steady the nerves before performing some bold act of violence. It is said that before the British Rule, hemp was sometimes served to the soldiers about to be called upon to undertake some particularly dangerous or difficult mission, with wonderful results.

Most of the cases of poisoning are due to overindulgence, but there may be accidental ingestion or inhalation. The experiences of the people vary depending on life experiences and personality styles. It is believed that Cannabis acts as an aphrodisiac agent, but the action is not certain. (Also see 'Body Packing and Body Stuffing', described in the beginning of this Chapter.)

- Fibres of the woody trunk of Cannabis (collectively called as Indian hemp) have been used to produce rope and twine, as well as clothing.
- THC in the form of a synthetic oral cannabinoid (dronabinol) has been reported to be effective in controlling nausea and diarrhoea associated with AIDS. It is also reported to possess analgesic properties. In the United States, laws have been approved in some States to allow the use of marijuana by seriously ill patients, including AIDS patients, for alleviating pain and other symptoms of disease. Though possession and distribution remains federal crimes where the same has not been approved by the federal programme, an implication of a judgement in a case [United States vs. Oakland Cannabis Buyer's Cooperative, 190F., 3d 1109 (9th Cir. 1999)] is believed to provide some relief under the concept of "medical necessity", i.e. the medical need to treat the symptoms in certain cases by prescribing marijuana may be a 'lesser evil' than the violation of laws against the possession and distribution of marijuana.

No authentically documented cases of lethality from Cannabis alone have been documented. Long-term excessive use of Cannabis may occasionally precipitate acute psychotic reactions, causing the victim to **run amok** in homicidal frenzy. This was well revealed during Vietnam War when several American soldiers started suffering from acute toxic psychosis arising out of heavy abuse. (The term "amok" was originally derived from the Portuguese–Indian name, 'amuco' referring to heroic warriors ready to die in battle, and was immortalised in Malayan epics, similar to the 'berserkr' in ancient Norse sagas. These days, 'running amok' (going berserkr) is usually used in relation to homicidal behaviour for attention-craving angry young men, often under substance influence. Typical amok reactions are preceded by a prodromal state of dysphoria and tension experienced in conjunction with interpersonal or situational problems. Changes in visual perception and threatening illusions causing fear and rage are common. This may be followed by a sudden kinetic discharge during which randomly aggressive, destructive and homicidal acts are committed. The 'amoke run' ends with suicide, or alternatively, the exhausted individual may be overpowered, who then may claim amnesia for his deeds/actions. A case mentioned in the literature is worth mentioning here: A *havaldar* in Kumaon battalion while seated in the orderly room with other native officers of the regiment, suddenly rose and killed one Nardeb (an acting *havaldar*), with a kukri. He then rushed about and wounded two sepoys of the corps. (It was alleged that the deceased had debauched the prisoner's wife.)

Illicit Use of Drugs in Sports (Doping)

As per dictionaries, the word 'dope' implies a drug given to a race horse to influence its speed/performance. In pursuit of gold and glory, many sportsmen use performance-enhancing substances in an effort to gain edge over others. In the world of sports, it has been described as "the deliberate or inadvertent use of a substance or method banned by Medical Commission of International Olympic Committee". Besides showing some

improved performance, these drugs and/or methods have also shown unhealthy side-effects and even deaths of athletes. Due to health and ethical reasons (as the practice undermines the basic joy of sports and given an unfair advantage against the competitor), the International Olympic Committee (IOC) in 1967 agreed to ban and/or restrict certain substances and methods that could be used in the attempt to enhance performance. A brief account of the drugs that have been abused and that have been prohibited by the IOC is furnished below:

Steroids are a group of complex substances that are naturally produced by many plants and animals. Among them are androgens (male hormones) produced by the testes and by the cortex of the adrenal glands. Most popular preparation is "anabolic steroids" which are synthetically derived chemicals that mimic male hormones. They were first used by Nazi soldiers to become better "war machines". They became a common household name when the Canadian sprinter, Ben Johnson, was found using these in the 1988 Seoul Olympics. **Nonsteroidal anabolic agents** include beta-2 agonists that are most commonly found in asthma medications. Their use requires a 'Therapeutic Use Exemption'.

Androgens influence the development of male reproductive organs and secondary sexual characters. They add to the building up of the body tissues and assimilation of proteins and hence, promote increasing muscle mass. However, side effects are many. All androgens suppress gonadotrophin secretion when taken in high doses and thereby suppress endogenous testicular function. This leads to decrease in endogenous testosterone and sperm production, resulting in diminished fertility. They also increase the risk of cardiovascular damage as they often cause hypertension, decreased high-density lipoproteins, and increased low-density lipoproteins. In women, virilisation including facial and body hirsutism and acne are the common side-effects. Boys and girls whose epiphyses have not yet closed experience premature closure and stunning of linear growth.

Stimulants, also called psychomotor drugs, are used to produce alertness, an increase in concentration and lower sensitivity to pain and fatigue. Amphetamines are most commonly used. They are a group of synthetic chemicals similar to the 'fight and fright' hormone (adrenaline), known as "pep pills". However, experiments have failed to prove the value of these drugs except as a psychic stimulant. Some athletes have been known to die during athletic events because of interaction between such drugs and the norepinephrine and epinephrine released by the sympathetic nervous system during the exercise. (Apart from amphetamines, caffeine (in higher concentrations) and a number of active ingredients in cold and flue preparations, i.e. pseudoephedrine, have also been banned.)

PEPTIDE AND GLYCOPROTEIN HORMONES AND ANALOGUES

Hormones act as messengers from one organ to another to do such things as stimulating growth, influencing behaviour, influencing sensitivity to pain, etc. Analogues are substances that act in a similar manner as the hormones. Examples include human chorionic gonadotrophin (hCG), human growth hormone (HGH), and erythropoietin (EPO). By using such products, athletes risk complications such as cerebral and coronary thrombosis due to accompanying increase in red cell mass.

Diuretics are also being used in order to dilute the urine and thus, complicating the demonstration and interpretation of levels of drugs therein. However, they can lead to electrolyte disturbances. Other masking agents like alpha-reductase inhibitors, plasma expanders, probenecid and epitestosterone have also been banned.

Narcotics and analgesics are very powerful painkillers and help athletes perform beyond their normal threshold of pain. They mask pain and the body's natural warning to injury. Hence, danger does exist of incurring serious injury to the body due to reduced pain. Further, many narcotics are illegal substances under the NDPS Act and they are potentially addictive.

Gamma-hydroxybutyrate (GHB): It has been used as a body-building drug, a growth hormone secretagogue, a treatment for narcolepsy, a drug of abuse by the "rave" crowd, and also has been referred to as a **date rape drug** (drug-facilitated sexual assault—DFSA) as it has reportedly been used by sexual predators (drugs that have been associated with sexual assaults include flunitrazepam, gamma-hydroxybutyrate, and ketamine. Most such drugs are easy to administer, impair consciousness and cause anterograde amnesia and therefore victims are disinhibited showing least resistance. They report late due to number of factors including embarrassment and the amnesia).

When taken in excess, it can cause depressive effects on the central nervous system leading to respiratory depression, and possibly coma and death. Urinary documentation of GHB may be more reliable indicator of its abuse. This is because GHB is not known to form de novo in the urinary bladder. Even if the level of GHB itself does not appear fatal, its combination with alcohol or other respiratory depressants or toxic drugs may prove fatal (GHB can easily be demonstrable in the postmortem blood and the levels are likely to rise as a product of decomposition. This necessitates the addition of sodium fluoride to the sample so as to avoid artifactual GHB in the blood).

ESCAPING THE NET

Drugs and drug testing is a cloak-and-dagger affair in sports. Given the demands of testing for illicit substances, doping has become highly sophisticated. Athletes take anabolic steroids in low doses or adjust the dose, which cannot be detected at the time of competition as much has already been eliminated from the body. This has triggered the concept of 'out-of-competition' testing in the recent years. Women have been known to insert into their vaginas spray bulbs containing urine from another (drug-free) woman to mislead organisations. Attempts to alter the integrity and validity of samples through manipulations like

intravenous infusions, catheterisation, and urine substitution, etc., have been reported.

The advent of **blood doping** is quite innovative. In this practice, blood is removed a week or two prior to competition and then replaced just before the competition. Meanwhile, the missing blood has been replenished by the body. Hence, the additional blood results in additional haemoglobin and thereby, more carriage of oxygen to the tissues including muscles. However, problem may arise if the blood is not stored correctly or another person's blood is used—exposing the athlete to infections like hepatitis, HIV, etc.

Street Drugs and Designer Drugs

As the name suggests, **street drugs** are obtained from surreptitious sources (on the street) rather than from chemists/pharmacies, because they are sold in contravention of the drug rules. Obviously, the quality control of these drugs is non-existent, and many such drugs are likely to be adulterated with substances that look, taste or even feel like the original drug. In the event of poisoning, the management poses a major problem and forensic implications arise in the event of the death. On account of the apprehension of contracting AIDS and other infectious diseases, addicts now avoid syringe-sharing and have turned to smoking, snorting, and ingestion of drugs. And the present-day drug addicts therefore may not show any needle mark that used to be a cardinal sign of the addicts a little while ago. This group of drugs usually includes the stimulants, depressants, narcotics, inhalants, hallucinogens and designer drugs.

The term **'designer'** is used to indicate an analogue, i.e. a compound similar in action to another compound but different in its structure. To circumvent legal problems in the storage, distribution, and sale of such drugs, clandestine laboratories manufacture new drugs by altering chemical structure of a known drug, either legal or illegal. The compounds so produced are called **designer drugs** (analogues). These designer drugs have some of better properties as compared to the normal drugs such as enhanced potency and longer duration of effect. These drugs are difficult to detect due to their being effective in small quantities in the order of a few micrograms, and, therefore, such drugs will be available in pico or femtogram level in the body fluids necessitating new methodology for their identification and interpretation of results. Four main groups have been suggested: (*i*) stimulants (amphetamine group), (*ii*) depressants (methaqualone group), (*iii*) narcotics (opioid group) and (*iv*) hallucinogens (phencyclidine group).

40 CHAPTER

Deliriant Poisons

After going through this chapter, the reader will be able to describe: Features of poisoning by *dhatura* and its alkaloids | Medicolegal aspects of *dhatura* poisoning

As the name suggests, poisons in this group are characterised by a well-marked deliriant stage. The important poisons in this group are *dhatura, Atropa belladonna, Hyoscyamus niger,* Cannabis and cocaine (Cannabis and cocaine have already been described under hallucinogens in the Chapter "Non-narcotic Drug Abuse").

Dhatura

In India, *dhatura* (*Ummatta or Shivashekhara*) plants are abundant and grow wild all over the country. ***Dhatura fastuosa*** grows in plain and ***Dhatura stramonium*** grows in the range of Himalayan altitude. **In the plains**, two varieties of *Dhatura fastuosa* are available—***Dhatura alba***, a white flowered plant **(*safed dhatura*)**, and ***Dhatura niger***, a black or rather deep-purple flowered plant **(*kala dhatura*)**. The flowers are bell shaped. The fruits are spherical and have sharp spines, giving the name **thorn apple** to the plant. They contain brown seeds resembling chilli seeds. Their differentiating features have already been outlined under the topic 'Capsicum'. An average sized fruit contains 450–500 seeds. Seeds yield what is called '*dhatura*'. All parts of the plant are poisonous, but the seeds and fruits are considered to be the most noxious. ***Dhatura stramonium*** (Jimsonweed/apple of Peru/devil's trumpet/Jamestown weed/mad apple/stinkweed/thorn apple) is an extremely deadly plant. There are about 15 species of *dhatura*; while all are poisonous, their fragrance can be sweet or unpleasant depending upon the season. The active principle contains the alkaloids, **hyoscyamine, hyoscine or scopolamine and traces of atropine** commonly referred to as Belladonna Alkaloids. (In Italian language, 'belladonna' means a pretty woman. In ancient times, women used to instill drops containing atropine into their eyes for producing pupillary dilatation so as to make themselves appear more attractive.)

Belladonna alkaloids competitively inhibit the muscarinic effects of acetylcholine. Central nervous system effects result from their central antimuscarinic actions, i.e. vagal stimulation leading to decreased heart rate. An **alkaloid** is a complex substance found in various plants. It is so called because it behaves like an alkali and combines with acids to form salts. The distribution of alkaloids in the plant is not uniform—some are concentrated in the roots, others in the seeds, still others in the bark, and so on. **Some of the important alkaloids are** nicotine, atropine, hyoscine, morphine, strychnine, aconitine, ergotoxine and cocaine. Some synthetic substances, such as amphetamine, heroin, pethidine, methadone also behave chemically like alkaloids. Some alkaloids like strychnine, morphine and aconitine are quite resistant to putrefaction, while others, e.g. cocaine, decompose rapidly.

MECHANISM OF ACTION

Alkaloids exert both central and peripheral actions. Small doses stimulate the central nervous system causing excitement and restlessness, while large doses produce depression, delirium and later coma. Peripheral effects are due to the blockage of cholinergic fibres with resultant parasympathetic paralysis. Therefore, they inhibit secretion of sweat and saliva, dilate the cutaneous blood vessels, dilate the pupils and stimulate the heat regulating centre. (The active principle of *dhatura* is excreted unchanged in the urine almost immediately after its administration and the excretion is usually completed in 10–20 hours. It is, therefore, advisable to preserve urine in cases of *dhatura* poisoning, since the urine may show active principle, whereas the stomach wash may not occasionally respond to the chemical test.)

SYMPTOMS AND SIGNS

They occur in two stage, i.e. delirium followed by coma. In most cases, *dhatura* seeds are powdered and administered in food. The earliest symptom is a bitter taste in the mouth. Dryness of the mouth and throat occurs due to inhibition of salivation (**dry as a bone**) resulting in difficulty in talking, dysphagia and thirst. The face gets flushed due to dilatation of cutaneous blood vessels (**red as a beet**). The pupils are dilated. Light reflex at first is sluggish and later absent. The power of accommodation is

interfered and hence the vision suffers (**blind as a bat**). The body temperature gets raised, skin is dry and hot (**hot as a hare**) due to inhibition of sweat secretion and stimulation of heat regulating centre. The pulse becomes rapid, full and bounding but later becomes weak and irregular, and respirations are increased. Mental changes include restlessness and agitation and the patient may not recognise relatives or friends. The patient becomes confused, giddy, staggers as if drunk and later becomes delirious. He mutters indistinct words and suffers from muttering delirium (**mad as a wet hen**). He may be noisy, violent and subject to visual and auditory hallucinations. The patient tries to run away from his bed, picks at the bedding/clothing, tries to pull imaginary threads from tips of his fingers (carphologia), threads imaginary needles. The acute delirium begins to pass off in an hour or 2 and the patient is inclined to be drowsy and passes into sleep or coma. Death is rare and may occur due to respiratory paralysis. A fatal end is however unusual. The important signs and symptoms can be summarised under the **Ds**, viz.:

(1) dryness of the mouth and throat,
(2) difficulty in talking,
(3) dysphagia,
(4) dilatation of cutaneous blood vessels,
(5) dilatation of pupils,
(6) dry hot skin,
(7) dysuria,
(8) drunken gait,
(9) delirium and
(10) drowsiness leading to coma.

FATAL DOSE AND FATAL PERIOD

Usual fatal dose is 50–100 seeds and fatal period 24 hours.

TREATMENT

Stomach wash with weak solution of tannic acid. Though prostigmine in 0.5–1 mg dose or pilocarpine in 5–15 mg dose is considered to be the physiological antidote, these agents may not give good result in severe intoxication as the cholinergic receptors are blocked. Delirium can be controlled by short-acting barbiturates. Light diet and free purgation should be carried out for 3–4 days to remove the seeds.

POSTMORTEM APPEARANCES

These are same as of asphyxia. *Dhatura* seeds or their fragments may be found in the stomach and intestines. It is, therefore, necessary to make a careful search for them in the vomited matter, stomach contents and faeces. Congestion of the gastrointestinal tract may be there. *Dhatura* seeds resist putrefaction and are found even when the body is decomposed. However, the mydriatic principle contained in them appears to be destroyed by putrefactive changes in the body, although it can be searched after some lapse of time in the vomit or from the earth upon which the patient has vomited. **The other poisons that resist putrefaction** include phosphorus, arsenic, antimony, hyoscine, strychnine, nicotine, yellow oleander and endrin.

MEDICOLEGAL ASPECTS

In India, *dhatura* is employed mainly as a stupefying poison mostly for purposes of robbery and rarely for kidnapping and rape. It is, therefore, sometimes known as **road poison**. The powdered seeds are mixed with food, drink or tea or may be given in ***paan***. Since the seeds are lighter and float when given in liquid, they are used in the form of infusion. Sometimes, the seeds are mixed with incense wood and the victim is exposed to the fumes, which cause lethargy. The victim soon falls into deep sleep and later wakes up to find his belongings lost.

By the judicious use of *dhatura*, a whole household can be so drugged that the thieves can ransack the house at their ease. In one case, the thieves had not only ransacked a house but had also prepared and eaten a meal before decamping. *Dhatura* was detected in the vomit of the victims, and in the remains of the food they had partaken.

The blood pressure is greatly increased in the early stages of *dhatura* poisoning. A moderate dose of drug in a person with diseased arteries may cause the rupture of cerebral vessel and so bring about a fatal result. As reported, this occurred in the case of an individual aged 70, with advanced arteriosclerosis, who was dosing himself with *dhatura* for its aphrodisiac effect. The old man had recently married a young wife.

Dhatura is occasionally used to fortify Indian liquors, and is sometimes an ingredient of the sweetmeat called **majun,** which is used as a mild intoxicant or aphrodisiac.

Children can be easily kidnapped by giving them candy or sweets mixed with *dhatura*. They comply with the instructions of the poisoner to follow him.

Accidental cases may occur in children and adults who eat the raw fruit or seeds mistaking them for edible fruits or capsicum seeds. Accidental cases may also occur from the injudicious use of *dhatura* seeds by quacks for treatment of various ailments.

Hyoscine poisoning **(Crippen case)**: An American quack was charged in 1910 with the murder of his wife by hyoscine, and with cutting up her body into small parts and burying it under the floor of a house in Hilldrop, Crescent, London. Identification was made through the scar of an old abdominal operation and the identity of pyjama in which some of the remains were wrapped. Two-sevenths of a grain of hyoscine were found by Dr. Wilcox in the viscera examined.

Beverly in 1676 described effects of accidental *dhatura* poisoning amongst soldiers sent to quell Bacon's rebellion in the state of Virginia. The incidence took place in Jamestown, and hence the name, often abbreviated to "Jimson weed".

Dr. SN Tiwari, Chemical Examiner of UP, reported a case that occurred at Muradabad in 1963 wherein a Mohammedan male, aged about 60 years, developed rigor soon after eating

"*kheer*" in the evening. Death occurred the same night, and he was buried the next morning. On suspicion, his dead body was exhumed after 2 months. Chemical, physiological and microscopic tests confirmed the presence of *dhatura* in the dissolved, disintegrated and liquefied visceral material. (The active principle of *dhatura* is excreted unchanged in the urine almost immediately after its administration. It is, therefore, advisable to preserve the urine in cases of *dhatura* poisoning, since the urine will show active principle on chemical analysis, whereas the stomach wash may not occasionally respond to the test.

41 CHAPTER

Spinal Poisons

After going through this chapter, the reader will be able to describe: Source, mechanism of action and features of strychnine poisoning with medicolegal aspects | Important poisons affecting peripheral nerves

This group of poisons acts mainly on the spinal cord, the cerebral symptoms are either slight or absent. The action may be a stimulant one resulting in the production of spasms or a depressant one resulting in paralysis and loss of sensation. Strychnine is an example of the former variety of poison and gelsemium of the latter.

Strychnine (Kuchila)

Strychnine is an alkaloid obtained from the seeds of ***Strychnos nux vomica*** and other species of strychnos plants that grow in India. The ripe fruits of the plant contain seeds, which are poisonous. **The seeds** are hard and flat, about 2 cm in diameter and 1/2 cm in thickness and slightly convex on the one side and concave on the other. They are ash-grey in colour and have a shining hard pericarp covered with fine downy hair. They have quite bitter taste and yield two principal alkaloids, **strychnine** and **brucine**. Besides, the seeds contain, to a small extent, a glycoside named **loganin**. The bark, wood and leaves of the plant contain brucine but no strychnine. Strychnine crystallises into colourless, inodorous, rhombic prisms, having an intensely bitter taste. It dissolves very sparingly in water or ether, but dissolves in alcohol (90%) and in benzene, and readily in chloroform.

MECHANISM OF ACTION

This alkaloid of nux vomica plant is a potent convulsant poison with a lethal dose of a few milligrams per kilogram body weight for most animals. It lowers the threshold for stimulation of spinal reflexes by blocking inhibitory pathways exerted by Renshaw cells over the motor cells in the spinal cord. As a result, any slight stimulus such as noise, light or even breeze causes a violent reflex action producing general contraction of the muscles. Brucine exerts the same physiological actions but of much milder degree. The amount of loganin that is present in the seeds is too small a quantity to produce any substantial toxic effects.

SYMPTOMS AND SIGNS (Table 41.1)

If the seeds are taken as a whole, they have no poisonous action on account of the hard pericarp that cannot be dissolved by the digestive juices. When broken seeds are taken or the seeds are chewed, there is an intensely bitter taste in the mouth. The symptoms usually take some time to appear. If the alkaloid is

Table 41.1 Difference between Strychnine Poisoning and Tetanus

Trait	Strychnine poisoning	Tetanus
History	Of poisoning	Of injury
Fever	Not usual	Usual
Onset	Rapid	Comparatively delayed
Convulsions	Generalised and affect all muscles; chest fixed during convulsions	Starts usually with face, lock-jaw being an early manifestation; chest not fixed during convulsions
State in between convulsion	Complete relaxation in between the seizures	Relaxation between the spasms is never complete
Fatality	Usually in a few hours	Death rare in a few hours and may be delayed for several days
Laboratory investigation	Chemical analysis reveals the poison	Microbiological test positive

swallowed, the symptoms occur very rapidly. The patient is anxious and restless. He may complain of stiffness of muscles before typical strychnine convulsions occur. These convulsions are at first **clonic** (intermittent) and then **tonic** (sustained). All the muscles are affected simultaneously, beginning with the victim's neck and face. Arms and legs begin to spasm next, and the spasms become worse. During this stage, the muscles become stiff and rigid, so that the body is thrown into an arch. This condition is known as **opisthotonus**. Sometimes, the body-curve is in the opposite direction or sideways and known as **emprosthotonus** and **pleurosthotonus**, respectively. The chest becomes more or less fixed so that the breathing is embarrassed and cyanosis ensues. Blood-stained froth may be seen at the mouth. The contraction of muscles of the face causes widening of the mouth as the angles of the mouth are drawn wide apart with creases appearing in and around the eyelids (a state known as **risus sardonicus**). The eyes appear prominent and staring, and pupils become dilated. Consciousness is not lost, and the mind usually remains clear till death. Usually, the duration of convulsion varies from half to 2 minutes. In between the convulsions, the muscles are completely relaxed, and the patient looks well, though somewhat exhausted. The cyanosis lessens, cold perspirations cover the skin, dilated pupils may contract. After 5–15 minutes or on slightest impulse, e.g. a sudden noise or a current of air gently touching the patient, another convulsion occurs. In fatal cases, the convulsions rapidly succeed one another and increase in severity and in duration. The patient cannot breathe because the diaphragm and thoracic muscle are under spasm. Hypoxia causes medullary paralysis and death.

FATAL DOSE AND FATAL PERIOD

The fatal dose of strychnine is about 15–50 mg, i.e. the alkaloid content of one seed of nux vomica. The usual fatal period is a few hours.

TREATMENT

(The patient is kept in a dark, noiseless room so as to minimise stimulation):

- Convulsions may be controlled initially with diazepam 0.1–0.5 mg/kg IV slowly and then phenobarbital IV. If these prove ineffective, consider general anaesthesia and/or muscle relaxation by using curare, gallamine or pancuronium bromide.
- Short-acting barbiturates like pentobarbital sodium or sodium amytal are antidotes to strychnine and should be given in dose of 0.3–0.6 gm IV.
- Stomach wash with warm water and dilute solution of potassium permanganate/tannic acid may be considered as soon as the patient is under the influence of anaesthesia/sedation. However, it needs be carried out cautiously. A suspension of activated charcoal may be administered to adsorb strychnine, which should be removed later.
- Acidification of urine will increase excretion of strychnine.
- Tracheostomy with oxygen supply may be necessary and helpful.

POSTMORTEM APPEARANCES

The postmortem appearances are those of asphyxia. Rigor mortis appears early but is not necessarily prolonged. Occasional rupture of stretched muscles may be there. Haemorrhages are sometimes found under the peritoneal coat of the stomach. The remains of the seeds may be found in the stomach. Strychnine is an extremely stable substance, not likely to be destroyed by putrefaction and can be detected even some years after death (strychnine was recovered from the stomach, duodenum and liver of a female exhumed 4 years and 9 months after burial). In deaths from suspected strychnine poisoning, brain and spinal cord need to be preserved for analysis in addition to usual viscera and blood. Failure to detect strychnine is not an absolutely conclusive evidence that the death has not been caused by its poisoning as it is just possible that if death has occurred from a minimum dose of strychnine, and the victim has lived for sometime after its administration, complete elimination of the poison might have taken place before death.

MEDICOLEGAL ASPECTS

Most fatalities from strychnine poisoning are accidental. Strychnine poisoning has occurred through mistakes in prescriptions. Strychnine is precipitated in an alkaline medium and may settle to the bottom of a bottle of medicine and may cause poisoning. Homicide with strychnine is rare because of its intensely bitter taste and the convulsions that follow rapidly after ingestion. Suicide with strychnine is rare because of painful death. Sometimes, nux vomica is used as a love philter and an aphrodisiac and may be the cause of accidental poisoning. Strychnine is also used for killing stray dogs, as a rodenticide and sometimes as an arrow poison. Other arrow poisons include *Abrus precatorius*, crotin, *Calotropis*, snake venom, aconite and curare.

It is said to be a cumulative poison. This may be due to the slowness with which it is excreted, one effect produced by it being to contract the renal vessels, and thus, tending to interfere with its own elimination.

Harley records an unusual case in which an infant at breast, suffered from symptoms of strychnine poisoning due to the result of the medical administration of strychnine to the mother, who remained unaffected. (About 80% of the strychnine taken is oxidised mainly in the liver. Therefore, in cases of fatal poisoning, strychnine is found especially in liver and kidneys, and an unabsorbed portion of it is generally found in the stomach and its contents. It is excreted to some extent in the bile, milk, saliva, and possibly in the sweat. Surprisingly, as reported, traces of strychnine have been detected in the organs in fatal cases of nonstrychnine poisoning where strychnine had been administered as a remedial agent 2 or 3 days prior to death. It is, therefore,

advisable to bear such points in mind while drawing inference from the report of the chemical examiner.)

Peripheral Nerve Poisons

These act primarily on the end plates of the motor nerve terminals. Poisoning from them is of rare occurrence. The important poisons in this group are curare and conium.

CURARE

This is also known as **urare and woorara**. Curare is a black resinoid mass, almost wholly soluble in water and is obtained from the bark and wood of the plant ***Strychnos curare***. The active principles are the alkaloids **curarine and curine**. Curarine occurs as a yellowish brown powder or in deliquescent prisms with an intensely bitter taste. It is soluble in water and alcohol. It selectively paralyses motor nerve endings in voluntary muscles by interfering with the production of acetylcholine. When swallowed, it usually causes no symptoms of poisoning. When introduced into the wound, it acts like coniine, paralysing the motor end nerves. Its poison is activated if injected or administered intravenously.

Symptoms and Signs

Curare affects the muscles of the toes, ears and eyes, then those of the neck and limbs, followed by muscles of breathing. Death occurs as a result of respiratory paralysis. The symptoms occur rapidly and therefore demand energetic treatment. In large doses, there is a central action too on the nervous system, which may produce a short phase of excitation with muscular movements and even convulsions, followed by depression with loss of consciousness and respiratory failure.

Fatal Dose and Fatal Period

Fatal dose is 30–60 mg. The fatal period may extend to a few hours.

Treatment

Atropine 0.6–1.2 mg followed by 1–2 mg of physostigmine is recommended. Artificial respiration, oxygen inhalations and stimulants.

Postmortem Appearances

These are those of asphyxia.

Medicolegal Aspects

Poisoning from curare is mainly accidental. Curare is used as an auxiliary to general anaesthesia (frequently with cyclopropane) to bring about muscle relaxation and to prevent injury in shock therapy in certain mental diseases. Curare may be used in strychnine poisoning. It has been used as a poison for arrows and for blowgun darts in Central and South America.

Conium maculatum (HEMLOCK—THE SOCRATIC POISON)

Common names: California fern/Deadly hemlock/Herb bonnett/Kill cow/Muskrat weed bunk/Nebraska fern/Poison parsley/Snake weed/Spotted hemlock/Spotted parsley/Wode whistle. In Europe, it is called 'fool's parsley'.

Also known as **spotted hemlock**, it is an umbelliferous plant and is so-called from the purple spots on its stem. It grows in waste places. The whole plant has a mousy odour, which is accentuated by crushing the leaves or stems. (The odour is partly attributable to the volatile coniine. Prolonged inhalation of the odour is said to cause narcosis.) All parts of the plant are poisonous. The toxic properties are due to alkaloids coniine and methyl coniine, which cause paralysis of the motor nerve terminals in the muscles, slowly spreading to the motor cells of the cord and brain.

Symptoms and Signs

The fresh leaves have a nauseating taste and unpleasant mousy odour. Therefore, breath may have a mousy odour. Ingestion causes burning in the mouth and throat, gastric inflammation, vomiting, diarrhoea, distressed respiration, increased and later slow pulse, mental confusion and progressive motor paralysis extending upwards from the extremities. Death may occur from respiratory paralysis.

Fatal Dose and Fatal Period

Fatal dose is 60 mg of coniine. The fatal period may extend to a few hours.

Postmortem Appearances

These are those of asphyxia. The remains of the roots or leaves should be looked for in the stomach. When these are mixed with caustic potash and crushed in a mortar, they give a peculiar mousy odour.

Medicolegal Aspects

Poisoning may be caused by ingestion, injection or even inhalation as coniine is a volatile alkaloid. Poisoning by it is mostly accidental, chiefly from the plant being mistaken for parsley or some other harmless herb. Falck searched 17 cases recorded in medical literature, of which 14 were accidental (chiefly from the plant being mistaken for 'parsley' or some other harmless herb). One case is recorded of a child who died of poisoning from blowing whistles made of conium twigs.

The ancient Greeks used the juice of the fruit or the infusion of the leaves as a state-poison. It was administered to Socrates, the Greek philosopher, in 399 BC as a form of execution. Though the fatal dose is quite meagre, but obviously the 'kind jailor' might have given him vastly more than needed for lethality.

"And though I have been talking at such length, comforting you and myself with the assurance that when I have drunk the poison, I shall not remain here, but shall go to a better place…". These were the last words of Greek philosopher Socrates (470–399 BC) before he drank hemlock.

He was condemned to death for his 'crime' of introducing new divinities.

The root of the plant resembles the wild carrot and its seeds have been mistaken for anise. Sometimes, quail may eat poison hemlock seeds and pass on the poison to a human who consumes the flesh. This type of **secondary hemlock poisoning** can cause diarrhoea, vomiting and paralysis in humans.

42 CHAPTER

Cardiac Poisons

After going through this chapter, the reader will be able to describe: Source, characters, mechanism of action, metabolism, features and management of nicotine poisoning | Nicotine replacement therapies | Medicolegal aspects of nicotine poisoning | Source, characters, mechanism of action and features of aconite and oleander poisoning

These are poisons having main action on the heart, either directly or through the nerves. Nicotine, aconite, oleander and digitalis are important in this group.

Nicotine

Nicotine is an alkaloid found in several species of tobacco and an extremely fast-acting poison. While there is no medicinal use for nicotine, some of its derivatives are used as botanic insecticides; in fact, nicotine is the oldest insecticide known. *Nicotiana tabacum* is the most common species, which grows all over the world and is responsible for widespread narcotic habits in the world. The whole plant is poisonous except the ripe seeds. The dried leaves (***tambaku***) contain 1–8% of nicotine and are used in the form of smoke or snuff or chewed. The leaves contain toxic alkaloids **nicotine** and **anabasine** (which are equally toxic). Dangers of poisoning may occur from eating the large leaves in a salad, using infusions for enemas or absorbing the alkaloid through the skin during harvest.

Nicotine is a colourless, hygroscopic liquid alkaloid. It is used extensively in agricultural and horticultural work, for fumigating and spraying, as insecticides, etc. The use of tobacco leaves to create and satisfy nicotine addiction was introduced to Columbus by Native Americans and spread rapidly to Europe. The use of tobacco as cigarettes, however, is predominantly a twentieth century phenomenon, as is the epidemic of disease caused by this form of tobacco. Smoking is more prevalent in those with lower education and income, in most ethnic groups, and is especially high in psychiatric patients including those with other substance-use disorders.

Unburned cured tobacco contains nicotine, carcinogens and other toxins capable of causing gum disease and oral cancer. When tobacco is burned, the resultant smoke contains (in addition to nicotine) carbon monoxide and many other compounds that result from volatilisation, pyrolysis and pyrosynthesis of tobacco and various chemical additives used in making different tobacco products. The aggregate of particulate matter, after subtracting nicotine and moisture, is referred to as **tar**. The vapour phase contains carbon monoxide, respiratory irritants and ciliotoxins as well as many of the volatile compounds responsible for the distinctive smell of cigarette smoke. **The alkaline pH of smoke** from blends of tobacco utilised for pipes and cigars allows sufficient absorption of nicotine across the oral mucosa. Therefore, smokers of pipes and cigars tend not to inhale the smoke into the lung, confining the toxic and carcinogenic exposure largely to the upper airway. **The acidic pH of smoke** generated by the tobacco used in cigarettes dramatically reduces absorption of nicotine in the mouth, necessitating inhalation of smoke into the larger surface of the lungs in order to absorb quantities of nicotine sufficient to satisfy the individual's need.

ACTION AND METABOLISM

Nicotine binds stereo-specifically to nicotine receptors (acetylcholine receptors), which are present throughout the body particularly in the central nervous system, spinal cord, neuromuscular junctions, chemoreceptors of carotid and aortic bodies, autonomic ganglia and adrenal medulla. At moderate doses, nicotine stimulates the reticular activating system with resultant favourable effects on memory, attention and anxiety. However, higher doses lead to tremors and convulsions through disinhibition mechanism.

- Stimulation of vagal centres in medulla causes nausea and vomiting. However, lowering of sphincter pressure and increased acid secretion leads to gastro-oesophageal reflux.
- Through direct actions on receptors present in the endocrine glands, as well as through stimulation of neurohumoral pathways in the CNS, nicotine accentuates release of catecholamines, vasopressin, growth hormone, cortisol, prolactin, ACTH and serotonin, etc.

- Nicotine suppresses appetite while increasing basal energy expenditure, leading to loss in weight.

Nicotine via cigarettes is rapidly absorbed directly into the arterial circulation and reaches the CNS in a few seconds. Peak behavioural and cardiovascular effects occur within a few minutes. Nicotine is metabolised via the liver and has a half-life of about 2 hours. Nicotine levels from smoking typically rise in the morning, plateau in the evening, and fall to near zero in the night. This pattern causes an acute tolerance such that the first cigarettes of the day are more potent than later cigarettes.

CLINICAL FEATURES

Mild to moderate intoxication presents with a burning in the mouth and throat along with the typical findings of cholinergic excess in the form of muscle fasciculations, miosis, salivation, lacrimation, urination, defecation, sweating, vomiting, increased pulmonary secretions, tachypnoea, bronchospasm, hypertension and bradycardia. (These are due to stimulation of nicotinic receptors at the parasympathetic ganglionic sites.)

Severe intoxication shows the initial stimulatory features mentioned above, which are rapidly followed by prolonged ganglionic and neuromuscular blockade resulting from persistent depolarisation. (Muscarinic findings are no longer produced with a ganglionic blockade as opposed to organophosphate poisoning where muscarinic effects continue to be produced by direct acetylcholine stimulation as the muscarinic effectors are not subject to a depolarisation blockade.) The patient rapidly shows hypotension and seizures. Death may occur rapidly due to vagal excitation and standstill of the heart. It may occur after some hours due to exhaustion of the nervous system and respiratory failure.

Chronic poisoning may result from the continued use of tobacco by chewing, smoking or from exposure to nicotine (which occurs during processing or extraction of tobacco during mixing, storage or spraying of insecticides containing nicotine). Chronic cough, bronchitis, laryngitis and pharyngitis are common in persons who chew tobacco. Those who handle tobacco suffer from dermatitis. In the habitual smoker, there may be amblyopia, narrowing of the field of vision and some blurring etc. Habitual smoking is reported to lead to **tobacco heart**—a condition characterised by irregularity, extrasystole and occasional attacks of pain suggesting angina pectoris.

FATAL DOSE

In adults, the lethal dose of nicotine is 40–60 mg (about 20 gm of tobacco). Nicotine content in regular cigarettes averages about 15–20 mg per cigarette; cigars range from 15 to 40 mg. Smoking usually results in absorption of only 50–150 µg of nicotine per puff or 1–2 mg per cigarette and, therefore, is not a cause of intoxication. Eating tobacco is not generally a serious toxic risk, since the stomach does not absorb nicotine well from cigarettes.

NICOTINE WITHDRAWAL

It is manifested by changes in mood, insomnia, difficulty in concentrating, restlessness, decreased heart rate and weight gain. The weight gain is due both to increased eating and the loss of nicotine stimulation of metabolism. Craving is common and increased coughing, and poor performance on vigilance-tasks can occur. The syndrome is worse in cigarette smokers, in users of smokeless tobacco and mild in users of nicotine replacement products. Most withdrawal symptoms peak at 1–3 days and last 3–4 weeks.

Abstinence can also have pharmacokinetic effects. Non-nicotine chemicals in tobacco smoke activate cytochrome P450 enzymes, thereby decreasing the levels of several medications. Therefore, smoking cessation may increase the concentrations of these medications, for example, haloperidol, clozapine and fluvoxamine, etc.

TREATMENT

Perform lavage and instill activated charcoal. In the case of alkaloid ingestion, a lavage should be carried out quickly as nicotine is rapidly absorbed. The lavage with potassium permanganate has been suggested.

In the initial phases or in mild to moderate intoxication, atropine is of value. Nicotine is completely eliminated from the body within 16 hours, so the victims, who can be kept alive for that period, may survive. Mecamylamine (Inversine) is a specific antagonist of nicotine but is available only in tablets. (Tablets are not suitable for a victim who is vomiting, has low blood pressure or is having convulsions.) Supportive treatment for various systems is advised.

Nicotine Replacement Therapies

All nicotine replacement therapies are helpful in cessation rates, presumably because they reduce nicotine withdrawal. Replacement therapies use a short period of maintenance (6–12 weeks) often followed by a gradual reduction period (6–12 weeks). They include the following:

- **Nicotine gum** (nicotine polacrilex): Popularly known by its trade name Nicorette, this is a nicotine resin that is used to help people stop smoking. It gradually releases nicotine when chewed. However, chewing too much of the gum will release too much nicotine and cause nausea. Acidic beverages (coffee, tea, soda and juice, etc.) should not be used before, during or after gum use because these decrease absorption. Adverse effects are minor, and include bad taste and sore jaws.
- **Nicotine patch:** Another innovation in the stop-smoking arsenal, the nicotine patch became available in the United States in 1994. In this form, nicotine is gradually absorbed through the skin as a means to help a smoker stop using cigarettes.

In 1996, a **nicotine nasal spray** and in 1998 a **nicotine inhaler** became available. Nicotine inhaler was designed to

deliver nicotine to the lungs, but the nicotine is actually absorbed in the upper throat. Resultant nicotine levels are low. The major asset of the inhaler is that it provides a behavioural substitute for smoking.

Various forms of **nicotine replacement therapy** differ in terms of route of administration and speed of absorption. Further, they also offer varying situational response to craving and replacement of cigarette smoking. No such therapy gives the high concentration arterial bolus of nicotine, characteristic of cigarette smoking (because of the large surface area of the lungs the mildly acidic smoke of cigarettes is absorbed almost immediately and completely on inhalation, giving rise to high concentration arterial nicotine boli, which usually reach the brain within seconds). This coupled with the fact that there are no toxic tar and gas phase components in these therapies that are there in cigarette smoking gives them a reassuring safety profile.

MEDICOLEGAL ASPECTS

- Tobacco is used in various ways, e.g. by chewing, swallowing, decoction or by smoking. The decoction is used extensively in agricultural and horticultural work. Poisoning is mainly accidental. It may occur from the absorption through the skin when applied as a poultice for ulcers or through mucous membrane when used as an enema. For malingering, tobacco leaves are soaked in water for some hours and placed in axillae and held in position by bandage. Malingerers may use it for avoiding duty. Nicotine has occasionally been used for suicidal or homicidal purposes. For homicidal purposes, tobacco or snuff prepared from it may be administered along with beer or other drinks.
- Other major forms of tobacco-use are moist snuff deposited between the cheek and the gum, chewing tobacco, pipes and cigars, and ***bidi*** (tobacco wrapped in tendu or temburni leaf and commonly used in India) and clove cigarettes. Oral tobacco use leads to gum disease and can result in oral cancer. All forms of burned tobacco generate toxic and carcinogenic smoke similar to that of cigarette smoke. The differences in disease consequences of use relate to frequency of use and depth of inhalation.
- Although not widely known, initiation and cessation of tobacco use are as heritable as alcoholism. Some of the genetic effects are shared with alcohol and some are specific to tobacco. The biological and behavioural mechanisms for genetic effects on tobacco use are not known.

NON-NICOTINE PHARMACOLOGICAL TREATMENT

Many other drugs to aid 'smoking cessation' have been advocated. Signs of promise seen in 'clonidine' (alpha-2-adrenergic agonist that has been found useful in alcohol and opiate withdrawal also) have been offset by an unacceptable side-effect profile. Recently, 'bupropion', an atypical antidepressant with some nonadrenergic and dopaminergic activity, became the first non-nicotine medicine licensed for smoking cessation in the United States, Canada and Mexico. There has been evidence that bupropion and the skin patch have addictive effects in enhancing outcomes.

Aconite

The term 'aconite' refers to the genus Aconitum, of which there are several species. The name may come from the Greek 'akonitos' (meaning without struggle or without dust), or from the Greek city 'Acona', where a naturalist in the third century once identified the plant. Other sources suggest that the name comes from the hill of Aconitus, where Hercules fought with Cerberus, the three-headed dog who guards the entrance to Hades. Saliva from this dreaded dog's mouth dripped onto monk's hood, making it a deadly poison. Before the birth of Christ, this extremely poisonous plant was dubbed as 'queen mother of poisons'. All the species of the plant are poisonous, and some so extremely so that the general Indian vernacular name for them is ***Bish*** or ***Bikh***, meaning 'the poison'.

The most poisonous species are: ***Aconitum ferox*** and ***Aconitum napellus***. The former is a native of Himalayas, and its root forms most of the aconite root of Indian bazaars. It is believed to be even more actively poisonous than the latter. *Aconitum napellus* (monk's hood/wolf's bane/blue-rocket/bear's foot/friar's cap/soldier's cap/helmet flower, etc.) is a common plant in England and also grows in the Himalayas. It is perennial, two to three feet in height, with dark-green digitate leaves and an erect terminal spike of blue helmet-shaped flowers **(hence called monk's hood)**.

Less poisonous species of aconite are *Aconitum palmatum* in the eastern temperate Himalayas, *Aconitum heterophyllum* in the North-Western Himalayas. The root of the former is known in the vernacular as *Bikhma/Bishma/Wakhma/Vakhma* and of the latter as *Atis* (Hindi)/*Ativisha* (Marathi)/*Ativadayam* (Tamil)/*Atavakha-inkali* (Gujarati). The roots of these are smaller than those of the stronger species.

All varieties and all parts of the plant are poisonous, least when young, more so when seeds ripen and most when bloom. The roots of *Aconitum napellus* and *Aconitum ferox* are commonly used. **The dry root** is conical or tapering in shape, usually shrivelled and with longitudinal wrinkles. It is 5–10 cm long, 1–2 cm thick at the upper extremity and dark brown externally. When freshly cut, it is whitish but becomes pink on exposure to air. It has no odour but somewhat sweetish taste, which gives it the name ***mitha bish*** (sweet poison). The same root, after subjection to some soaking process, generally stated to be soaking in oil (hence called *mitha-teliya*) and cow's urine. After soaking, the root becomes black, heavy, hard and brittle with a strong offensive odour. **Aconite root may be confused with** horse radish. Horse radish root is not very much conical like aconite root. Aconite, when tasted, causes tingling, numbness of tongue,

mouth and lips. But horse radish is pungent to taste. Aconite, when cut, shows whitish surface, which slowly reddens but horse radish remains white.

ACTIVE PRINCIPLES AND ACTION

Aconitine is the chief alkaloid, one of the most virulent of known poisons. Other less active alkaloids include pseudoaconitine, aconine, picratonitine, benzoylamine, neopelline, etc. Aconitine or aconite root itself first stimulates the sensory nerves, producing tingling and then paralyses the sensory nerve terminals, causing numbness. It produces similar effects on the motor nerves and centres of the medulla and cord, while the higher cerebral centres are little affected. The motor ganglia of the heart are paralysed, the respiratory centre is slowed, death being usually due to arrest of respiration.

SYMPTOMS AND SIGNS

The clinical manifestations of aconite poisoning are swift in onset and are rarely delayed by an hour or so:

- The ingestion of the root or tincture of aconite produces a feeling of warmth in the mouth and throat which soon becomes a tingling or prickling sensation, eventually extending to the skin. The smarting of the skin is replaced by numbness and the cutaneous sensibility is reduced as outlined under the mechanism of action. This tingling followed by numbness is a characteristic symptom of aconite poisoning.
- The tingling sensation is followed by salivation, nausea, vomiting, etc.
- Besides its paralysing action on the motor nerves (or centres) and on the heart, other symptoms may include muscular weakness (the patient staggers if he attempts to walk), slow and weak respiration, slow, weak and irregular pulse, etc. Large doses also act directly on the heart producing arrhythmias.
- The pupils in the early stages alternately contract and dilate (**hippus**) but become dilated in the later stage. Intermittent visual disturbances can include blurred vision or colour patches in the visual field.
- Death may occur from shock or syncope, but usually occurs from asphyxia due to paralysis of the respiration. Convulsions may precede death. The mind usually remains unaffected.

FATAL DOSE AND FATAL PERIOD

One gram of the Indian root, 250 mg of the extract, 20–30 ml of tincture or 4 mg of alkaloid may be fatal. The shortest fatal period reported is three-quarters of an hour, the longest 24 hours and the average being about 6 hours.

TREATMENT

Milk or activated charcoal are advocated that delay absorption.

- Stomach is washed with warm water and then with potassium permanganate or a mixture of iodine and potassium iodide.
- Atropine may be given to avoid vagal inhibition of the heart.
- Artificial respiration and oxygen inhalation to combat respiratory embarrassment.

POSTMORTEM APPEARANCES

Pallor of the mucous membrane of the mouth may be present. Mucous membrane of the stomach is inflamed. When crushed root is taken, remains of the root may be present in the stomach. Congestion and engorgement of the brain and lungs. Aconitine is extremely unstable alkaloid and is destroyed by decomposition.

MEDICOLEGAL ASPECTS

Monk's hood is a strikingly beautiful plant rich in myth and medicine, history and magic. In mythology, monk's hood formed the cup that Medea prepared for Theseus. In Rome, Nero ascended to the throne after poisoning Claudius by tickling his throat with a feather dipped in monk's hood. While it is named for the shape of its flowers, it was so associated with political intrigue among the ranks of the Roman Catholic clergy. It is held that Romeo (of Shakespeare's Romeo and Juliet) committed suicide with monk's hood.

- Poisoning is relatively more common from accident rather than from homicide or suicide. Accidental poisoning may arise from therapeutic applications of liniment containing aconite or drinking them accidentally. The root has been eaten in mistake for horse radish. Poisoning has resulted from remedies adopted by quacks.
- Homicidal cases are not infrequently reported but considering the ease with which aconite can be obtained, they are not that frequent as one might expect. It is sometimes administered to the victim with betel leaf to disguise its tingling taste.
- The root of aconite is extensively used by tribals as an arrow poison. The use of root as a cattle poison has also been reported. Sometimes, the root is used as an abortifacient.
- The Lepchas of Sikkim have a saying that aconite is 'useful to hunters for destroying tigers and elephants, useful to the rich for putting troublesome relatives out of the way and useful to jealous husbands for destroying faithless wives.' The Lepchas poisoned the water supply of a detachment of British troops with powdered aconite root during the expedition of 1887.
- In view of the minute quantity of the poison usually used to produce a fatal result, and in view of its liability to decompose, it is probable that it has never been detected after absorption into the tissues. It may be found in the contents of the stomach before absorption, and also, more frequently, may be detected in the vomit. In a case that occurred in the United Provinces, it was detected in a stain on the pyjama of a woman, but it could not be found either in the vomit or in the viscera. The vomit in this case had been mixed with wood ashes. Such ashes contain a quantity of alkali,

which is known to decompose aconite. Dr. Hankin, therefore, recommends the mixing of the vomit and wood ashes with a mixture of two parts of rectified spirit and one part of acetic acid, which has the power of checking this decomposition.
- Sometimes aconite is added to Indian liquors to increase the intoxicating effect and causes poisoning.

Aconite offers following advantages **as a homicidal poison**:

- It is cheap and easily available.
- Its lethal dose is small and the fatal result also follows in a short time.
- Its colour can be disguised by mixing it with pink coloured drinks.
- It can be mixed with sweets and its sweet taste can thus be disguised.
- Its taste can also be masked by giving it with betel leaves.
- Symptoms resemble natural disease (heart disease).
- The alkaloid is largely destroyed in the human system and therefore difficult to be detected by chemical analysis.

Oleander (*Kaner*)

The oleander plant is widely cultivated in India for its graceful flowers, which are used as offering in the temples. There are two varieties, namely ***Nerium odorum*** (white oleander) and ***Cerbera thevetia*** (yellow oleander). The white one bears white or pink flowers and is also called **true oleander** in contradistinction to **yellow oleander,** which bears yellow bell-shaped flowers.

Nerium odorum (WHITE OLEANDER/*KANER*)

The plant grows wild in all parts of our country. All parts of the plant are poisonous, containing several cardiac glycosides, namely **neriodorin, neriodorein** and **karabin,** the last one being so called after the vernacular name of the plant. The nectar yields poisonous honey. The principal action of neriodorin is similar to that of digitalis causing death from cardiac failure. Neriodorein has a picrotoxin-like effect, i.e. it causes muscular twitchings and tetanic spasms more powerful than those of strychnine. Karabin acts on the heart like digitalis and on the spinal cord like strychnine.

Symptoms and Signs

The plant is occasionally a source of contact dermatitis. Emanations from flowers may cause headache, nausea, dizziness and respiratory difficulty. Ingestion causes difficulty in swallowing, abdominal pain, vomiting, profuse frothy salivation and diarrhoea. Pulse is first slow, and later rapid and weak, blood pressure falls, respirations are increased, pupils are dilated. Muscular twitchings, tetanic spasms, lockjaw are the other manifestations.

Fatal Dose and Fatal Period

Fatal dose is 15–20 gm of the root. Fatal period usually is 24–36 hours.

Treatment

Stomach wash and symptomatic.

Postmortem Appearances

Organs are congested. Fragments of root may be present in the stomach. Petechial haemorrhages are usually seen on the surface of the heart. *Nerium odorum* resists heat and, therefore, can be detected even from the burnt dead body. **The other poison that resists heat is arsenic.**

Medicolegal Aspects

Suicide with *Nerium odorum* is common among villagers in certain parts of India. The root is used as a decoction for this purpose. The root is commonly used both locally and internally for procuring abortion. Accidental poisoning is sometimes met with when any part of the plant is used as a love philter or, when the decoction of the leaves is applied externally to reduce swellings. Homicide with *Nerium odorum* is rare. Its use as a cattle poison has been recorded (for this purpose, it is mixed with the fodder or a rag smeared with the juice of the root and thrust in the animal's rectum). Smoke from burning plant carries the toxic material and may cause serious illness.

Cerbera thevetia (YELLOW OLEANDER—*PILA KANER*/EXILE/BASTARD OLEANDER)

This plant is also highly poisonous. The active principles are the three glycosides, namely **thevetin, thevotoxin** and **cereberin**. Thevetin and thevotoxin are isolated from the kernels of the seeds. Thevetin and cerberin reside in the milky juice, which exudes from all parts of the plant. Thevetin is a powerful cardiac poison. Thevotoxin is less toxic and resembles the glycosides of digitalis in action. Cerberin has strychnine-like action.

Symptoms and Signs

The sap of the plant may cause inflammation in sensitive individuals. Chewing the bark or seed kernel causes a slight numbing sensation and feeling of heat in the mouth and purging. Ingestion causes burning pain in the mouth, dryness of throat, tingling and numbness of tongue, vomiting, diarrhoea, headache, giddiness and dilated pupils. Loss of muscular power is another important finding. Pulse is rapid, weak and irregular, and blood pressure low. Heart block, collapse and death from peripheral circulatory failure.

Fatal Dose and Fatal Period

Fatal dose is 8–10 seeds or 15–20 gm of root. Death has been recorded in 2–3 hours in an adult who took his meal mixed with powdered root.

Treatment

Stomach wash. Sodium molar lactate transfusion with glucose and atropine are helpful. Rest of the treatment is symptomatic.

Postmortem Appearances

Not specific. Stomach and duodenum may be congested and may show fragments of seeds. Subendocardial ecchymoses may be the other finding. Yellow oleander resists putrefaction and can be detected long period after death.

Medicolegal Aspects

Suicidal uses are common in some pockets of rural West Bengal.

Homicide is rare due to the requirement of high dose. Accidental poisoning is comparatively common in children by eating the seeds. Root is used as an abortifacient. It is also used as cattle poison (for this purpose, the seeds are crushed and fed to the animal with corn or bread).

Cerbera odollam (DABUR/DHAKUR/PILIKIRBIR)

This plant is closely allied botanically to *Cerbera thevetia*. It is a small plant or a shrub that grows wild all over India. The leaves are dark green, fleshy and lanceolate. The flowers are white like those of jasmine. The fruit resembles a mango and has a thick fibrous mesocarp, which encloses usually a single seed. The seed is flattened and ovoid and contains two kernels, which are pearly white but when dry, it may have a bluish tinge. Milky acrid juice exudes from all parts of the plant. **The active principles** are a glycoside cerberin and a weak alkaloid cerebroside having a digitalis like action.

Symptoms and Signs

Initial symptoms are gastrointestinal. Cardiac toxicity may occur within a couple of hours of ingestion. There is bitter taste, nausea, severe vomiting, abdominal pain and in a few cases, diarrhoea. General weakness, blurring of vision, sinus bradycardia, irregular respiration are the other symptoms. Death usually occurs from heart failure.

Fatal Dose and Fatal Period

Kernel of one fruit is fatal. Fatal period usually is 1–2 days.

Postmortem Appearances

Organs are usually congested. Subepicardial, subendocardial and subpleural petechial haemorrhages are usually found. Stomach mucosa is congested with submucous haemorrhage.

Treatment

Stomach wash. Atropine is recommended. Rest of the treatment is symptomatic.

Medicolegal Aspects

For suicide, the kernels are taken as such or after grinding it with jaggery or molasses or by preparing a curry with it. The powder kernel may be added to alcohol with homicidal intent. Bark, leaves and milky juice are used as emetic and a purgative. Accidental poisoning may occur in children who eat the fruit by mistake for an edible one.

43 CHAPTER

Agro-chemical Poisoning

After going through this chapter, the reader will be able to describe: Classification and toxicity of various pesticides | Toxicology, management and medicolegal aspects of organophosphates, carbamates, organochlorine group of compounds | Toxicology of herbicides like paraquat, diquat, and rodenticides etc.

Pesticides

The United States Environmental Protection Agency (USEPA) defines pesticide as any substance or mixture of substances intended for preventing, destroying, repelling or mitigating any pest. It may also be described as any physical, chemical or biological agent that will kill an undesirable or troublesome animal, plant or micro-organism. **Pesticide is a generic term** for a variety of agents that may be classified more specifically on the basis of the pattern of use and organism killed. In addition to three major agricultural groups (insecticides, herbicides and fungicides), many others have been described as detailed ahead.

In the post World War II era, a plethora of insecticides, fungicides, herbicides and other chemical agents were introduced. Despite man's persistent efforts to develop mechanisms of actions in selectivity and specificity of these agents towards certain species while reducing toxicity to other forms of life, all pesticides possess an inherent degree of toxicity to human beings. [All the chemical insecticides are neurotoxicants and act by poisoning the nervous systems of the target organisms. While the peripheral nervous system (PNS) of insects is not as complex as that of mammals, there are many similarities. These chemicals elicit similar effects in higher forms of life including humans. The target sites and/or mechanism(s) of action may be similar in all species, only the dosage (level of exposure and duration) will dictate the intensity of biological effects.]

Incidence of poisoning, as reported, is 13-fold higher in developing countries than in highly industrialised nations, which consume 85% of world's pesticide production. Most pesticide-related poisonings in developing nations can be attributed to lack of training in their use, poor regulatory/legislative control towards their access, and carelessness in providing protection to the body during their application. (Minimum protection of certain parts of the body can markedly reduce exposure to an agent. Literature showing the absorption of pesticides applied to the skin of different areas of the human body has revealed marked regional variations in per cutaneous absorption, with the greatest uptake by the scrotal region, followed by the axilla, forehead, face, scalp, the dorsal aspect of hands, the palm of the hand and the forearm in decreasing order.)

Several modes of exposure may be recognised in context with poisoning, namely (*i*) accidental and/or suicidal poisonings, which cannot be prevented through legislation or preaching; (*ii*) occupational resulting from manufacturing, mixing/loading, application, harvesting and handling of crops; (*iii*) bystander exposure resulting from off-target drift from spraying operations; and (*iv*) the general public exposure who consume food items containing pesticides residues as a consequence of incorrect application rate resulting in residue concentrations above established tolerance levels. (The identification of pesticide-related adverse health effects in the general population, consuming inadvertently pesticides daily via food and water, is extremely difficult. As reported, the residue levels in these media are often lower than those encountered in occupational or bystander exposure. Any biological effects resulting from such low-level exposure are unlikely to be distinctive and any causal relationship with a particular chemical or class of chemicals is likely to be flimsy and confounded by many other endogenous and exogenous factors attending a particular lifestyle.)

Despite the current controversy over pesticide use and the presence of low levels of residues in food, ground water and air, these agents remain integral component of agricultural protection. The risk–benefit ratio is highly weighted in favour of their continued use for the control of organisms causing devastation to crops and health-protection programmes. Hence, as long as pesticides continue to be used, accidental and/or intentional poisoning of wild life, domestic stock and humans can be anticipated. Between two extremes of overwhelming use and total ban, lies a situation/proposition advocating the careful and rational use of these beneficial agents/chemicals.

Dr. Vishal Garg et al. conducted a retrospective study (1st April 2007 to 31st March 2009) at AIMSR, Bathinda to go through the trends of poisoning cases occurring in the rural areas of South-West Punjab. Out of 784 cases studies, 95 (12.1%) were of poisoning. The trends showed (*i*) more involvement of males (80.0%) than females (20.0%), (*ii*) preponderance of 21–30 years age group, (*iii*) dominance of rural over urban and (*iv*) suicidal manner of poisoning (65.3%) overriding accidental (34.7%). Aluminium phosphide was responsible for 36.8% of cases followed by insecticides (31.6%). The study concluded that maximum casualties were due to agricultural and domestic use of chemicals like organophosphorus compounds and aluminium phosphide. Easy availability, lack of knowledge in respect of handling of these chemicals, delay in hospitalisation and deficient emergency facilities in hospital set-ups in rural areas contributed towards such casualties.

HERBICIDES

Compounds that kill weeds/prevent growth of undesirable herbs or weeds in the field. Examples may include paraquat, diquat, atrazine, propazine, simazine, trichloroacetic acid and chlorophenoxy compounds.

FUNGICIDES

Compounds that kill fungi and moulds. Examples may include thiocarbamates, captan, captafol, bavistin, vitavax, hexachlorobenzene and sodium azide.

RODENTICIDES

('Rodent' implies a mammal of the order Rodentia, with strong incisors and no canine teeth, for example: rat, mouse, squirrel, beaver, porcupine. The word is derived from Latin 'rodent'—'gnawing'). Compounds that kill rats, mice, moles and other rodents are called rodenticides. Examples may include anticoagulants, thallium, phosphorus, vacor, cholecalciferol, arsenic, barium carbonate, bromethalin, red squill, strychnine and zinc phosphide.

ACARICIDES

Compounds that kill mites, ticks and spiders. Examples may include azobenzene, chlorobenzilate, tedion, etc.

NEMATICIDES

'Nematode' implies a worm of phylum Nematoda, with a slender unsegmented cylindrical shape. Nematicides kill nematodes, e.g. ethylene dibromide.

MOLLUSCICIDES

'Mollusc' implies any invertebrate of the phylum, mollusca, with a soft body and usually a hard shell, for example, snails, cuttlefish, slugs, mussels, etc. Molluscicides kill molluscs such as snails and slugs, e.g. metaldehyde.

MISCELLANEOUS PESTICIDES

Compounds of lead, copper and mercury, nicotine, hydrogen cyanide, methyl bromide, naphthalene, tetrachloroethylene, trichloroethane, dinitrophenol, dinitrocresol, dinitrobutylphenol, pentachlorophenol, chlorfenson and chloralose.

Organophosphates

Much of the success in the agricultural field in our country is due to the knowledge and use of agricultural insecticides. Their preparations are most popular on one side and on the other side, also take many lives every year, though mostly in the rural areas but also to a considerable extent in the urban areas.

MODE OF USAGE

In the agricultural fields, these are either used as aerial spray (mixed with suitable liquid or dust as the vehicle), or they are mixed with soil. When sprayed in air, absorption in the plants occurs through leaves and stems. When used by mixing with the soil, absorption occurs through the roots of the plant. When the insect sits on the plant, the poison is absorbed through its exoskeleton or when it eats the leaves of the plant, it consumes the poison along with. These chemicals act on insects and other arthropods by inhibiting cholinesterase, and their toxic effects on man are caused by the same mechanism.

CLASSIFICATION

Chemically, organophosphates are derived from phosphoric acid and two chemical groups are available: (*i*) **alkyl phosphates**, e.g. tetraethyl pyrophosphate (TEPP), hexaethyl tetraphosphate (HETP), octamethyl pyrophosphoramide (OMPA), dimefox, isopestox, sulfotepp, demeton, malathion (Kill bug; Bugsoline), etc. and (*ii*) **aryl phosphates**, e.g. parathion (Follidol; Kill phos; Ekato), paraoxon, methylparathion (Metacide), chlorthion, diazinon (Diazion; Tik 20) (Table 43.1).

Table 43.1 Fatal Dose, Fatal Period and Toxicity Rating of the Important Organophosphate Compounds

Compound	Fatal dose	Fatal period (hours)	Toxicity rating
TEPP	100 mg	0–6	06
Parathion	100 mg	0–6	06
Malathion	1 gm	0–6	04
OMPA	200 mg	0–6	–
Diazinon (Tick 20)	1 gm	0–6	04

ABSORPTION, FATE AND EXCRETION

Organophosphorus compounds are well-absorbed through the mucous membrane of GI tract, respiratory tract and through the skin. Parathion is stored in the body fat and is slowly released in the circulation, prolonging the duration of its toxic action. Malathion is metabolised in the liver by the esterases. A part of the metabolised product is excreted in the urine as phosphate. Parathion may be retained for a period of about a week and malathion for a period of more than a week (Table 43.1).

MECHANISM OF ACTION

Acetylcholine is a neurotransmitter that affects the preganglionic and postganglionic parasympathetic synapses (**muscarinic actions**), sympathetic preganglionic synapses including the adrenal medulla (**nicotinic actions**) and the neuromuscular junctions (**nicotinic actions**). It is also a transmitter in the central nervous system. At the synapses, it is hydrolysed by the enzyme, acetylcholinesterase. The toxic effects of organophosphates are due to the inhibition of acetylcholinesterase (**that is why they are called as cholinesterase inhibitors**), resulting in the excessive accumulation of acetylcholine at the synapse. This initially stimulates and later paralyses the cholinergic transmission in the CNS, autonomic ganglia, parasympathetic nerve endings, some of the sympathetic nerve endings and neuromuscular junctions.

CLINICAL FEATURES

The onset of features of organophosphate poisoning occurs most rapidly after inhalation and least rapidly if absorbed percutaneously. Massive exposure can produce problem within minutes. The various signs and symptoms of poisoning may be classified into the following categories (an acute garlic odour is a characteristic feature of poisoning):

- **Muscarinic features** (parasympathetic): These can be remembered using the acronym SLUDGE, denoting salivation, lacrimation, urination, defecation, gastrointestinal cramping and emesis. Bronchorrhoea and bronchoconstriction may be intense and may lead to disturbed respiratory functions. **Miosis** is a characteristic feature but is not present in all cases. The cardiovascular features include hypotension and bradycardia.
- **Nicotinic features** (somatic motor and sympathetic nerve endings): These features include muscle fasciculations, muscle cramps, fatigue, loss of deep tendon reflexes, paralysis. Tachycardia and hypertension may be there.
- **CNS features:** Various neurological features include severe headache, restlessness, tremors, ataxia, generalised weakness, emotional lability, confusion, coma, seizures and depression of the cardio-respiratory centre.

Intermediate Syndrome

This has been reported in some patients and is characterised by weakness of the upper extremities and neck musculature, cranial nerve palsies and secondary respiratory arrest. It may occur between 24 and 96 hours after ingestion.

Delayed Neuropathy

May sometimes occur 1–4 weeks after exposure. The symptoms occur distally and progress proximally. The neuropathy is usually mixed with the patients complaining paraesthesias and motor weakness. In a few cases, recovery may occur gradually.

FATAL PERIOD

The most common cause of death by organophosphate poisoning is respiratory impairment. Death usually occurs within the first 24 hours of ingestion in untreated cases. Complete recovery may occur if there is no hypoxic damage to the brain. However, delayed sequelae as mentioned above can occur.

DIAGNOSIS

At least 10 ml of blood should be taken with sterile precautions, using minimum amount of heparin to prevent clotting. The sample needs immediate centrifugation, separating the serum from the cells. Both should then be promptly refrigerated until cholinesterase determinations are performed because haemolysis introduces error into ChE activity measurements. Furthermore, ChE activity in the blood decreases with exposure to room temperature. Refrigeration (not freezing) usually results in retention of the ChE level as long as 3 weeks.

Normal Cholinesterase Values

Two types of ChE are clinically significant in humans. One is the specific enzyme, acetylcholinesterase, whose affinity is limited mainly to its naturally occurring substrate, acetylcholine, although it can hydrolyse several synthetic esters. Acetylcholinesterase occurs abundantly in the nervous tissue, muscles, glands and erythrocytes (red blood cell or **true ChE**). The second type, **plasma ChE** or pseudo-ChE, is relatively nonspecific and hydrolyses a wide range of naturally occurring and synthetic esters as well as acetylcholine. It occurs in plasma as well as in other tissues including the central nervous tissue. Under ordinary conditions, ChE levels are independent of age, race or cause of death. Although red cell ChE can vary as much as 5% from day to day, but it is free from changes from ordinary diseases, drug therapy and the like. However, the concentration of pseudo-ChE is much more labile than that of the red cell variety, and it is decreased in some liver and systemic diseases. Prolonged low-level exposure to ChE inactivators does not usually cause clinical illness until true ChE activity decreases to 20–25% of the individual's pre-exposure level, as gradual depletion in ChE activity is better tolerated than abrupt decline. If death does not supervene promptly, or if the victim survives, plasma ChE recovers much more rapidly than red cell ChE. Usually, only 1–3% of normal erythrocyte ChE activity returns each day, the enzyme re-appearing only as the new red cells enter the peripheral circulation.

- If the RBC cholinesterase level is < 50% of normal, it indicates organophosphate toxicity. However, false depression of RBC cholinesterase level is seen in pernicious anaemia, antimalarial treatment, haemoglobinopathies and in the blood collected in oxalate tubes.
- Depression of plasma cholinesterase level (to < 50%) is a less reliable indicator of toxicity, but is easier to assay and more commonly done. Being a liver protein, plasma cholinesterase activity is depressed in cirrhosis, malnutrition, infections and neoplasia.
- Cholinesterase at the motor end-plate can be demonstrated histochemically in the muscles kept at room temperature for 1–2 days, and up to several months in the tissues stored at 4–6° C. Fixation of tissue with phosphate buffered formalin and cold acetone for 24 hours or the embalming of the body does not affect the cholinesterase activity at the myoneural junctions.

TREATMENT

- Ensure the adequacy of the airways and circulation.
- **Decontamination:** After one dose of atropine, the patient may be decontaminated depending upon the route of administration/absorption. If **skin spillage** has occurred, it is imperative that the patient be stripped and washed thoroughly with soap and water. If **ocular exposure** has occurred, copious eye irrigation should be done with normal saline. If it is not available, tap water can be used. In case of **ingestion**, stomach wash can be done. Activated charcoal is beneficial.

Antidotes

- **Atropine** is the cornerstone of therapy, which competitively blocks the acetylcholine at the muscarinic receptors, ameliorating the parasympathomimetic effects of anticholinesterase compounds. Atropine has mild or negligible effects on the neuromuscular junction (nicotinic receptors), the autonomic ganglia (nicotinic receptors), and the CNS synapses protected by the blood–brain barrier. Initially, administer atropine in a dose of 2–4 mg for adults and 0.05 mg/kg for children intravenously, or, if necessary, intramuscularly. Repeat the dose every 5–15 minutes until there is a cessation of parasympathomimetic effects like bradycardia, salivation and tracheal secretions. (Do not consider the dilatation of the pupils as the end point for the use of atropine.) After the end point is reached, administer atropine in lower doses and at less frequent intervals so as to maintain atropinisation for 24–48 hours.
- **Pralidoxime** (pyridine-2-aldoxime methiodide; 2-PAM): It is a cholinesterase reactivator, which primarily acts to counter the nicotinic effects of organophosphates, though it can reverse some of the CNS effects also. Since it regenerates cholinesterase at all sites including muscarinic sites, its use is not contraindicated if only muscarinic effects are present. While it is advisable to begin pralidoxime therapy within 48 hours of poisoning, it can be administered even much later with beneficial effects. **Dosage:** For adults, 1–2 gm IV slowly over 10–15 minutes; while for children, the dose is 25–40 mg/kg to a maximum of 1 gm (too rapid administration may cause weakness, blurred vision, dizziness, headache, nausea and tachycardia). Dose may be repeated every 6–12 hours for 24–48 hours.
- **Diazepam** is also thought to be helpful through its central anticonvulsive properties and is effective against muscle fasciculations. It is recommended in doses of 5 mg intravenously every 10 minutes to a maximum dose of 15 mg.
- For the treatment of ventricular arrhythmias, **isoproterenol, lidocaine** may be used.

(To guard against mortality of the intermediate syndrome, keep all the patients under observation for 3–4 days.)

POSTMORTEM APPEARANCES

The changes are suggestive of asphyxia. **Externally:** Presence of cyanosis, deep postmortem staining, froth at the nose and mouth, which may be bloodstained. Kerosene-like smell due to the diluent of the poison may be perceived. **Internally:** The mucosa of the stomach and the intestine is congested; the stomach content may give a kerosene-like smell due to the vehicle or diluent used for the poison. Petechial haemorrhagic spots may be present at the subpleural and submucosal surface of other viscera. Gross congestion and oedema of the lungs usually present. Blood-stained froth in the respiratory tract is a common finding. Congestion of the other organs.

TOXICOLOGICAL ANALYSIS

Postmortem toxicology is usually conclusive in acute poisoning. The compound resists postmortem autolysis and can be recovered from putrefied bodies. Other poisons that resist putrefaction are arsenic, antimony, strychnine, *dhatura*, nicotine and yellow oleander, etc. Since alcohol masks the smell, these compounds have been used with alcohol for homicidal purposes. The viscera for chemical examination should be preserved in saturated saline in suspected cases of organophosphate poisoning.

The range of blood parathion concentrations in fatal cases may be from 0.5 to 34 mg/L with an average of 9.0 mg/L. Urinary excretion ranges from 0.4 to 78 mg/L with an average of 10 mg/L. Malathion is much less toxic, but deaths have been reported when given in greater dose. Depending upon the interval between death and postmortem examination, decreased cholinesterase in erythrocytes and at myoneural junctions may be demonstrable.

MEDICOLEGAL IMPORTANCE

Organophosphate compounds are extensively used as pesticides in agriculture. Suicidal poisoning is common in our country because of easy availability of the poison. Accidental poisoning occurs in manufacturers, packers, sprayers and other users.

A number of accidental deaths through contamination and leakage of these compounds to edible commodities have also been recorded (Kerala food poisoning cases). Homicidal poisoning is rare due to detectable smell of the substance used as diluent for the poison and due to alarming signs and symptoms that appear rather early. For homicidal purposes, they are usually mixed with alcohol to mask the smell.

Carbamates

They are as popular as organophosphates in their role as insecticides and share a number of similarities with the organophosphates. They are marketed in the form of dusts or solutions, such as aldicarb (Temik), aminocarb (Matacil), approcarb (Baygon), carbaryl (Sevin), carbofuran (Furaxdan). Absorption occurs through all routes.

MODE OF ACTION

Carbamates (like organophosphates) are inhibitors of acetylcholinesterase, but carbamylate the serine moiety at the active site instead of phosphorylation. This is a reversible type of binding, and hence symptoms are less severe and of shorter duration. Also, as carbamates do not penetrate the CNS effectively, toxic features related to CNS are not much prominent in the event of poisoning. In the carbamate poisoning, the measurement of cholinesterase activity in the RBC and plasma is unreliable as the enzyme activity returns to normal within a few hours, as stated earlier. (Therefore, blood samples must be drawn and assayed immediately so as to have some idea of poisoning.)

TREATMENT

An important differentiating point from organophosphates is that oximes are generally not recommended, while atropine can be given. Oxime therapy is usually not needed due to the rapid regeneration of cholinesterase. However, oximes are considered to offer some antidotal properties in patients exposed to aliphatic oxime carbamates (aldicarb, methomyl).

Organochlorines (Chlorinated Hydrocarbons)

These are no longer considered as an important class of **insecticides** in North America and Europe because of their property of remaining persistent in the environment, bioconcentration plus biomagnification in food chains, etc. Further, being lipophilic, these agents tend to accumulate in the adipose tissue, and cleansing from the systems occurs very slowly. However, in the developing countries, the agents are being used extensively and the risk–benefit ratio is highly placed in favour of their continued use for the control of insects harmful to the crop and human health. These agents can be absorbed transdermally, orally and via inhalation. Absorption by all these routes is affected by the solvents in which they are contained. The common solvents include kerosene, toluene and other petroleum distillates that themselves are toxic.

COMMON PREPARATIONS

- DDT (dichlorodiphenyltrichloroethane) and analogues: DDT is available in a technically pure form and in dry mixtures of several insecticides or solutions of one or more insecticides in various organic solvents.
- Benzene hexachloride group: Available as wettable powders, emulsions, dusts and solutions for use as a general garden insecticide. Gamma-benzene hexachloride (GBH) is used for the control of lice and scabies. Gamma-hexachlorocyclohexane (lindane) is another example.
- Cyclodienes and related compounds: Aldrin, dieldrin, endosulfan (thiodan), endrin, isobenzan, chlordane, chlordecone (kepone), mirex (dechlorane).
- Toxaphene and related compounds.

ABSORPTION, FATE AND EXCRETION

For the insects, chlorinated hydrocarbons are contact poisons and are absorbed through the exoskeleton. In the human beings, chlorinated hydrocarbons (except dieldrin) in dry powder form are poorly absorbed through the mucous membrane of GI tract. Dieldrin and endrin are absorbed well through the skin in dry powder form. When dissolved in kerosene and other solvents, all are readily absorbed both through skin and mucous membrane. These are mainly deposited in the body fat and also to a small extent in the liver, kidneys and brain.

MODE OF ACTION

DDT and analogues affect the sodium channel and sodium conductance across the neuronal membrane, especially of the axon. It acts chiefly on the cerebellum and motor cortex of the central nervous system. They also alter the metabolism of serotonin, norepinephrine and acetylcholine.

FATAL DOSES

For aldrin, dieldrin and endrin: 2–6 gm; for chlordane: 5–7 gm; for DDT and lindane: 15–30 gm.

CLINICAL FEATURES

The toxic effects of DDT may follow ingestion, extensive contamination of skin or prolonged inhalation. Following ingestion of toxic doses, salivation, nausea, vomiting and abdominal pain may occur within an hour. **The most common and serious feature** is the occurrence of seizures, which can occur without any prodromal features of GIT toxicity. (The organic solvents

present in many commercial insecticides decrease the convulsive effects of DDT and increase the depression of the CNS.)

Other features that are seen particularly in DDT intoxication are dizziness, myoclonus, weakness of legs, agitation and confusion, which may occur prior to or independent of seizures. Hepatic and renal damage may occur in rare cases. Death occurs due to respiratory failure.

CHRONIC POISONING

Long-term exposure to some of these compounds results in cumulative toxicity with manifestations such as weight loss, weakness, tremor, ataxia, abnormal mental changes and increased tendency to leucaemias, thrombocytopenic purpura, aplastic anaemia, etc.

DIAGNOSIS

Abdominal radiograph may reveal presence of certain organochlorines, which are radiopaque. Organochlorines can be detected in serum, adipose tissue and urine by gas chromatography.

TREATMENT

- The important dangers of an organochlorine overdose are hypoxia secondary to seizures, respiratory depression and aspiration of vomitus. Therefore, maintenance of ventilation should be given priority.
- Skin decontamination is essential because of the significant absorption of many organochlorines through this route.
- Seizures should be controlled with benzodiazepines, phenytoin or phenobarbitone.
- Cholestyramine, a nonabsorbable bile acid binding anion exchange resin, is effective in enhancing the faecal excretion of organochlorine compounds. Usual dose is 16 gm/day for several days. It can be mixed with fruit juice and given orally.

POSTMORTEM APPEARANCES

Deaths from such compounds are complicated by the presence of other insecticides and of solvents. In acute poisoning, the findings are those of asphyxia and congestion of GIT. The stomach usually gives kerosene-like smell (solvent commonly used). Pulmonary oedema is common. Froth, sometimes mixed with blood, may be present in the respiratory tract. In chronic poisoning, the effect is greater on the liver and kidneys, which may show necrosis and degenerative changes.

MEDICOLEGAL ASPECTS

Accidental poisoning is more prevalent in children who have access to the poison. It is also because tolerance for the poison is less in children. Accidental poisoning may also occur in persons who handle the poison. Suicidal poisoning is common, more in the rural areas. Homicidal poisoning is very rare because of presence of smell and involvement of large dosage. However, smell may be masked by administering the poison with alcohol. (Many of the signs and symptoms of poisoning may be due to the effects of the solvents like kerosene.)

Endrin

Of all the chlorinated insecticide compounds, endrin is the most toxic. It is chiefly used against pests of cotton, paddy, sugarcane and tobacco. It is a popular insecticide with action against a wide variety of plant pests and, therefore, **also known as plant penicillin**. It is sold in the market under the trade names of Endrin-We-16, Endox-DB 50, Endtox EC-20, Endrex, Tafdrin, etc. These products contain about 20–50% of endrin mixed with petroleum hydrocarbon, such as aromax, which smells like kerosene.

SYMPTOMS AND SIGNS

Toxic effects follow ingestion, inhalation or skin contamination. Their onset is quite rapid. The main symptoms are nausea, vomiting, abdominal pain, tremors, convulsions, oozing of froth (occasionally blood-stained) from mouth and nostrils, and severe dyspnoea. Later on, convulsions become severe followed by coma, which may terminate in death due to respiratory failure.

FATAL DOSE AND FATAL PERIOD

Fatal dose is 5–6 gm (by ingestion, it is three times as toxic as aldrin, dieldrin and ten times as toxic as DDT). Fatal period may extend from an hour to several hours.

POSTMORTEM APPEARANCES

These are suggestive of asphyxia. **Externally:** Kerosene-like smell may be noticed emanating from mouth and nostrils. Froth, occasionally blood-stained, may be present. **Internally:** Respiratory passages usually contain frothy mucus, and the tract is congested. Lungs appear large and bulky, and pulmonary oedema is a constant feature. GIT shows congestion. Stomach emits kerosene-like smell. Other organs are congested. (Endrin resists putrefaction and can be detected in the viscera quite sometime after death. Viscera should be preserved in saturated saline in suspected cases of endrin poisoning.)

MEDICOLEGAL ASPECTS

Endrin is freely available and comparatively cheap. It is, therefore, mostly used for suicidal purposes. Occupational and accidental exposure may also occur. Homicide is rare. It is reported to have been used for this purpose by mixing it with alcohol, especially 'toddy', which masks its smell. It may also be mixed with sweets or other food. The toxic dose being small, even if the victim recognises the smell while eating, toxic symptoms may ensue.

Herbicides

Herbicides are agents that are used to destroy wild plants. Although these are commonly used chemicals, they rarely cause poisoning. Most herbicides belong to two classes—bipyridyl and chlorophenoxy herbicides. The first class includes paraquat and diquat; of which paraquat is the most widely used and has significant toxicity. Diquat is only half as toxic as paraquat.

BIPYRIDYL HERBICIDES

Mode of Usage

Paraquat is a 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. Paraquat is produced commercially as a brownish concentrated liquid of the dichloride salt at 10–30% strength, under the trade name 'Gramoxone' and, for horticultural use, as brown granules called 'Weedol' at about 5% concentration. Human poisoning usually occurs either by deliberate self-ingestion of the liquid, or by accidental drinking from the unmarked/ill-marked or incorrectly marked bottles.

Absorption and Excretion

Five to 10% of the dose is absorbed, and the rest is excreted in the faeces. It is distributed to all the organs, but the highest concentrations are found in the kidneys and the lungs, followed by muscles from which paraquat can redistribute back into the circulation as plasma concentration decreases. Most of the absorbed paraquat is excreted unchanged in the urine within the first 24 hours but can be detected in urine up to several days after ingestion.

Clinical Features

Toxicity can occur either as an acute episode or as a subacute problem. The major effects of poisoning are corrosion of the gastrointestinal tract, renal tubular necrosis, hepatic necrosis, and pulmonary fibrosis.

- Potentially lethal poisoning is most common after ingestion of paraquat. Probably, no more than 5% of the ingested amount is absorbed, but absorption is rapid, the volume of distribution is high, and there is energy-dependent accumulation in some organs (notably the lungs). The poisoning results in burning and ulceration of the tongue, throat and oesophagus, followed by abdominal pain, vomiting and diarrhoea. One of the most peculiar clinical features is the formation of a pharyngeal membrane, which closely resembles that of diphtheria. The membrane affects the tongue also while the diphtheritic membrane generally spares the gums, floor of the mouth and the tongue. Death is likely within several hours to a couple of days with features of vomiting, diarrhoea, fluid loss, shock, coma, convulsions, cardiac, hepatic and renal failure, and typically, pulmonary oedema.
- If rapid death does not occur due to hepato-renal failure (either due to low dosage or energetic clearance of the residual poison), then progressive lung damage may lead to death within the next 2 weeks. This is known as **subacute poisoning** in which the pulmonary involvement begins 24–48 hours after ingestion. The picture resembles that of ARDS, which progresses to pulmonary fibrosis within a few days. Renal failure due to acute tubular necrosis and liver damage showing centrilobular necrosis are common. Death usually occurs secondary to pulmonary fibrosis. The mechanism appears to be that the paraquat reacts with the tissue elements to produce peroxides, including hydrogen peroxide, which is responsible for the damage.

(**The features of diquat ingestion** are similar to that of paraquat. However, an important difference is that unlike paraquat, it does not produce pulmonary toxicity.)

Management

The aims of treatment are to remove paraquat from the gastrointestinal tract, increase its excretion and prevent pulmonary damage.

- Perform a gastric lavage using Bentonite (1 litre of 7% aqueous suspension) or Fuller's earth (1 litre of 15% aqueous suspension).
- Maintain proper fluid and electrolyte balance.
- Haemodialysis and charcoal perfusion are effective for removing paraquat in some cases and, therefore, institute these measures as soon as possible as paraquat rapidly becomes fixed to the tissues.
- Steroids, immunosuppressive agents, fibrinolytic agents have been tried to prevent pulmonary damage. Oxygen inhalation enhances pulmonary damage; therefore, do not administer oxygen in high concentrations.

Postmortem Appearances

There may be ulceration around the lips and mouth from escape of paraquat. The mucosa of the mouth may be reddened or desquamated, and the oesophagus may show worst changes (none of these changes are by any means inevitable, and the upper gastrointestinal tract may be normal). Similarly, the stomach may show erosion and patchy haemorrhages. The liver may show pallor or mottled fatty change. Other organs show no specific changes, apart from the lungs. The kidneys may reveal cortical pallor if there is renal failure.

- If the victim has lived a week or more, then the lungs may reveal typical changes in that they are large and stiff. There may be fibrinous pleurisy and sometimes slight bloody pleural effusions. The main appearances are microscopic and,

unless the history is known, the lung may be mistaken for a diffuse pneumonia. Within the first week, the air spaces become occluded by mononuclear cells forming rounded-up fibroblasts. If patient continues to survive, the alveoli begin to fibrose, with reticulin and collagen being laid down to form a rigid, stiff lung. Samples required at autopsy include the usual blood samples, urine, stomach contents, lung and liver, apart from histopathology of the organs.

Toxicologic Analysis

Blood levels of paraquat of 0.2 mg/L are sufficient to cause lung damage. Survival has been recorded, however, with plasma levels of up to 1.6 mg/L. Paraquat is excreted over a long period and can be detected in urine at autopsy some days after ingestion. (The concentrations found on analysis after autopsy naturally depend upon the dose taken and the time that has elapsed since ingestion.)

CHLOROPHENOXY HERBICIDES

These are used widely to control weeds with broad leaves, which grow among cereals. These herbicides are also available to the public for use in gardens and for total vegetation control. They are formulated as esters or salts, sometimes containing up to 50% of the active ingredient. Though systemic effects can follow absorption through the bronchial tree, most instances of serious poisoning have been due to deliberate ingestions.

Common Compounds

Bromoxynil, DCPP (2,4-DP, Dichlorprop), Dicamba, Ioxynil, MCPA (4-chloro-2-methylphenoxyacetic acid), MCPB.

Fungicides

The commonly used fungicides include various carbamates (which unlike carbamate-insecticides do not inhibit cholinesterase enzymes) and hexachlorobenzene. Carbamate fungicides have a low toxicity. A list of the various carbamate fungicides includes the following:

- Monothiocarbamates: cycloate, pebulate, molinate, diallate.
- Bisdithiocarbamates: thiram.
- Metallobisdithiocarbamates: ziram, nabam, ferbam.
- Ethylene-bisdithiocarbamates: maneb, zineb.

CLINICAL FEATURES

Carbamate Fungicides

These compounds have a low toxicity, which include irritation of the skin and respiratory and gastrointestinal tracts. The reported features after exposure are nausea, diarrhoea, abdominal pain, fever, weakness, coughing, and dyspnoea.

Thiram is structurally similar to disulfiram and can produce a reaction when ethanol is ingested after exposure to thiram. This is characterised by sweating, flushing, headache, weakness, tachycardia and hypotension.

Hexachlorobenzene

Chronic ingestions of seeds contaminated with hexachlorobenzene have produced cutaneous porphyria characterised by hypertrichosis, hyperpigmentation, weight loss, hepatomegaly and painless arthritis.

Rodenticides

A wide variety of chemicals, which defy classification, have been used in the control of rats and mice (Table 43.2). Although they are used to kill rats, mice, moles, voles and squirrels, which resemble humans in their physiology and biochemistry, there are wide differences in degree of hazard to humans. Since rodenticides can be used in baits and placed in inaccessible places, their likelihood of becoming widespread contaminants of the environment is much less than that associated with the use of insecticides and herbicides. The toxicologic problem posed by rodenticides, therefore, is primarily acute accidental or suicidal ingestion. Basically, they fall into following two categories.

- **The single-dose type:** This is fatal for rats through a single feeding (e.g., sodium monofluoroacetate, fluoroacetamide, norbormide, red squill, thallium sulphate, zinc phosphide, and some of the super warfarins).
- **The multiple-dose type:** This requires repetitive feedings to be effective (e.g., warfarin, diphacinone and pindone).

WARFARIN AND ANTICOAGULANT VARIETIES

Warfarin, 3-(alpha-acetonylbenzyl)-4-hydroxycoumarin, is one of the most widely used rodenticides. Its safe usage is based on the fact that it requires repeated dosing for toxicity to develop. Thus placed in baits accessible to rodents; repeated ingestion results in fatalities to rodents with little likelihood that pets or

Table 43.2 Classification of Rodenticides Based on Toxicity

Rodenticides with high toxicity	Rodenticides with moderate toxicity	Rodenticides with low toxicity
Zinc phosphide, strychnine, thallium, sodium monofluoroacetate and fluoroacetamide, yellow phosphorus and arsenic	Barium salts, alpha-naphthyl thiourea, cholecalciferol, alpha-chloralose	Hydroxycoumarins (warfarins), super warfarins, red squill, norbormide

children would be repeatedly exposed. **The mechanism of action** of warfarin is as an anticoagulant. It is an antimetabolite of Vitamin K, and hence it inhibits the synthesis of prothrombin. Multiple doses are usually required to maintain inhibition of synthesis until prothrombin levels are sufficiently depleted to result in haemorrhage throughout the body, which is the cause of death. In addition to its anticoagulant action, direct capillary damage has also been attributed to warfarin. The concern about resistance of rodents to it has led to the introduction of substances such as brodifacoum, bromodiolone, coumatetralyl, difenacoum, chlorophacinone and flocoumafen, which are commonly referred to as **'super warfarins'**. They are characterised by durations of action, which are measured in weeks and months.

Treatment has to be tailored to individual circumstances. It is doubtful if gastric emptying will be of value unless a very large amount has been consumed within an hour or so. Repeated oral administration of adsorbents such as cholestyramine not only prevents absorption but also shortens the plasma half-life of anticoagulant already absorbed. Vitamin K_1 (5–10 mg intravenously for an adult) could be given as a prophylactic measure. Fresh frozen plasma will be necessary for immediate control since Vitamin K_1 will not be effective for about 24 hours. Repeated doses of Vitamin K may be required depending on the amount and specific nature of the compound involved. Monitoring of the prothrombin time will be necessary to determine the need for further treatment.

RED SQUILL

The bulbs of red squill (*Urginen maritima*) have been used for many years as a relatively safe rodenticide. The active principles are glycosides scillaren-A and scillaren-B. These glycosides have cardiotonic actions like the digitalis glycosides. Crude red squill also contains a central-acting emetic, which causes vomiting in animals other than rodents. This emetic action is the main factor that contributes to the safety of the rodenticide to humans. **Symptoms** that are associated with ingestion of large doses of red squill include vomiting and abdominal pain, cardiac irregularity, blurred vision, convulsions and death from ventricular irregularities. The selective rodenticidal usefulness of red squill takes advantage of the physiologic peculiarity of the rat's inability to vomit.

NORBORMIDE

It is another rodenticide that takes advantage of a physiologic peculiarity of the rat for its selective toxicity. This compound acts directly on the smooth muscle of peripheral vessels causing them to constrict irreversibly resulting in widespread ischaemia leading to death.

Sodium fluoroacetate (Compound 1080) and **fluoroacetamide** (Compound 1081): The use of these rodenticides is largely restricted to licensed pest control operators. These are amongst the most potent rodenticides known and are highly toxic to other animals. Fluoroacetate produces its toxic action by inhibiting the citric acid cycle (Kreb's cycle). Estimates of mean lethal dose of fluoroacetate in man range from 2 to 10 mg/kg, and there have been a number of human fatalities. There are apparent species differences in the quality of symptoms that lead to death. Dogs die of convulsions or respiratory paralysis, but in man, monkeys, horses and rabbits, central nervous system actions are usually incidental and the dangerous fatal complication is ventricular fibrillation.

STRYCHNINE SULPHATE

This alkaloid of nux vomica plant is a potent convulsant poison with a lethal dose of a few milligrams per kg body weight for most animals. It lowers the threshold for stimulation of spinal reflexes by blocking inhibitory pathways exerted by Renshaw cells over the motor cells in the spinal cord. As a result, poisoned animals go into tetanic convulsions, in response to rather minimal sensory stimuli. Nux vomica was introduced into Germany in the sixteenth century for use as a rodenticide. Although its use has declined, it is still used in poisoned baits in control of vermin, and accidental poisoning in humans continues to occur.

INORGANIC RODENTICIDES

A number of inorganic compounds are used in rodent-control. Most of these are nonselective in their toxicity and are generally hazardous to humans and domestic animals so that their use has declined in favour of more selective or less hazardous organic compounds. Some of them are described as follows.

Zinc Phosphide

It is a dark grey crystalline powder with an odour similar to that of a rotten fish. It is available as a powder or as pellets. It is a popular rat poison. Inhalation of the dust or accidental or intentional ingestion can give rise to fatal poisoning in humans. It reacts with hydrochloric acid in the stomach releasing highly toxic phosphine gas, a very potent respiratory poison. (Phosphine has been described extensively under aluminium phosphide.)

Toxic Dose

The ingestion of as little as 4–5 gm of zinc phosphide may be fatal.

Fatal Period

Death usually occurs within 24 hours.

Clinical Features

Clinical features of poisoning are similar to those of aluminium phosphide but are usually slower to start because of the

slow release of phosphine. The cause of death is respiratory failure.

Treatment

It is mainly supportive.

Postmortem Appearances

The mucosa of the stomach and intestine usually presents haemorrhagic appearance. Often the mucosa is coated with fine black powder. A garlicky odour emanates on opening the stomach. The blood is cherry red in colour. The lungs show hyperaemia and oedema. Other organs show congestion. (Zinc is an essential trace element for plant and animal life. Its deficiency is recognised as a cause for various diseases in crops, animals and human. Zinc is omnipresent in the environment, in various eatables as also the various body organs. Red blood cells tend to accumulate zinc. Pancreatic fluid contains significant amounts of zinc, as it is active in metabolism of glucosides and proteins and is required for synthesis of insulin. An adult contains about 1.4–2.3 gm of zinc, and plasma level is about 96 mg per 100 ml for healthy adults.)

Thallium

It is a general systemic toxicant. It has high affinity of sulphydryl groups throughout the body. It is capable of breaking down all cells in the body, especially hair follicles and central nervous system. From 1 to 3 weeks after ingestion, hair begins to fall out. Because of its high cumulative toxicity, the use of thallium has been restricted to applications by qualified personnel, with a resultant marked decline in its use as a rodenticide.

White or Yellow Elemental Phosphorus

It has caused poisoning because of the practice of spreading pastes containing this element on bread as a rodenticide bait. A dose of 15 mg of phosphorus can cause severe poisoning in humans and as little as 50 mg may be fatal. Shortly after ingestion, phosphorus produces severe gastrointestinal irritation, and if a sufficient dose is ingested, haemorrhage and cardiovascular failure may prove fatal within 24 hours. The vomitus after phosphorus ingestion is luminescent and has a characteristic garlic odour. If the patient survives the initial phase, secondary systemic poisoning due to liver necrosis may ensue.

Barium Carbonate and Arsenic Trioxide

These have been used as rodenticides, but currently have little application for this purpose. Barium produces severe colic, diarrhoea and haemorrhage. It has a direct action on smooth muscles of the arterioles and cardiac muscle. Death occurs due to cardiac irregularities.

A variety of other compounds have some occasional application in rodent-control. Carbon monoxide, methyl bromide and hydrogen cyanide have been used as fumigants to kill rodents in enclosed spaces. These chemicals are generally toxic to all species. DDT, commonly thought of as an insecticide, is used as a poison for the house mouse and for bats. The principle of this treatment is to treat inaccessible areas where mice travel so that they will pick up a sufficient amount of DDT on their feet and fur.

Pyrethrum and Other Insect Repellents

Pyrethrum is one of the most widely used household insect sprays, which inactivates insects quickly. It is derived from the flowers of *Chrysanthemum cineriae*. The residue of the powder made from its dried flowers contains about 1% of pyrethrum. It contains six active chemicals collectively known as **pyrethrins**. Synthetic derivatives of pyrethrins are known as **pyrethroids**. Most insecticides containing pyrethrins usually also contain piperonyl butoxide, which increases the effectiveness of pyrethrins and, therefore, acts as a synergistic agent. The toxicity of pyrethrum is low, and systemic poisoning is uncommon. A more common problem is allergy to pyrethrum, which can lead to rhinitis, asthma, and occasionally, anaphylaxis.

Insect repellents are not true insecticides. These compounds are intended for human use and, therefore, generally are not toxic. The important insect repellents are N,N-diethyltoluamide (DET) and paradichlorobenzene. **The former** is highly effective insect repellent that is used as a spray in concentrations ranging from 5% to 100%. It is often combined with isopropyl alcohol. **The latter** was initially used as an insect and moth-repellent. It is currently commonly used as a bathroom deodorant in toilet bowls, diaper pails and occasionally, garbage pails. It is much less toxic than naphthalene found in moth balls.

44 CHAPTER
Fumigants

After going through this chapter, the reader will be able to describe: Toxicology, diagnosis and management of aluminium phosphide poisoning | Medicolegal aspects of aluminium phosphide poisoning

Fumigants are used in the control of insects, rodents and soil nematodes. They have in common the property of being in the gaseous form at the time they exert their action and are used because they will penetrate to areas otherwise inaccessible for pesticide application (e.g., grain storage areas, rodent runways). Fumigants may be liquids that readily vapourise, solids that release a gas by chemical reaction or gases contained in cylinders or ampoules. Thus, they provide a potential hazard from the standpoint of inhalation exposure as well as in the case of solids and liquids, accidental ingestion or dermal exposure. Fumigants used in the protection of stored foodstuffs include acrylonitrile, carbon disulphide, carbon tetrachloride, chloropicrin, ethylene dibromide, hydrogen cyanide, methyl bromide and phosphine.

In connection with the pesticides, it is worthy of comment that methyl bromide caused many deaths in the past amongst occupationally exposed persons. Acrylonitrile as a fumigant is limited by its flammability and high cost. Chloropicrin is a strong irritant and sensory irritation gives early warning of its presence. Ethylene dibromide was found to rapidly produce highly malignant gastric squamous cell carcinoma in rats and mice.

Aluminium Phosphide

Aluminium phosphide (ALP) is a solid fumigant pesticide and in use as a pesticide since the 1940s. In India, it is marketed as tablets of Celphos, Alphos, Quickphos, Phostoxin, Phosphotex, etc. ALP has the advantage of being cheap, efficacious, easy to use and freely available in the market. On coming in contact with moisture, ALP liberates phosphine (PH_3), which is toxic to pests, insects and rodents. Human toxicity, which is usually acute, occurs due to the toxic effects of phosphine, which is released in the stomach after ingestion of ALP. Isolated reports of fatal exposure to phosphine gas have been reported when ALP was used as a grain fumigant for bulk shipment of wheat.

PHOSPHINE—PHYSICAL PROPERTIES AND ABSORPTION

ALP is available in the form of dirty white-coloured tablets each weighing 3 gm. Each tablet of Celphos or Quickphos (3.0 gm) contains 56% ALP and 44% ammonium carbonate and has the capacity to liberate 1.0 gm of PH_3. The chemical reaction is accelerated by the presence of HCl in the stomach. In pure form, phosphine (PH_3), hydrogen phosphide, is a colourless, odourless gas. ALP, however, has a foul odour (like garlic or decaying fish) due to the presence of substituted phosphines and diphosphines (see Table 44.1 also). Phosphine in air reacts with hydroxyl radical and is removed by this mechanism with a half-life of 5–24 hours. After fumigation, the nontoxic residues

Table 44.1 Diagnostic Odours

Odour	Substances
Garlicky	Arsenic, organophosphates, phosphorus, selenium, tellurium, thallium
Musty (fishy)	Aluminium phosphide, zinc phosphide
Rotten egg	Disulfiram, hydrogen sulphide, mercaptans, N-acetylcysteine
Bitter almond	Cyanide
Burnt rope	Marijuana (Cannabis)
Coal gas	Carbon monoxide
Moth balls	Camphor, naphthalene
Shoe polish	Nitrobenzene
Vinegar	Acetic acid
Acetone	Ethanol, chloroform, isopropanol

left in the grains are the phosphite and hypophosphite of aluminium. In general, the residues are below the WHO recommended permissible levels of 0.1 mg/kg of PH_3 for raw cereals, which do not lead to any ill effects in human on consumption.

MECHANISM OF TOXICITY

After the ingestion of aluminium phosphide, phosphine is liberated in the stomach, which is absorbed into the circulation. Some of the parent compounds, i.e. aluminium phosphide itself, is also absorbed and is metabolised in the liver with a slow release of phosphine. This may result in the prolongation of symptoms. As reported, phosphine produces widespread organ damage due to cellular hypoxia produced as a result of its binding to cytochrome oxidase. Acute cardiotoxicity is possibly related to the subcellular transmembrane exchange of ions (Na, K, Mg and Ca) due to focal myocardial necrosis produced by phosphine.

TOXIC DOSE

Less than 500 mg of an unexposed pellet of aluminium phosphide is lethal for an adult (usual being 150–500 mg for a 70 kg individual). The inhalation of phosphine at a concentration of 300 ppm is dangerous, while at a level of 400–600 ppm, it is lethal within an hour.

CLINICAL FEATURES

On contact with moisture, the pellets release phosphine gas, the active poison. Because of this, pellets exposed to air rapidly lose potency. The clinical features are more or less the same whether phosphine is inhaled or aluminium phosphide is ingested; however, the initial symptoms pertain to the route of entry.

POISONING DUE TO INHALATION OF PH_3

Occupational threshold value of PH_3 is 0.3 ppm. **Mild inhalational exposure** produces mucous membrane irritation and acute respiratory distress. Other symptoms include dizziness, easy fatigability, tightness in the chest, nausea, vomiting, headache and diarrhoea. **More severe toxicity** produces ataxia, numbness, paraesthesia, tremors, diplopia, jaundice, muscular weakness, incoordination and paralysis. Very severe toxicity is accompanied by development of adult respiratory distress syndrome (ARDS), cardiac arrhythmias, congestive heart failure, pulmonary oedema, convulsions and coma.

POISONING DUE TO INGESTION OF ALP

Mild Ingestional Intoxication

Marked systemic signs and symptoms do not occur except nausea, vomiting, headache and abdominal pain or discomfort.

Moderate and Severe Poisoning

Systemic manifestations are early and progressive and mostly fatal. Symptomatology is as under:

GIT	Nausea, vomiting, diarrhoea, retrosternal pain.
Cardiovascular	Hypotension, shock, and tachycardia can occur. Bradycardia is not uncommon in patients with shock. The mortality rate may be higher in patients with either bradycardia or tachycardia. Myocardial depression leads to congestive heart failure. Various cardiac arrhythmias are of common occurrence. (ALP is highly toxic to the cardiovascular system causing pulmonary oedema, shock and arrhythmias. Acute cardiotoxicity in ALP poisoning is reported to be due to binding of cytochrome oxidase in the mitochondria by phosphine leading to anoxic myocardial damage. The possible explanation of arrhythmias is either a direct toxic effect or hypomagnesaemia brought by focal myocardial damage by the action of PH_3. However, the exact role of magnesium depletion in the pathogenesis of focal myocardial necrosis and clinical cardiotoxicity is obscure. Administration of $MgSO_4$ has been reported to ameliorate arrhythmias.)
Respiratory	Cough, dyspnoea, cyanosis, rales and rhonchi, bilateral basal crepts, respiratory failure.
Hepatobiliary	Jaundice, hepatitis, soft tender hepatomegaly, raised transaminases, etc.
Renal	Oligouric and nonoligouric renal failure (ATN).
CNS	Headache, dizziness, altered mental state, restlessness without alteration in consciousness, convulsions, acute hypoxic encephalopathy, coma. (Severe metabolic acidosis is common in the later stages of poisoning.)

Shock

Causes of intractable shock in ALP poisoning may include the following:

- **Cardiogenic:** Arrhythmias, conduction disturbances and myocardial damage.
- **Peripheral circulatory failure:** Due to widespread small-vessel injury leading to peripheral vasodilation.
- **Vomiting—fluid loss:** Initially, major part of the circulation is diverted from the peripheries towards the internal organs and that is possibly the reason why most patients maintain an adequate urinary output despite very low to unrecordable blood pressure. Once the circulation to internal organs is decreased as evidenced by oliguria, shock gets established and danger increases steeply.

- **Adrenal cortex:** Adrenal function is compromised in very severe cases. Reports suggest that ALP acts as a direct metabolic cellular poison, its toxic effect on adrenal is by liberation of PH_3. The effect is similar to that produced on other organs.

DIAGNOSIS

The diagnosis of ALP poisoning is based on reliable history of ingestion and/or the production of the remaining tablets/empty container by the relatives, garlic/decaying fish like odour, evident clinical manifestations, positive silver nitrate paper test and chemical analysis.

The sensitivity of **'silver nitrate paper test'** is high; even low concentration of PH_3 (even trace amount) darkens the silver nitrate paper. Its specificity is also high except sometimes when silver nitrate produces blackening due to reaction with H_2S in the air. For this reason, another filter paper impregnated with silver nitrate has to be kept outside as control. The procedures adopted include the following:

- **With gastric fluid:** Take 5 ml of gastric aspirate with 15 ml of water in flask and cover its mouth with a filter paper impregnated with $AgNO_3$ (0.1N). On heating this at 50°C for 15–20 minutes, blackening of filter paper develops, which is indicative of presence of PH_3.
- **In breath:** The filter paper impregnated with $AgNO_3$ (0.1N) is used in the form of face mask and the patient is asked to breathe in and out of this filter paper for 15–20 minutes. The blackening of filter paper is indicative of presence of PH_3.

MANAGEMENT

In the absence of specific antidote, treatment is mainly conservative. The main object of treatment is to sustain life till PH_3 is excreted through the lungs and kidneys. The measures adopted may be as follows.

Reduction of Absorption of PH_3

- Gastric lavage with potassium permanganate (1:10,000) is done immediately after admission and is repeated twice or thrice to remove the unabsorbed poison as well as to oxidise it.
- Slurry of activated charcoal (100 gm) is given orally to adsorb PH_3 from the gastrointestinal tract.
- Judicious use of antacids is recommended to reduce symptoms pertaining to the stomach as well as to reduce absorption of PH_3 through the stomach.
- Liquid paraffin may be given to accelerate the excretion of ALP and PH_3 from the gut.

Reduction of Absorbed PH_3

The liberated PH_3 cannot be detoxified as there is no specific antidote. Organ toxicity may be prevented by using membrane stabilisers like magnesium sulphate. $MgSO_4$ may correct the hypomagnesaemia, which may be a precipitating factor for arrhythmias.

It acts by modulating sympathetic, parasympathetic and slow channel kinetics.

Enhancement of Excretion of PH_3

PH_3 is stable and is excreted through the lungs. To enhance its excretion through kidneys, adequate hydration and renal perfusion must be maintained with IV fluids and other resuscitative measures. Diuretics are not useful in presence of shock. In the presence of acute renal failure and with BP maintained by appropriate measures, dialysis has been advocated.

Treatment of Shock

- Fluids need to be administered within the first 3–6 hours, of this 50% must be normal saline. Therapy may be guided using CVP and PCWP measurement.
- Low-dose dopamine infusion is useful to combat shock (higher dose may lead to higher mortality probably due to potent vasoconstrictive effect).
- Intravenous hydrocortisone is also advocated.

Treatment of Arrhythmias

Conventional anti-arrhythmic drugs are not effective. $MgSO_4$ due to its membrane stabilising action has been reported to be effective against arrhythmias, especially during the first 24 hours. $MgSO_4$ also corrects the hypomagnesaemia seen in these cases, which may be the precipitating factor for arrhythmias. The usual dose is 1.0 gm IV stat, 1.0 gm every hour for the next 2 hours, and then 1.0–1.5 gm every 6 hours for 5–7 days in the form of continuous IV infusion. Magnesium levels should be maintained below the toxic levels of 10 meq/L.

Treatment of Metabolic Acidosis

It is seen in all the cases with varying severity. Moderate to severe acidosis needs to be treated with IV soda bicarb till the causative factor (PH_3) is removed, i.e. for 3–4 days. If the patient becomes haemodynamically stable but metabolic acidosis persists, peritoneal or haemodialysis may be useful.

Mortality depends upon dose/amount of poison consumed, freshness of the compound, delay in arrival at the hospital, delay in institution of treatment, duration and severity of shock, vomiting (earlier the vomiting better the prognosis), presence or absence of complications and, above all, nonavailability of antidote.

POSTMORTEM APPEARANCE

There is widespread hypoxic organ damage with congestion and petechiae. Contents of stomach are often haemorrhagic with mucosal shedding, and there is usually an intense garlicky odour. Microscopy may reveal necrotic changes in liver and kidneys. Heart may show features of toxic myocarditis with fibrillar necrosis. Lungs may demonstrate evidence of ARDS with or without pulmonary oedema.

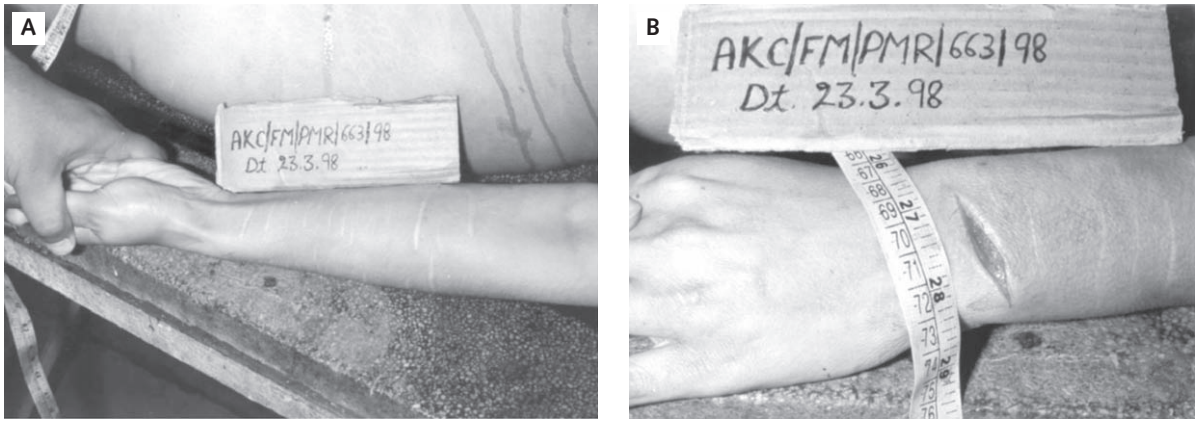


Fig. 44.1 Photograph showing **(A)** multiple old linear scar marks on the front of left forearm running more or less parallel to one another; **(B)** fresh, somewhat obliquely placed incised wound on the left wrist. At the medial end, three small superficial cuts were appreciable with the help of magnifying lens, often called as "hesitation cuts/tentative cuts". The victim was a 27-year-old female, who within 7 months of marriage committed suicide by consuming poison as the Chemical Examiner's Report revealed presence of aluminium phosphide. The police had presented the case under Section 304B IPC (dowry death). The victim was driven to commit suicide because of bringing insufficient dowry, as alleged by the mother of the victim. The old scar marks and fresh incised wound reflect prior and concomitant attempts towards committing suicide. (Contributed by Dr. AK Chanana et al.)

MEDICOLEGAL ASPECTS

The mode of poisoning is usually intentional (suicidal), occasionally accidental and rarely homicidal. In children, accidental poisoning may also occur. The agricultural community irrespective of sex is more at risk. This seems to be related to easy availability, illiteracy and frustration due to nonavailability of avenues for generation of income.

Today, it is the leading cause of suicidal (and sometimes accidental) death in northern states of India (see Figs. 44.1 and 44.2 carrying informative description of related cases). (Normal blood level of aluminium is 17 μg per 100 ml. The human body burden of aluminium is 50–150 mg and is apparently unaffected by normal daily intake levels. The degree of absorption of ingested aluminium and its compounds is minimal. The use of aluminium in cooking utensils and cans usually does not contribute significantly to either total body burden or toxic effects.)

CASE: DETERMINATION TO COMMIT SUICIDE—HOW FAR STRETCHABLE

Occasions, though rare, may be there where an individual in order to ensure absolute death, uses two totally different methods/means for killing oneself. Thus, one may find an individual dead of a gunshot wound with potentially lethal levels of drugs (apparently, the drugs do not work fast enough and the individual decides to resort to some other means). Reportedly, a lady shot herself twice in the chest. However, only one bullet entered the chest cavity piercing the left lung. The internal haemorrhage thus produced, apparently, was not quick enough as the woman then cut her wrists with a broken bottle. Another individual placed a noose around his neck, tied one end to a support, and then shot himself in the head. As he collapsed, he suspended himself by the neck (preconceiving some possibility

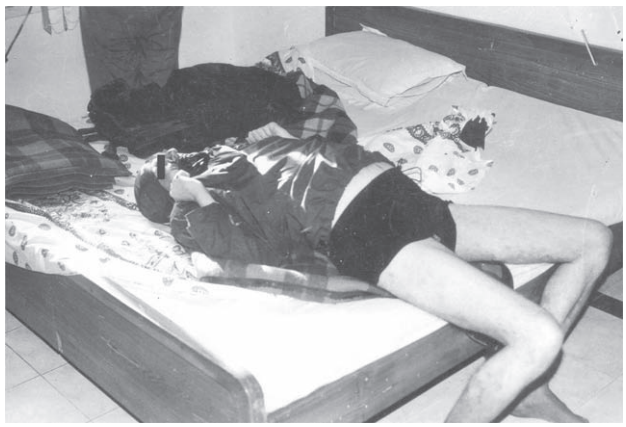


Fig. 44.2 Photograph of deceased showing sleeping bag wrapped around head and neck (face uncovered for recognition).

of survival for any length of time, the determined victim had arranged an additional design for back-up, conveying volumes of his determination).

The case being cited here is of similar nature wherein, as reported in the News Papers, a foreign national was found dead in the room of some '*Bhawan*'. He was noticed lying on the bed in a strange manner by the cleaning worker who had come to clean windowpanes of the room. Experiencing the windows and doors to be bolted from inside, he knocked hard at the windowpanes but received no response. Thereafter, some high official of the *Bhawan* was called to arrange for breaking the front door. The investigating agency was informed. It was found that the face of the victim was tightly fastened in a sleeping bag, apparently in an attempt to cause death through suffocation (see Fig. 44.2). Another theory being probed by the investigating agency was that the deceased might have consumed some intoxicant and feeling that it was not working fast

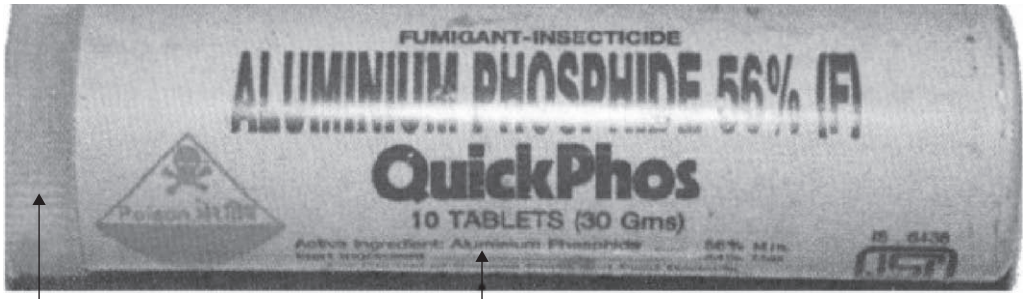


Fig. 44.3 Characteristics of the container in which the tablets of aluminium phosphide are marketed. Generic Name: Aluminium phosphide, Trade Name: Quickphos/Celphos/ Alphos/Synfume/Phosfume/Phosphotek/Phostoxin.

enough, resorted to an additional design to ensure death (this was later supplemented by the demonstration of organophosphorus compounds in the Chemical Examiner's Report).

As per reports, paraphernalia found at the scene went a long way in ruling out any foul play, viz.; finding of a bag containing substantial amount of money including some foreign currency, a passport, literature of Saibaba, a set of credit cards, some travel documents, a driving license, shoes plus some clothes of the deceased, and a laptop computer (from where some vital information could be deciphered by the investigating agency through the services of some IT experts), etc.

Fumigant literally means 'which exudes fumes/vapours/gas (especially when harmful or unpleasant). Aluminium phosphide (ALP) is a grain preservative used widely by the northern wheat producing States. It is available in the form of greenish-grey tablets (3 gm each) with garlic-like odour. Active ingredient is aluminium phosphide (56%, m/m) and inert ingredients include urea and ammonium carbonate (44%, m/m). Upon exposure to air, the tablets are disintegrated releasing phosphine (PH_3), carbon dioxide and ammonia gases. That is why the tablets are encased in an aluminium casing so as to avoid exposure to air (Fig 44.3) [air is a mixture of nitrogen (78.1%), oxygen (20.93%), carbon dioxide (0.03%) plus *water vapours* and suspended matter such as dust, bacteria, spores and vegetable debris].

Illustration: In 1980, an alarming accident was reported wherein the grain leaving Canada for European destination was fumigated by adding a certain number of sachets of ALP per ton of grain in the hold of the ship so as to provide continual protection during trans-shipment of the grain. The ship ran into a bad storm off Nova Scotia and began to leak; hastening the breakdown of ALP to form PH_3. The toxicant penetrated the quarters of the crew and 29 out of 31 members became seriously affected. One died before reaching the hospital in Boston. The highest concentrations of PH_3 (20–30 ppm) were measurable in a void space on the main deck near the air intake for the ship's ventilation system. In some of the living quarters, PH_3 levels of 0.5 ppm were detected. Although this could be considered a bizarre situation, it goes a long way in illustrating an apparent problem involving the use of such an agent in an atmosphere of excessive moisture.

45 CHAPTER

Asphyxiants

After going through this chapter, the reader will be able to describe: Harmful action of hydrocyanic acid and its salts | Diagnosis and management of hydrocyanic acid poisoning with medicolegal aspects | Major air pollutants, their sources and harmful effects

The general expression for irrespirable or toxic gases is **asphyxiants**. These may be classified as follows:

- **Simple asphyxiants:** These gases displace oxygen from the ambient air and reduce the partial pressure of available oxygen. Examples include carbon dioxide, nitrogen, aliphatic hydrocarbon gases like ethane, methane, butane and propane, and noble gases like argon, helium, xenon, etc.
- **Respiratory irritants:** These gases damage the respiratory tract by destroying the integrity of the mucosal barrier. Examples include ammonia, chlorine, hydrogen sulphide, methyl isocyanate, phosgene, sulphur dioxide and formaldehyde.
- **Systemic asphyxiants:** These gases produce significant systemic toxicity by specialised mechanisms. Examples include hydrogen cyanide gas (HCN), carbon monoxide and smoke, etc.

Carbon monoxide and carbon dioxide have already been described in detail in the Chapter "Asphyxial Deaths". Therefore, hydrocyanic acid and its salts will be discussed here.

Hydrocyanic Acid and Its Salts

Hydrocyanic acid or cyanogen is very potent, extremely lethal and most rapidly fatal. Poisoning with hydrocyanic acid is almost always fatal because of the low fatal dose and the rapidity with which it acts.

Availability and usage of hydrocyanic acid (HCN), sodium cyanide (NaCN) and potassium cyanide (KCN): Hydrocyanic acid is also known as cyanogen. An aqueous solution of hydrocyanic acid is known as dilute hydrocyanic acid or **prussic acid** (an acid met within commerce and commonly used in the veterinary practice, called the **Scheele acid,** contains approximately 4% of the pure acid). HCN at ordinary temperature and pressure is a gas having penetrating odour resembling that of bitter almonds. All persons cannot smell the gas, and the ability to detect the same is a sex-linked recessive trait. However, in cold temperature and under pressure, it can be kept in liquid form. HCN as a gas was once used in the disinfestation of houses but proved too dangerous. It is still used to fumigate ships, buildings and citrus trees.

Hydrocyanic acid is widely distributed in nature. It occurs in the leaves of the cherry-laurel, in bitter almonds, in the kernels of the common cherry, plum, apricot, peach and other stone fruits, the ordinary bamboo shoots and in certain oilseeds and beans (see Table 45.1). These plants contain a cyanogenic glycoside, known as amygdalin. **Amygdalin** is a chemical combination

Table 45.1 Cyanogenic Plants

Plant	Toxic part	Cyanogenic glycoside
Prunus species: Cherry laurel, bitter almond, chokeberry, peach, apricot, plum and wild black cherry	Leaf, bark, seed	Prunasin or amygdalin
Sorghum species: Sorghum, sudan grass, and arrow grass	Grain, shoot	Dhurrin
Apple, pear, crab apple	Seed	Amygdalin
Cassava, lima beans	Bean, root	Linamarin
Miscellaneous: Christmas berry, jet berry bush, velvet grass, bamboo, cycad nut	Bead, leaf, shoot sprout	Linamarin

of glucose, benzaldehyde and cyanide from which the latter can be released by the action of β-glucosidase or emulsin. Although these enzymes are not found in mammalian tissues, the human intestinal microflora appears to possess these or similar enzymes capable of effecting cyanide-release resulting in human poisoning. For this reason, amygdalin may be as much as 40 times more toxic by the oral route as compared with intravenous injection. Amygdalin is the major ingredient of laetrile, and this alleged anticancer drug has also been responsible for human cyanide poisoning.

Hydrocyanic acid **forms cyanides with metals**. Of these, potassium or sodium cyanide, mercuric cyanide and silver cyanide are used in photography, electroplating, hardening of steel, silver and gold processing and dyeing. These are soluble in water, alkaline in reaction and highly poisonous. Calcium cyanide is cheaper and is used in mining industry. Magnesium cyanide (cyamg) and cyanogen chloride are used as insecticides.

Cyanides are produced in many major fires where the burning of wool, silk, nylon, polyurethane, polyacrylonitrile releases hydrogen cyanide. Extrapolating data from combustion studies, Bertol et al. (1983) calculated that a toxic concentration of HCN could be developed in an average-sized room by the burning of 2 kg polyacrylonitrile. There is a wealth of information suggesting that humans exposed to combustion products have absorbed cyanide. The first detailed description of cyanide in the blood of fire victims was given by Wetherell (1966) who found cyanide in the blood of 39 of 53 individuals dying in fires. Exposure of HCN vapour released in a fire can lead to muscle weakness, difficulty in coordination, physical incapacitation and partial or complete loss of consciousness. This clearly will impede escape from the site of a fire.

MECHANISM OF ACTION

Cyanide toxicity is produced by **cytrochrome oxidase inhibition**, which forms as cytochrome-oxidase-cyanide complex. This results in the paralysis of the electron transport system leading to cellular hypoxia, as oxygen cannot be utilised by the tissues. As a result of the blockade of the cytochrome oxidase system, pyruvate is converted into lactate leading to metabolic acidosis. Thus, poisoning causes asphyxia at the tissue/cell level, though there may be ample supply of oxygen and the blood also may be saturated with oxygen.

ABSORPTION, FATE AND EXCRETION

Liquid HCN can be absorbed through all mucous membranes and skin. **The gaseous form** is readily absorbed through the respiratory tract. Absorption of HCN is quicker than its salts. **Salts** (cyanides) may vary in their rate of absorption. The rapidity with which the salts, upon ingestion, cause death will depend upon the amount of hydrochloric acid present in the stomach and the subsequent liberation of hydrogen cyanide on reaction with the acid of the stomach. It has, therefore, been suggested that those who are achlorhydric cannot be poisoned by cyanides. This is doubtful because water in the gastric juice and the tissues of the stomach can hydrolyse cyanide and liberate hydrocyanic acid. Apart from this, food in the stomach delays the conversion of the salts to HCN and further delays the process of absorption. All these account for higher fatal dose and longer fatal period of KCN and NaCN.

After absorption, the greater part is converted by a mitochondrial enzyme, rhodanese, into thiocyanate, which is nontoxic. A small amount is eliminated through the expired air, which is appreciable in the form of bitter almond like smell of the expired air. The main route of excretion is urine. (Cyanide is 60% protein-bound concentrated in red cells, and has a volume of distribution of 1.5 L/kg body weight.)

FATAL DOSE

Fatal dose of hydrocyanic acid can be given as follows:

- HCN gas: 100–200 ppm in air. HCN liquid: 50–60 mg.
- KCN, NaCN: 150–300 mg. (The fatal dose of cyanides being small, prominent Nazis allowed it to be used as hidden suicide-pills/capsules at the end of the last war. However, much depends upon the purity of cyanides, as they tend to decompose in storage. Harmless carbonate may be formed by the action of atmospheric carbon dioxide and moisture on the potassium cyanide, if it is kept for a sufficiently long time. An old sample of KCN may also be converted by hydrolysis into potassium formate, a comparatively harmless salt.)

Seventeen to thirty drops of oil of bitter almonds have produced fatal results, but in some cases, recovery has taken place after doses of 20–30 ml. Sixty to eighty bitter almonds are sufficient to destroy the life of an adult. Cherry-laurel water in quantities of 45–60 ml has caused death.

The concentration of 0.2–0.3 mg of the gas per litre of air is regarded as sufficient to kill humans almost immediately, while the concentration of 0.13 mg/L of air and an exposure of over an hour are sufficient to prove fatal to human beings.

FATAL PERIOD

Cyanide directly stimulates the chemoreceptors of the carotid and aortic bodies with a resultant hyperpnoea. Cardiac irregularities are often noted, but the heart invariably outlasts the respirations. Death is due to respiratory arrest of central origin. It can occur within seconds or minutes of the inhalation of high concentrations of hydrogen cyanide gas. Because of slower absorption, death may be more delayed after the ingestion of cyanide salts, but the critical events still occur within the first hour.

SIGNS AND SYMPTOMS

When **inhaled as a gas**, its action occurs within seconds. Massive doses may produce sudden loss of consciousness and prompt death from respiratory arrest. **After ingestion,** symptoms

appear within minutes, during which the victim may perform certain voluntary acts, such as corking, or throwing away the bottle or walking a little distance. The major organs/systems involved are the GIT, CNS, respiratory and cardiovascular systems.

Gastrointestinal Tract

The features of GIT involvement occur after the ingestion of cyanides and include a burning taste, throat numbness, salivation, frothing at the mouth, nausea, vomiting, and substernal and epigastric pain.

Central Nervous System

The involvement of CNS leads to dizziness, headache, sweating, anxiety, confusion, drowsiness, syncope, opisthotonus, seizures, coma and death.

Respiratory System

Initially, tachypnoea and dyspnoea develop due to the stimulation of respiratory centre and carotid chemoreceptors caused by local hypoxia. Bradypnoea, hypopnoea and irregular respiration (characteristically, a short inspiration and prolonged expiratory phase), pulmonary oedema, cyanosis and respiratory arrest in the later stage. A bitter-almond like odour may be detected in the breath.

Cardiovascular System

Initially, hypertension along with reflex bradycardia. This is followed by hypotension, tachycardia, arrhythmias, etc. The venous oxygen tension approaches that of arterial oxygen tension and, therefore, the venous blood in the initial stages is bright red. This may be easily demonstrable by examining the fundus for retinal arteries and veins.

Lactic Acidosis

Lactic acidosis develops in the later stages as cyanide inhibits mitochondrial cytochrome oxidase, thereby blocking electron transport and preventing oxygen utilisation and oxidative metabolism. Lactic acidosis occurs as a consequence of anaerobic metabolism.

DIAGNOSIS

The diagnosis is based upon the history and physical examination. Although the measurement of whole-blood cyanide level will confirm the diagnosis, cyanide assays are not routinely available and the decision for the treatment needs be based on clinical finding. Lactate levels have been used as surrogate marker. (A blood cyanide level of $>0.2\,\mu g/ml$ is considered toxic. Lethal cases have usually had levels above $1\,\mu g/ml$.)

TREATMENT

The treatment regimen consists of stabilisation, decontamination and antidote therapy.

Stabilisation

It includes assisted ventilation, oxygen administration, cardiac monitoring, treatment of metabolic acidosis, vasopressors for hypotension. (Oxygen at 1 atmosphere is advocated along with nitrite and thiosulphate. The major effect of oxygen appears to be on the rhodanese reaction, although the enzyme itself is not known to be sensitive to oxygen.)

Decontamination

In case of **cutaneous exposure**, remove clothing and wash with soap and water. In case of **ingestion**, stomach wash with 5% sodium thiosulphate solution. Instill activated charcoal at the beginning and end of the stomach wash (the lavage should be done after stabilising the patient and instituting antidotal therapy).

Antidotal Therapy

It comprises three steps: **The first step** consists of administration of amyl nitrite as a first-aid measure. (One ampoule of 0.2 ml is broken between two pads of gauze and placed over the airway. It is inhaled for 30 seconds of each minute and using a fresh ampoule every 3 minutes.) **The second step** consists of giving sodium nitrite (as a 3% solution at a dose of 10–15 ml/300–450 mg slow infusion intravenously over 5–10 minutes). These nitrites induce the formation of methaemoglobinaemia. The affinity of methaemoglobin for cyanide exceeds that of cytochrome a_3, leading to dissociation of the cyanide–cytochrome complex. **The third step** involves the administration of sodium thiosulphate (as a 25% solution at a dose of 50 ml/12.5 gm intravenously, 3–5 ml per minute). Thiosulphate serves as a substrate for the enzyme rhodanese, which mediates the conversion of cyanide to the much less toxic, thiocyanate, which is excreted in the urine. Although the enzyme rhodanese is widely distributed in the body, liver rhodanese probably plays the major role in cyanide detoxification. It is an endogenous mechanism for cyanide metabolism, but the administration of exogenous sulphur greatly accelerates the rate of reaction (nitrite-thiosulphate therapy can be repeated after an hour, if need arises).

Hydroxycobalamin, a Vitamin B_{12} precursor that also binds cyanide ion, is an alternative antidote that is not yet widely available.

POSTMORTEM APPEARANCES

The skin presents a livid or violet appearance. Postmortem staining is often bright red due to formation of cyan-methaemoglobin, and also due to the fact that the tissues cannot take up oxygen of the blood, leaving it bright red even in the veins (cyanide being lethal in small quantities and, therefore, the total amount of the poison in the body may not be sufficient for generalised discolouration). The fingers may be clenched, fingernails blue and there is usually froth at the mouth and nostrils. The eyes may be bright, glistening and prominent with dilated pupils.

Jaws are usually firmly closed. Rigor mortis sets in early and lasts longer.

Internally

The odour of hydrocyanic acid may be noticed on opening the body, but it is liable to fade quickly. The cranial cavity should be opened first, as the odour is usually well-marked in the brain tissue. Blood-stained froth may be found in the trachea and bronchi. Pulmonary oedema is evident. The mucosa of the stomach and intestines is often congested.

In case of cyanides, lips and mouth may be corroded and the mucous membranes of the stomach and duodenum may be bright red to brown in colour due to the effect of potassium carbonate (present as an impurity in the potassium cyanide) and the probable formation of cyanhaemochromogen from the effects of the cyanide on haemoglobin in the presence of an alkali.

The brain, lungs and blood, in addition to other viscera, should be preserved for chemical analysis. (Lung should be sent intact sealed in nylon bag. Spleen is said to be the best specimen for cyanide analysis since it generally has the highest concentration of the poison owing to enough presence of RBCs.)

TOXICOLOGICAL ANALYSIS

The usual blood, stomach contents, urine and any vomit should be submitted to the laboratory, taking particular care that the samples present no hazard to those packing, transporting or unpacking them.

It is important to get the samples to the laboratory as soon as possible to avoid the spurious formation of cyanide in stored blood samples. If there is to be a delay, refrigeration is essential. In contrast, some positive samples may actually decrease on storage, as described by Curry. Up to 70% of the cyanide content may be lost after some weeks, from reaction with tissue components and conversion to thiocyanate. The amount demonstrable on analysis naturally depends on the amount taken and the time between administration and death. (As already stated, the blood concentration of persons dead of cyanide poisoning are usually in excess of 1 mg%. HCN is an extremely volatile substance and, therefore, blood and viscera should be preserved by adding an alkali and stored in well-stoppered bottles for analysis.)

If it is not demonstrable in the analysis, death may nevertheless have been due to poisoning by hydrocyanic acid. A case is recorded of death from hydrocyanic poisoning, in which analysis 26 hours after death failed to detect the poison. On the other hand, it has been detected by analysis 17, 21 and even 23 days after death, and may be detected even if no odour of the acid is perceptible. Furthermore, even the positive results of the analysis may be confronted with the objection that the chemical has been yielded by the apple pips, cherry kernels or the like. Therefore, the contents of the stomach, vomited matter, etc. should be searched for such bodies and, if found, should be separated before proceeding for the analysis. Another obvious objection may be that the chemical has been yielded by the decomposition of sulphocyanide of potassium present in the saliva. This, however, only accounts for a minute trace of hydrocyanic acid but not for more (perhaps, never more than one-tenth the minimum quantity found in the viscera, in a cyanide-poisoning case).

MEDICOLEGAL ASPECTS

Hydrocyanic acid and its various salts are often used for suicidal purposes, as their swift and sure action is generally known. Hydrocyanic acid and cyanides, being very powerful poisons, are used principally as suicidal agents, the cyanides being about three times as common as the acid owing to their more common usage in the garden and in photography. Nazi Germans used to carry a capsule of HCN to crush in the mouth to commit suicide when required. Accidental poisoning occasionally occurs especially in the docks where it is used for disinfecting purposes. Fatal results have followed from fumigation of trees and fruits owing to the vast quantities and concentrations used. The gas is capable of being absorbed through the skin especially when moist with perspiration. Chemists and laboratory assistants are sometimes overcome by the sudden evolution of hydrocyanic acid following the pouring away of cyanide solutions into sinks already containing strong acid residue. The fumes may be inhaled accidentally by those working with the gas. Eating bitter almonds has produced toxic effects. Laurel-water has also caused poisoning. Hydrocyanic acid is also used as a cattle poison. Cattle poisoning has been known to have occurred from eating *kadvi juar* and also linseed plant due to the natural development of a cyanogenetic glycoside, which may liberate hydrocyanic acid.

Its use as an agent for homicidal purposes is rare except for some mass homicides, which still occur such as Jonesville tragedy in Guyana or the use of cyanide as a weapon of war against the civilians in the Middle East. Two features go against the concept of cyanide being an ideal homicidal poison, viz., its possible detection by smell and the suspicion likely to be aroused by the dramatic nature of death.

The issue of interval between the ingestion of the poison and insensibility may assume importance in rare cases. In large doses, insensibility may come within seconds and rarely be delayed beyond about a couple of minutes. Still, however, considerable power of volition and locomotion may remain and various acts may be performed between the swallowing of the poison and supervention of insensibility. Hence, the finding of the bottle (out of which the poison has been consumed) on a shelf or table or some place out of reach of the body may be consistent with a supposition of suicide.

It has also been used for **Judicial Execution** in some countries. The condemned person is strapped in a chair and several cyanide 'eggs' are dropped into a pan of strong acid, which produces large quantities of the poisonous gas immediately. Unconsciousness takes place very rapidly, although the heart continues to beat for some minutes.

Table 45.2 Combustion Products of Various Materials

Material burnt	Combustion products
Wood, cotton, paper	Carbon monoxide, acrolein, acetaldehyde, formaldehyde, methane
Petroleum products	Carbon monoxide, acrolein, acetic acid, formic acid
PVC (polyvinylchloride)	Carbon monoxide, phosgene, chlorine
Plastics	Cyanide, aldehydes, ammonia, nitrogen oxides, phosgene, chlorine
Rubber	Hydrogen sulphide, sulphur dioxide
Wool	Carbon monoxide, hydrogen chloride, phosgene, cyanide, chlorine
Silk	Sulfur dioxide, hydrogen sulfide, cyanide, ammonia
Nylon	Ammonia, cyanide
Nitrocellulose	Nitrogen oxides, acetic acid, formic acid
Acrylic material	Acrolein, hydrogen chloride

Smoke

Smoke consists of a suspension of small particles in hot air and gases. It has a particulate phase and a gaseous phase. **The particles consist of** carbon, and they are coated with combustion products such as organic acids and aldehydes. The majority of particles are filtered out by the nasal, oral and pharyngeal mucosae, but the acids and aldehydes coating these particles cause symptoms of local irritation, e.g. lacrimation, burning of the throat, and nausea and vomiting when swallowed. Inhalation of some of the small particles into the airways contributes to the bronchospasm that can occur following inhalation of smoke. **The gaseous phase** of smoke has an extremely variable composition, but carbon dioxide and carbon monoxide are always present and constitute the bulk of this fraction. The adverse effects of smoke not only result from its chemical composition but also from the fact that the particulate and gaseous fractions are space occupying, and they can rapidly fill an enclosed space at the expense of air. Composition of smoke depending upon the material burnt is furnished in Table 45.2.

CLINICAL FEATURES

Adverse respiratory effects resulting from smoke inhalation range from mild irritation of the upper airways to severe tracheobronchitis, bronchospasms, pulmonary oedema and bronchopneumonia, which may result in pulmonary insufficiency and death. Laryngitis and laryngeal oedema can also occur, and they may progress to laryngeal obstruction. Increased cough and sputum production coupled with aggravation of chronic pulmonary disease and asthma may be some other features.

DIAGNOSIS

Arterial blood gas analysis is an important investigation. Xenon ventilation studies can detect small airway and alveolar injury before radiographic changes become apparent. Carbon monoxide and methaemoglobin concentrations need to be determined.

TREATMENT

After removal from further exposure to smoke, supplemental humidified oxygen needs to be administered, bronchodilators, as necessary. Management of carbon monoxide or cyanide toxicity, if the need arises. Methaemoglobinaemia can be treated with methylene blue. (It should be carried out cautiously, as it can itself induce methaemoglobinaemia in high doses.)

MEDICOLEGAL CONSIDERATIONS

'Smoke' needs to be differentiated from 'fumes' that refer to a suspension of fine solid particles in a gas resulting from condensation. When smoke mixes with fog, the resultant air contaminant is called as 'smog'.

The most common culprits for air pollution include carbon monoxide, carbon dioxide, sulphur dioxide, nitrogen oxides, hydrogen sulphide and heavy metals such as lead and arsenic. Photochemical oxidants include ozone, hydrocarbons and particulate matter (Table 45.3).

Table 45.3 Major Air Pollutants, their Sources and Adverse Effects

Pollutant	Source	Health impact
Carbon monoxide (CO)	Produced during incomplete combustion of carbon containing materials such as wood, fuel in automobiles, industrial processes and incinerators	The fluctuations in the ambient concentrations are slowly reflected in the carboxy-Hb levels in humans, as it takes 4–12 hours for approximate equilibrium between air levels and blood levels to occur
Carbon dioxide (CO_2)	Produced by burning of coal, oil and natural gases	Though being natural constituent of air, its global concentration is rising above the natural level by an amount that could increase global temperature enough to affect the climate
Lead	Combustion of alkyl lead additives in motor fuels accounts for the major part of all lead emissions	Children are at greater risk because of incomplete development of blood–brain barrier. Further, neurological and haematological effects occur at lower threshold in children than in adults
Arsenic	It is the 20th most abundant element in the earth's crust. Industrial/occupational exposure or consumption of contaminated water or food	A devastating health crisis began to unfold in West Bengal in the early 1980s due to exposure to arsenic laced well water. Skin, GIT and nervous manifestations are common
Ozone	Emissions from vehicles and industries	Burning, itchy, watery eyes and lowered resistance to respiratory diseases
Hydrocarbons	Automobile exhaust, incineration, combustion of coal, wood and use of petroleum	Exert their pollutant action by taking part in the chemical reactions that cause photochemical smog
Suspended particulate matter	Tend to divide into two main groups: (*i*) coarse particles larger than 2.5 mm in aerodynamic diameter and (*ii*) fine particles smaller than the former. The smaller particles contain secondarily formed aerosols, combustion particles and recondensed organic and metal vapours	Can get lodged in lungs and cause damage
Sulphur dioxide (SO_2)	It is one of the several forms in which the sulphur exists in the air. Results from combustion of sulphur containing fossil fuel and other industrial processes	SO_2 and NOx are the primary pollutants causing **acid rain** (a broad term used to describe several ways that acids fall out of the atmosphere). Sulphate and nitrate particles can penetrate human lungs and damage the same. Scientific studies have identified a relationship between elevated levels of fine particles and increased illness and premature death from heart and lung disorders, such as asthma and bronchitis
Oxides of nitrogen (NOx)	Automobile exhaust, gas stoves and heaters, wood-burning stoves, kerosene space heaters etc.	

Usual sources of air pollution include automobile exhaust, industries, domestic combustion of coal, kerosene, wood, etc. Tobacco smoking, burning or incineration of refuse and pesticide spraying are some of the other sources.

Historically speaking, production of ill-health (especially in the form of respiratory diseases) can be traced back to seventeenth century. During that period, widespread use of coal as heating fuel in London led to the development of thick fogs over the city. In 1952, the incidence of settling of smoke from coal gas along with industrial smoke over the city as a pall of smog tripled the daily mortality rate from respiratory diseases, and over 4000 people died. In 1955, more than 240 deaths per day were reported in Los Angeles (USA) due to increased pollutant concentration as a result of the temperature inversion.

46 CHAPTER

Poisoning in Conflict: Chemical and Biological Warfare Agents

After going through this chapter, the reader will be able to describe: Chemicals used in warfare | Toxicology of compounds causing pulmonary oedema and other complications | Biological agents used in warfare

Chemical Warfare Agents

Throughout history humans have sought more effective means of killing and disabling their fellow men. Stones, clubs, spears, bows and arrows, gunpowder, high-explosives, machine guns, war planes, rockets and nuclear weapons, etc. comprise an apparently unending catalogue of increasing military sophistication, designed for the destruction of one's enemies while exposing one's forces to decreasing risk. Accompanying this development of military hardware, there has been a much less marked development of chemical means of attack. Some chemicals have been used as a means of killing and others as a means of incapacitating. The First Hague Convention (1899) led to a wide-ranging prohibition of the use of chemicals in war. Despite this prohibition, chemical warfare was on a large scale during World War I, some 113,000 tons of chemical weapons were used. It has been reported that on 9 March 1918, German Forces fired some 200,000 mustard gas shells.

The term 'gas', as used in chemical warfare, denotes a chemical compound, whether gaseous, liquid or solid, which is employed to produce poisonous or irritant effects on the enemy forces or even the civil population. The term 'war gases', therefore, may be meant to designate not the physical state but rather its applicability as an agent fitted to carry damage mostly in times of war, but if need arises, on other occasions also as, for example, to disperse unruly mobs. Hence, these agents may include the following:

- **Agents liable to be met in warfare**
  - Vesicant agents (sulphur mustard, Lewisite, etc.)
  - Lung damaging agents (phosgene and chlorine)
  - Nerve agents
  - Miscellaneous agents: cyanide, arsine, herbicides, MIC, etc.
- **Agents liable to be met in riot control/or war**
  - Sensory irritants (lachrymators/tear gases)
  - Vomiting agents (sternutators)

VESICANTS OR BLISTERING GASES

These gases chiefly include mustard gas (dichlordiethyl sulphide) and Lewisite gas (2-chlorovinyl-dichlor-arsine). **Mustard gas is also known as** 'Yellow Cross' or 'Yperite' (it was used first by the Germans on 12 July 1917 at Ypres producing 14,276 British casualties during the first 3 months and some 168,000 casualties by the end of the war). Mustard gas is a heavy, dark coloured, oily liquid, having a mustard-like or garlic odour, and giving off vapour at the ordinary temperature of the air. It is almost insoluble in water and evaporates slowly, so that it persists for a long time after it is discharged. It dissolves freely in paraffin, petrol, ether, benzene, rubber, alcohol, acetone and carbon bisulphide. The terrain covered with mustard gas usually remains poisonous to human occupation for 2–4 days. Exposed drinking water may be contaminated by dangerous oily surface film of mustard.

Sulphur mustard gas is a strong alkylating agent and extremely dangerous both in the liquid and in the vaporous state. It is insidious in its onset, and produces toxic symptoms, usually after the lapse of two to four hours. It causes irritation of the eyes with profuse lachrymation and nasal secretion, laryngitis involving the trachea and bronchi. It enters the skin through the clothes and produces intense itching, redness, vesication, ulceration etc. It attacks chiefly the axillae, groins, perineum and scrotum that are moist and oily. Owing to secondary infection, these ulcers are often difficult to heal. The skin of the exposed parts, such as the face, neck and hands is also affected. In rare cases, the stomach may be inflamed as

a result of swallowing the chemical, and this results in nausea, vomiting and gastric pain. (**Nitrogen mustard**, a therapeutically more useful compound, has been investigated in detail. It is converted in the body to an active ethylenimonium intermediate, which binds to a range of molecules including proteins, enzymes and nucleic acids. Sulphur mustard is converted in part to a similar sulphonium ion. The binding of these compounds to guanine residues causes severe disruption of DNA structure and function. Damage to DNA will exercise effects upon dividing tissues, and bone marrow depression, hair loss and GIT effects are well-recognised.)

Diagnosis

Estimation of thiodiglycol in the blood. Standard haematological tests and regular chest X-rays are helpful in diagnosing the condition.

Treatment

First aid measures carry importance, since there is no specific therapy (a respirator and adequate protective clothing should be worn when dealing with contaminated individuals):

- Removal of the patient from the source of the sulphur mustard vapour.
- Clothing should be removed and affected areas washed with soap and water. For areas of erythema and minor blistering, calamine lotions are suggested.
- Liquid contamination of eyes should be rinsed out immediately using 0.9% saline solution, if available, or water. Topical prednisolone drops (1%), ascorbate (potassium ascorbate 10%), and citrate (sodium citrate 10%) drops are suggested.
- Codeine linctus may ease severe coughing.
- Bone marrow depression resulting from sulphur mustard poisoning has been considered irreversible but 'granulocyte colony stimulating factor' may be helpful.
- Haemodialysis and haemoperfusion have both been suggested.

Lewisite was developed as a chemical warfare agent in 1918 by Lee Lewis. In the early 1920s, Lewisite was considered as a compound likely to produce more severe effects than mustard gas and was nicknamed as 'The Dew of Death'. It has been used as a mixture with sulphur mustard, the Lewisite lowering the freezing point of the mustard and making it more effective under cold conditions. Fears that Lewisite might be used during World War II prompted the scientists and led to the development of the chelating agent dimercaprol or British Anti-Lewisite (BAL).

Lewisite, a lipid-soluble arsenic, is a heavy, oily dark liquid, having an odour of geraniums. It is neutralised by water and alkalies. It dissolves in oils, benzene and ordinary organic solvents. It is both a vesicant and an asphyxiant and is more rapid in action than mustard gas. It produces more discomfort on inhalation and more irritation when comes into contact with the skin. It is absorbed rapidly through the skin and mucous membranes. Its distribution in the body is assumed to follow that of other arsenical compounds and heavy deposits in the liver, kidney, wall of the gut, spleen and lung would be expected. Being liquid, it can be disseminated by shells, bombs, rockets and aircraft spray. The effect of Lewisite is thought to be due to the combining of the arsenical part with lipoic acid to form a cyclic compound—lipoic acid is an essential part of the pyruvate dehydrogenase system acting as a co-enzyme in the formation of acetyl CoA from pyruvate. Its use as a chemical weapon is limited because of rapid hydrolysis and difficulties in maintaining effective vapour concentrations.

It needs to be remembered that a vesicle caused by Lewisite is clearly defined, covers the whole erythematous area, and is filled with a cloudy fluid containing arsenic and leucocytes. While a blister produced by mustard gas is surrounded by a zone of erythema and contains a clear yellow serum, it does not contain mustard. Blood and urine need to be analysed for arsenic.

Treatment of lewisite include first aid measures as described for mustard gas exposure. Dimercaprol has shown to be effective against the effects of Lewisite and also in reversing the enzyme inhibition produced by the gas. It is applied as drops (5–10% in vegetable oil) into the conjunctival sacs of the affected eyes. Skin lesions should be treated with dimercaprol ointment. Dimercaprol can also be given intramuscularly to antagonise the systemic effects of the absorbed arsenical. DMSA (succimer), DMPS (unithiol) and DMPA have been developed more recently and should be considered as possible alternatives to BAL.

PULMONARY OEDEMA INDUCING COMPOUNDS/LUNG IRRITANTS

During World War I, the chemical warfare compounds with the highest lethality were those that induced pulmonary oedema, and included phosgene and chlorine. **Phosgene** was first used by German Forces on 19 December 1915 and soon gathered a reputation as a dangerous compound—around 85% of deaths resulting from exposure to chemical warfare compounds during World War I were caused by this gas (HMSO, 1972).

Phosgene is rapidly dispersed by wind and is regarded as an agent of short persistence likely to be used only in surprise attacks. It may, however, linger in cellars, tunnels and hollows as it is heavier than air. Phosgene ($COCl_2$) or carbonyl chloride is a colourless gas at ordinary temperature and pressure, possessing a smell of musty hay. It is three-and-a-half times as heavy as air, and is decomposed by water into hydrochloric and carbonic acids. It is one of the most dangerous gases, being practically ten times more toxic than chlorine, but owing to its poor solubility, its action is very slow. Hence, it may sometimes produce poisonous symptoms a few hours after exposure. The victim during this almost symptom-less latent period could collapse with florid pulmonary oedema, if exposed to physical stress.

Pathophysiological hypothesis has been put forward to explain the production of pulmonary oedema. The gas damages the blood–air barrier in the lung and allows the leak of fluid

from the pulmonary capillaries, ultimately leading to production of pulmonary oedema. Some authors have suggested that phosgene could lead to massive reflex vasoconstriction and the production of oedema as in neurogenic pulmonary oedema.

Symptoms and Signs

Eye irritation, coughing, lacrimation, choking and a feeling of tightness of the chest are the usual early symptoms and signs (that is why such agents are also known as **choking gases**). However, **the hallmark** of phosgene poisoning has been recognised to be the occurrence of a latent period intervening between exposure and the onset of the symptoms and signs of pulmonary oedema. During this period, it is notoriously difficult to distinguish the mildly exposed from the severely exposed. The cause of death is usually cardiac failure and circulatory collapse caused by hypoxia.

Diphosgene is an oily liquid having a smell of phosgene. It is heavier than phosgene, is as toxic as phosgene and is intensely lachrymatory. Both phosgene and diphosgene are known as **'Green Cross'**.

Treatment

Removal from the risk of further exposure by suitably protected attendants. Practically speaking, the management of phosgene poisoning is the management of pulmonary oedema. No antiphosgene drug of any proven value is available. Steroids, antibiotics, bronchodilators, respiratory stimulants and cardiac stimulants have all been suggested. Two measures are, however, generally agreed:

- All persons thought to have been exposed to phosgene should be confined to bed. It was revealed during World War I that exertion during the latent period following exposure to phosgene could precipitate acute and even fatal pulmonary oedema.
- Supplemental oxygen therapy in patients who are unable to maintain an adequate arterial oxygen tension while breathing air.

NERVE AGENTS

First produced in Germany during the late 1930s, these are often described as second-generation chemical weapons to distinguish them from those used during World War I. Schrader synthesised the first nerve agent, **Tabun** in late 1936. **Sarin** followed later in 1937 and **Soman** in 1944. Tabun, Sarin and Soman are usually referred to by the abbreviations GA, GB and GD, respectively. During the 1950s, another group of nerve agents was developed, the more toxic V agents including VE, VM and VX. Nerve agents may be disseminated by a variety of means and may be encountered as vapour, liquid or artificially thickened liquid in the case of GD.

Nerve agents are organophosphorous anticholinesterases (anti-AchEs) and exert their toxic effects by long lasting inhibition of acetylcholinesterase (AchE) at the sites of activity of acetylcholine (ACh) in the body. (For details, see organophosphorous compounds under in the Chapter "Agro-Chemical Poisoning").

Hydrogen Cyanide

Of the poisons known to the general public, cyanide, arsenic and strychnine are perhaps the best known. Hydrogen cyanide has been but little used as a chemical warfare agent and its physiochemical and toxicological characteristics make it unsuitable for such use on any other than a fairly small scale. It has, however, been used as a means of judicial execution and was used for the large scale homicides of prisoners in German concentration camps during World War II.

Hydrogen cyanide is less dense than air and, therefore, rapid dispersion of hydrogen cyanide greatly reduces its value as a chemical warfare agent. It is almost impossible to establish a lethal concentration of hydrocyanic acid in the field and this is particularly true when the gas is put over in artillery shells. Below 26° C, hydrogen cyanide occurs as a colourless to yellowish-brown liquid. In its usual slightly impure state, it is unstable, although it is said to be stable when highly purified. On standing, the polymerisation takes place, and the compound may present an explosive hazard. It emits bitter almond–like smell, but not all individuals are able to detect the odour. The capacity to detect hydrogen cyanide rapidly wanes on exposure owing to failure of cells of the olfactory mucosa.

Dizziness and nausea have been recorded in people exposed to sublethal quantities of hydrogen cyanide, the effects lasting for some hours. Therefore, the usual notion that the use of hydrogen cyanide leaves 'the quick and the dead' but no incapacitated or partly incapacitated individuals may not be correct.

Lesser Used Chemical Warfare Agents

A few comments need to be made on some older compounds of interest and on the riot-control agents, which could be encountered during modern warfare.

EARLY IRRITANT COMPOUNDS (LACRIMATORS/ TEAR GASES)

In the early twentieth century, the Paris police used grenades containing ethyl bromoacetate against rioters, and it has been alleged that some of the police involved, later conscripted into the French Army, used the same grenades against the German Forces (the military abbreviation for ethyl bromoacetate, SK, is said to stand for South Kensington, the location of Imperial College). In addition, other compounds including xylyl bromide, benzyl bromide, ethyl and methyl chlorosulphonate, bromobenzyl cyanide (BBC), ethyl iodoacetate (KSK) and 2-chloracetophenone (CAP/coded as CN) have also been used.

These chemicals produce intense irritation of the eyes with a copious flow of tears, spasm of the eyelids and temporary blindness. There may be some irritation of the air passages also. BBC and KSK being liquids before volatilisation of the gaseous state may cause, in addition, blistering and ulceration of the skin or conjunctivae from liquid splashes. While CAP is not persistent, BBC may persist on the ground for 30 days in cold climate and KSK for 10 days. The effects are purely temporary incapacitating persons for some hours and are not dangerous to life. However, deaths have been reported after exposure to abnormally high concentrations of chloracetophenone (tear gas).

In addition to the above lacrimators, more **toxic irritant compounds were developed**, which included orthochlorobenzylidene malononitrile (CS) and dibenzoxazepine (CR).

CS was introduced as a riot-control device to replace CN. It is a white crystalline solid, ten times more potent an irritant than CN but significantly less toxic. It has a pungent, pepper-like odour. The immediate irritative effects of its smoke act as a rapid sensory warning, which compels the victim to seek fresh air and so limit exposure. Under ordinary circumstances, the victim is able to affect his escape into the fresh air where he recovers completely in a few minutes without the need for treatment.

CR is another highly irritant chemical, which can be used for riot control. It is a yellow crystalline solid about 6–8 times more potent than CS, though less toxic. It can be used as an aerosol or in solution.

STERNUTATORS OR NASAL IRRITANTS

Sternutators are solid, organic compounds of arsenic, which are dispersed by heat or detonation in the form of very fine, particulate clouds or smokes. The compounds that may be used during war are as follows:

- **Diphenylchlorarsine** (DA), a colourless crystalline solid. It is slightly soluble in water, but dissolves in phosgene and chloropicrin.
- **Chlorodihydrophenarsazine** (DM or Adamsite), a yellow, almost odourless, crystalline solid. It is not soluble in phosgene and tarnishes metals.
- **Diphenylcyanarsine** (DC), a white, odourless, crystalline solid.

The vapours of these substances, when inhaled, cause intense pain and irritation in the nose with excessive sneezing, malaise, headache, painful gums, salivation, nausea, vomiting, pain and tightness in the chest and temporary prostration. The marked sneezing led these compounds to be called as sternutators (Latin word 'sternuto' meaning sneezing). Water and food contaminated by these substances may give rise to symptoms of arsenic poisoning.

Treatment

It is fresh air. The nose should be irrigated with a 5% solution of sodium carbonate. Gargles of same solution may be used if there is irritation of the throat. Excessive concentrations, overage formulations, and careless use of these agents may result in undesirable effects, a situation that is true for any chemical. However, there must be balancing of desired effects against unavoidable but possible detriment. If these agents are used properly, their obvious value for the purposes intended, greatly overshadows the infrequent unwanted toxic reactions that have been recorded.

MISCELLANEOUS

Very little is known about this group. Sometime back, 'yellow/red rain' struck in Laotian tribesmen, and MIC (methyl isocyanate) produced the most gruesome tragedy in Bhopal, India.

Yellow/Red Rain

Laotian tribesmen were struck by two gases, one coloured yellow and the other red. They experienced the combined effects of mustard gas, phosgene, chlorine and nerve poison. Both the gases made their victims feel as if their body was going to blow up. Coughing yielded blood. There was burning in the throat and swallowing was painful. These symptoms were followed by eyes turning yellow as if the victims had jaundice, the vision becoming blurred, and the nose tingling as if hot pepper had been inhaled. Breathing caused sharp pain, teeth felt loose, and the gums smelled rotten.

Methyl Isocyanate

Methyl isocyanate is a fairly stable liquid at room temperature except during summer. It has a boiling point of 31° C. It reacts vigorously with water including moisture, alkaloids and many common solvents. It, therefore, requires to be stored under completely inert conditions.

Clinical Features

Methyl isocyanate is known to be intensely irritating to the eyes, nose and throat. Lacrimation, sore throat, choking sensation, coughing and vomiting develop quickly after exposure. Subsequent corneal ulceration and opacities (some 50,000 people were blinded at Bhopal) and pulmonary oedema were the most common complications responsible for high morbidity and mortality. Exposure to MIC may also result in a higher loss of pregnancy and neonatal mortality. Animal studies have demonstrated that MIC produces hyperglycaemia, lactic acidosis, uraemia, hypotension and hypothermia, as well as long-term pulmonary impairment after a single exposure.

Treatment

Decontamination of skin and eyes with saline (0.9%). An ophthalmic opinion should be sought as permanent blindness may ensue. The pulmonary complications should be treated with humidified supplemental oxygen and bronchodilators. The use

of mechanical ventilation with positive end expiratory pressure may be life-saving in severe cases. Antibiotics will be helpful for preventing infection. (Isocyanates are not the same as cyanides, and antidotes for the latter such as nitrites and sodium thiosulphate need not be used for the former.)

Medicolegal Aspects

Methyl isocyanate was involved in one of the most devastating gas disasters, which occurred in Bhopal (Madhya Pradesh) in 1984, leaving more than 2000 people dead and more than 200,000 injured. The unfortunate event occurred in Union Carbide Company manufacturing carbaryl (a carbamate). It was alleged that phosgene along with MIC was responsible for the tragedy, as MIC was being prepared by the mixture of methylamine and phosgene.

Biological Warfare Agents

Today, chemical and biological weapons are considered to form a spectrum ranging from the classical chemical weapons, such as nerve agents and mustard gas to the classical biological agents, such as the anthrax bacillus or the smallpox virus. Between the two extremes are the compounds originally discovered as natural products, for example, bacterial toxins (which were classified as biological weapons, but which are in fact chemicals). Biological agents have gained attention in recent years. What started off as a cold war between nations and led to the manufacture of biological weapons in the early half of the twentieth century has today manifested itself as bioterrorism. Based on recent events, one can anticipate an uncertain tomorrow.

Biological warfare is the intentional use of microorganisms and toxins (microbial, plant or animal origin) to produce disease and/or death in humans, livestock and crops. Biological weapons of mass destruction are usually clubbed with the nuclear and chemical weapons in the acronym NBC. However, biological weapons (BWs) are very different, while nuclear and chemical attacks cause their damage maximally immediately, biological attacks become manifest after sometime. BWs are defined as microorganisms that infect and grow in the target host producing a clinical disease that kills or incapacitates the targeted host. Such microbes may be natural, wild-type strains or may be the result of genetically engineered organisms. These may be the products of metabolism (usually of microbial origin) that kill or incapacitate the targeted host. These include biological toxins, as well as substances that interfere with normal behaviour, such as hormones, neuropeptides and cytokines.

ADVANTAGES

The attraction for BWs is attributed to their following features:

- BWs are aptly called the 'Poor Man's Atomic Bomb'/'Poor Man's Weapons of Mass Destruction'. For atomic bombs, conventional weapons and nerve-gas weapons, the cost per casualty would be approximately $2000, $800 and $600, respectively. However, for BWs the cost would be about $1 per casualty.
- Nondetection by routine scrutiny systems and easy access to a wide range of disease-producing biological agents.
- BWs have the added advantage of destroying an enemy while leaving his infrastructure intact as booty for the winner.
- Perpetrators can escape long before BWs cause casualties (due to the incubation periods of the agents).
- BWs are typically invisible in aerosol clouds and may not be detected until humans become ill. Panic would result as medical capabilities are quickly overwhelmed.

DISADVANTAGES

- Difficulty in protecting the workers at all stages of production, transportation, loading of delivery systems and final delivery, etc.
- Most biological materials, including spores, are likely to be inactivated by exposure to UV light and drying. Agents released in the air may disperse in unexpected ways due to the vulgarities of wind patterns. (BW attacks would most likely occur late at night or early in the morning when agents would be less likely to undergo inactivation by ultraviolet radiation.)
- Special conditions are required to maintain efficacy of these agents. Further, they are often difficult to maintain in weapons delivery state (e.g. loaded and ready to be fired in a rocket).
- Difficult to control once released.

SELECTION CRITERIA OF AN IDEAL BW

- Readily grown and produced in large quantities.
- Efficiently dispersible.
- Highly infectious and highly effective.
- Resistant to treatment.
- Stable on storage.
- Resistant enough to environmental conditions.

SOME EXAMPLES

Bacillus anthracis (Anthrax): It is a toughest known biological agent and is highly lethal (a millionth of a gram of anthrax constitutes a lethal inhalation dose). It is easy to weaponise, is extremely stable and can be stored almost indefinitely as a dry powder. It can be loaded, in a freeze-dried condition, in ammunitions or disseminated as an aerosol with crude sprayers.

Smallpox: Smallpox virus has long been used as a lethal weapon in a biological warfare—the decimation of the American Indian population in 1763 is attributed to the wide distribution of blankets of small pox patients as gifts by the invading powers.

Botulinum toxin (BOTOX): Often tainted as the most toxic substance in the world. Addition to water or food supplies are likely ways of introducing botox into a population as it is tasteless and odourless. Since its symptoms are delayed (2–14 days), the irreversible damage is done before the victims realise the hazard.

Ricin: It is a protein toxin extracted from the castor bean plant. Ricin is already being investigated for its 'magic bullet' properties as an agent that might selectively destroy cancer cells. The same principle could be used to specifically target an enemy.

DISSEMINATION/DELIVERY OF BWs

Dissemination of BWs may occur by aerosol spray, explosives or food or water contamination. Variables that can influence the effectiveness of a delivery system include particle size of the agent, stability of the agent under desiccating conditions, UV light, wind speed, wind direction and atmospheric stability. (Use of an explosive device to deliver and disseminate biological agents is not very effective, since such agents tend to be inactivated by the blast.) Remote-control devices can be used. They can even be set to release material periodically over several days depending on the direction and nature of the wind.

47 CHAPTER

Hydrocarbons—Petroleum Distillates

After going through this chapter, the reader will be able to describe: Toxicity of hydrocarbons with medicolegal aspects | Abuse of volatile substances with medicolegal aspects

Hydrocarbons

Hydrocarbons can be studied under the following two classes:

- **Straight chain saturated and unsaturated hydrocarbons (aliphatic):** The basic aliphatic hydrocarbons include methane, ethane, propane and butane. Liquid petroleum gas is a mixture of propane and butane, while natural gas is a mixture of methane and ethane. **Petroleum distillates are included in this category of hydrocarbons.** The important compounds included in this category are kerosene, petrol (gasoline), mineral seal oil (used for red furniture polish) and naphtha (used in charcoal lighter fluid).
- **Cyclic hydrocarbons (aromatic):** These contain a benzene ring and include benzene, toluene and xylene.

[**The halogenated aliphatic hydrocarbons** (carbon tetrachloride, trichloroethane and related compounds, freons and DDT) have unique toxicities and are considered separately. **The halogenated aromatic hydrocarbons** include various organochlorines, which are widely used as insecticides.]

DETERMINANTS OF TOXICITY

The volatility, surface tension and viscosity of a hydrocarbon are directly responsible for the type and extent of toxicity that results in case of their ingestion or inhalation.

- **Petrolatum, lubricating oil and paraffin wax:** These hydrocarbons have extremely high viscosity, low volatilities and high surface tensions and are therefore, generally nontoxic and harmless except for some mechanical problems that their ingestion may produce (e.g., choking).
- **Methane, propane, ethane and butane:** These compounds exist almost exclusively as gases at standard temperatures and pressures. Butane is used as cigarette lighter fuel. It is occasionally inhaled for getting a 'kick'. These gases exert their toxicities as simple asphyxiants. They are easily absorbed across the alveolar-capillary membrane and produce CNS symptoms.
- **Benzene, toluene and xylene:** These aromatic hydrocarbons are also highly volatile but are well-absorbed from the gut. Of all the aromatic hydrocarbons, benzene is the most toxic.
- **Gasoline (petrol), naphtha, mineral spirit, kerosene, light gas oil and mineral seal oil:** These are petroleum distillates that are predominantly aliphatic mixtures and are poorly absorbed from the GIT. Gasoline and naphtha have relatively high volatilities and can cause CNS depression after the inhalation of fumes even in the absence of pulmonary toxicity.
- **Mineral spirit, kerosene oil and mineral seal oil** have comparable toxicities. They are low-volatility and low-viscosity liquids that produce aspiration pneumonitis. The majority of CNS symptoms are due to the hypoxia produced as a result of aspiration.

COMMON USAGE

Gasoline, naphtha, mineral spirit, kerosene, light gas oil and mineral seal oil are used in a wide variety of products including fuels, industrial degreasers, lacquer thinner, charcoal lighter fluid and furniture polish. Turpentine is a common household solvent, used for mixing oil-based paints and removing paint stains.

FATAL DOSE AND FATAL PERIOD

Ingestion of >10 ml of kerosene may be fatal, although recovery has followed ingestion of 250 ml. The maximum allowable concentration in air is 500 ppm. The presence of benzene in gasoline increases the toxicity. Petroleum products have a low surface tension with the result that small quantities spread over a large surface area, such as the lung and cause intense pulmonary irritation leading to chemical pneumonitis. Fatal period is, therefore, a few hours.

CLINICAL FEATURES

- **Gastrointestinal toxicity:** Virtually all hydrocarbons when ingested produce nausea, vomiting, abdominal pain and sometimes diarrhoea. The ingestion of benzene and turpentine can produce haematemesis.
- **Pulmonary toxicity:** Immediately upon aspiration, there are signs of irritation of the oral mucosa and the tracheobronchial tree in the form of a burning mouth, coughing, choking and gasping. As the chemical spreads rapidly to the lower levels of the respiratory tract, bronchospasm may develop resulting in a mismatching of ventilation and perfusion. This results in hypoxia and CNS depression. Intercostal and subcostal recessions are common. The damage of airway mucosa can cause atelectasis, bronchopneumonia and the formation of a hyaline membrane. In severe cases, haemoptysis may occur.
- **CNS toxicity:** Volatile petroleum distillates and halogenated hydrocarbons may rapidly attain a high concentration in the CNS and suppress the central ventilatory drive. The other acute effects include coma and seizures. Some compounds may produce initial euphoria, agitation, hallucinations, tremors and seizures followed by CNS depression.
- **Systemic toxicity:** This includes hepatic and renal damage (carbon tetrachloride), renal failure (diesel fuel), proteinuria and haematuria (turpentine), haemolysis and disseminated intravascular coagulation (gasoline), myocardial injury, cardiac arrhythmias and myoglobinuria, etc.

SPECIFIC FEATURES OF KEROSENE INTOXICATION

Kerosene oil is used as a fuel in stoves. Ingestion is quite common in India, particularly in children, as it is often placed in unlabelled bottles within their reach.

- **Ingestion of kerosene oil** generally produces an immediate burning sensation in the mouth and pharynx, as well as nausea and vomiting. In the absence of aspiration, about 40–60 ml/kg of kerosene can be tolerated without any significant systemic effects. CNS depression accompanying kerosene ingestion is the result of hypoxia. It is manifested by giddiness, blurred vision, pale or cyanosed face, dyspnoea, drowsiness deepening to stupor, coma and death. The pupils are at first contracted, but become dilated when coma supervenes. Convulsions may occur in some cases. (The breath, vomit and urine usually give off the peculiar smell of kerosene.)
- **Aspiration of even 0.2 ml of kerosene** can produce chemical pneumonitis. Depending upon severity, the picture varies from mild tachypnoea and coughing to dyspnoea, cyanosis and pulmonary oedema.

  [The chest X-rays taken early in the course of ingestion may not demonstrate aspiration pneumonia and, even if demonstrated, the severity cannot be gauged by degree of X-ray findings. However, X-rays should be repeated on follow-up to detect development of pneumonitis or demonstrate pneumatoceles. The decision for hospitalisation should be based on clinical criteria (e.g., cyanosis, respiratory distress) and X-ray findings.]
- The inhalation of kerosene may produce dyspnoea, fever, severe hypoxia, bilateral pulmonary infiltrates and a notable decrease in differential leucocyte count. These features usually resolve spontaneously.
- Intravenous use of kerosene may produce acute apnoea and cyanosis and later, pulmonary oedema and pulmonary infiltrates.

TREATMENT

- Suction the secretions from the airways.
- Evaluate and maintain the ventilatory status of the patient. Administer oxygen to all patients with respiratory symptoms.
- Due to the risk of aspiration, do not perform gastric decontamination or induce emesis in patients ingesting any volume of hydrocarbons. However, a lavage is indicated if the ingested hydrocarbon contains benzene, toluene, halogenated hydrocarbons or other dangerous toxic compounds. (The stomach should be washed carefully with warm normal saline, avoiding aspiration into the lungs by keeping the head low. Oils or fats should not be given, instead liquid paraffin and smashed banana is advised as it delays absorption.)
- Evaluate patients who show initial symptoms of pulmonary aspiration following ingestion and observe for the development of any respiratory complications.
- Obtain a chest X-ray. If the patient is asymptomatic at that time and the initial chest X-ray is normal, the patient may be discharged for observation at home. Admit any asymptomatic patient with a positive chest X-ray for observation. The patient may be discharged if he remains asymptomatic for 6–12 hours. Always admit patients with respiratory symptoms on arrival without regard to radiographic findings (kerosene spreads easily and rapidly in the lungs because of its low surface tension and viscosity).
- Antibiotics, oxygen and positive end-expiratory pressure need to be instituted as indicated. Corticosteroids have disputable role.

POSTMORTEM APPEARANCES

There may be acute gastroenteritis, and the odour of the material may be observed in the contents of the stomach and lungs. Pulmonary oedema and bronchopneumonia are present. Degenerative changes in the liver and kidneys, and hypoplasia of the bone marrow usually occur after prolonged inhalation of high concentrations. Other signs of asphyxia may be seen. The lungs and the brain together with other viscera should be preserved for chemical analysis in saturated saline.

MEDICOLEGAL ASPECTS

Kerosene is occasionally used for suicidal purposes. It may be taken by mouth, or poured onto clothes and the clothes then ignited (self-immolation). Homicidal attempts by pouring kerosene on clothes and igniting them are not uncommon in our country (dowry deaths). However, most fatalities are accidental. The majority of cases have been young children who have taken kerosene by mistake for water/any other fluid. Isolated cases of petroleum poisoning have been recorded among persons attempting to suck petrol out of car tanks through a rubber tube and becoming choked by a sudden rush of petrol into the mouth and throat. Cases of poisoning from the inhalation of petrol fumes may be seen in an industry.

Persons exposed to the petrol fumes for a long time suffer usually from polyneuritis. They may also show evidence of damaged liver and bone marrow depression. Those who work constantly in petroleum distilleries are often found to suffer from skin eruptions.

Volatile Substance Abuse

Volatile substance abuse (VSA) may be defined as the intentional inhalation of volatile organic chemicals other than conventional anaesthetic gases. These may be used alone or in combination with other nonvolatile ingredients. Initially, the most common substance inhaled was a toluene-based adhesive and therefore, the name **glue sniffing** is also being attached to the habit. Table 47.1 shows solvent composition of commonly abused products.

Table 47.1 Chemical Constituents of Commonly Abused Substances

Products	Chemical constituents
Glues/adhesives	Toluene, benzene, xylene, acetone, n-hexane
Petrol (gasoline)	Hydrocarbons, tetraethyl lead
Cleaning fluids	Trichloroethylene, tetrachloroethylene, carbon tetrachloride, toluene
Aerosols	Fluorocarbons
Lighter refills (fluid)	Butane
Acrylic paint	Toluene
Paints, varnishes, lacquers	Trichloroethylene, toluene, methylene chloride
Dyes	Acetone, methylene chloride
Nail polish remover	Acetone, amyl acetate
Typewriter correction fluid	Butyl and isobutyl nitrite, halogenated hydrocarbons
Fire extinguishers	Bromochlorodifluoromethane

Volatile substance may be poured into a container for **sniffing**, or it may be **huffed** (sprayed onto a cloth held to the mouth), or **bagged** (sprayed into a plastic bag and then inhaled). Abusers often begin with 'sniffing' (lower concentrations), and progress subsequently to 'huffing' and 'bagging' (higher levels of exposure). Glue is most often sniffed from a potato crisp bag and repeated abuse in this manner leads to the development of erythematous spots around the mouth and nose ('glue sniffer's rash').

Such chemicals/substances are widely available and cheap also. Most solvent abusers are male adolescents from poor socio-economic background. Boys may take-up 'glue sniffing' as a relief from boredom, in response to peer group pressure, out of curiosity, or in an attempt to gain status. Some studies have found a higher incidence of solvent abuse in children from single parent families and families with paternal unemployment.

CLINICAL FEATURES

The clinical features of intoxication with volatile substances are similar to those of alcohol intoxication with initial CNS stimulation followed by depression. Other symptoms may include euphoria, headache, abdominal pain, nausea, vomiting, blurring of vision, tinnitus, slurring of speech, ataxia, anorexia, chest pain, bronchospasm, impaired judgement, irritability and excitement. Clouding of consciousness and hallucinations may be seen sometimes. Convulsions and coma may occur. Self-destructive and antisocial acts may be carried out by the individuals under the influence of volatile substances. Psychological dependence and tolerance may develop, but physical dependence is rare. The hair, breath or clothing may smell of solvent, and the clothing is often stained.

DIAGNOSIS AND MANAGEMENT

Clinical features described above and the circumstances are usually enough to arrive at the diagnosis. Confirmation needs to be obtained by detection of solvents in the blood. In some cases of volatile substance abuse, metabolites may be detected in the urine.

Prevention is by far the most important aspect of management of volatile substance abuse. Acute intoxication is usually brief and self-limiting. However, if complications like respiratory depression, cardiac arrhythmias supervene, they need to be treated conventionally. Renal and hepatic complications also require corresponding supportive management. Sedative drugs such as benzodiazepines are contraindicated, since they may aggravate intoxication. Severe agitation may require cautious management with haloperidol, given intramuscularly.

MEDICOLEGAL CONSIDERATIONS

- The major cause of death seems to be sudden cardiac arrest, following an arrhythmia. Solvents appear to have the ability to

sensitise the myocardium to the action of catecholamines. As reported, any sudden 'flight-or-fight' situation, even some considerable time after sniffing solvents, has the ability to precipitate ventricular fibrillation.

- In addition to this physiological mechanism as described above, actual myocarditis can occasionally occur.
- Persistent rebreathing can produce hypoxia and hypercapnoea, which is additive to the toxic effects of the solvent. It is difficult to know if some of these cases are related to erotic hypoxia. (It is believed that partial hypoxia accentuates sexual behaviour and therefore, some may indulge in such activities to achieve such sexual accentuation.)
- Another potent risk is vomiting following the effects of the solvent incapacitating the victim sufficiently to prevent his reflexes from protecting or clearing his air passages.
- When gaseous substances are used, such as butane or propane from cylinders or propellants from aerosol cans, reflex cardiac arrest may occur due to sudden cooling impact on palate, pharynx and larynx from the released gas under high pressure. The mechanism of this phenomenon is disputed, but sudden death due to cooling of the sensitive pharyngolaryngeal site may be comparable to sudden immersion deaths wherein cold water impacts the nasopharynx.
- Chronic painter syndrome: Solvent-induced encephalopathy may lead to development of neurobehavioural syndrome, characterised by anxiety, sleep disorders, memory loss and personality changes.
- Some specific manifestations with chronic use of some of these substances may include peripheral neuropathy (n-hexane), cerebellar dysfunction (petrol), dementia (leaded petrol), increased incidence of leucaemia, aplastic anaemia and multiple myeloma (benzene), etc.

AUTOPSY

The scene should be visited if possible. The paraphernalia of sniffing may be seen in its original state. Clothing should be examined carefully. Any soiling with adhesive or solvent stains should be detected and should be kept for investigation. They should be packed as early as possible in a nylon bag to retain any vapour (these solvents will penetrate a polyvinyl-chloride bag and may be lost). **Externally,** the face should be observed for any signs of chronic or recent solvent abuse. The act of holding a bag against the lower face often contaminates it with glue or solvent and produces skin lesions. The rest of the autopsy may be unrewarding. Rarely, there may be a fatty liver from long-standing damage from the solvent, especially the halogenated hydrocarbons. Samples for histopathology should be taken to determine the state of the myocardium, liver and brain.

In cases where vigorous resuscitation and oxygen administration has been carried out, much of the solvent is likely to blow off. However, some absorbed solvent will travel back into alveolar air from the lung tissue and be detected by gas chromatography. Blood samples should be taken in the usual manner, including sample for alcohol determination. A lung should be taken intact for the investigation. The appropriate method is to open the pleural cavity and pass a string ligature round the hilum of the lung, pulling it tight to occlude the main bronchus. This is then drawn tight and the hilum transected. A urine sample may also be obtained for investigation.

48 CHAPTER

Food Poisoning and Essential Metals' Toxicity

After going through this chapter, the reader will be able to describe: Types of bacterial food poisoning | Toxicity of some harmful bacteria | Foods acting as poisonous materials | Food allergy | Essential metals' toxicity

Food Poisoning

The term **food poisoning** in its wider sense includes all illnesses resulting from ingestion of food containing nonbacterial or bacterial products. But it is usually restricted to acute gastroenteritis due to bacterial infection of food or drink. The nonbacterial products include poisons derived from plants and animals, and chemicals migrating into the food from the packaging materials and/or containers. Such foods are sometimes called as **poisonous foods.**

Causes of food poisoning involve the following:

- Bacteria and their toxins (bacterial food poisoning).
- Food poisoning through plants (natural food poisons).
- Food poisoning through animals, for example, poisonous fish, mussel, etc.
- Chemicals: intentionally added such as flavouring agents in processed food, colouring agents, preservatives, etc. Accidentally added such as pesticides and insecticides. Migrants from the packaging materials and metallic contaminants etc.

BACTERIAL FOOD POISONING

Bacterial food poisoning may be divided into two groups—infection type and toxin type.

Infection type results from ingestion of viable microorganisms that multiply in the gastrointestinal tract producing infection, for example, Salmonella group of organisms.

Toxin type may further be of two types, i.e. **when the incubation period is short** (1–6 hours after consumption), the toxin is usually preformed and present in the contaminated food/improperly preserved food. Vomiting is a major complaint and fever is usually absent. Examples include intoxication from *Staphylococcus aureus* or *Bacillus cereus* and toxin can be detected in the food. **When the incubation period is longer** (between 8 and 16 hours), the organism is present in the food and produces toxin after being ingested. Vomiting is less prominent; abdominal cramps are frequent and fever is often absent. The typical example of this disease is that due to *Clostridium perfringens,* toxin can be detected in food or stool specimens.

From the diagnostic and therapeutic standpoint, the infectious diarrhoea may be divided into two syndromes—those that produce inflammatory or bloody diarrhoea and those that are noninflammatory, i.e. nonbloody or watery. In general, the term **inflammatory diarrhoea** suggests colonic involvement by invasive bacteria or parasites or toxin production that affects the large bowel. Common causes of this syndrome include Shigella, Salmonella, Campylobacter, Yersinia, invasive strains of *E. coli, Entamoeba histolytica* and *Clostridium difficile*. **Noninflammatory diarrhoea** is generally a milder disease and is caused by viruses or toxins that affect the small intestine and interfere with salt and water balance, resulting in large-volume watery diarrhoea, often with nausea, vomiting and cramps. Common causes of this syndrome include viruses, vibrios, enterotoxin-producing *E. coli, Giardia lamblia, Cryptosporidia,* and agents that can cause food-borne gastroenteritis.

Staphylococcus aureus

It is perhaps the commonest cause of food poisoning. **I.P.:** 1–8 hours. **Sources:** Meat, milk, dairy products, potato, egg, salad, etc. **Pathogenesis:** Production of enterotoxin. **Features:** Abrupt onset, intense vomiting. Recovery is usual in 24–48 hours. **Diagnosis:** stool culture. No treatment usually necessary except to restore fluids and electrolytes.

Bacillus cereus

A Gram-positive, facultative aerobic, spore-forming rod-shaped organism, producing two types of toxins. One is a heat-labile, large molecular weight protein that produces effects that are similar to those caused by *Clostridium perfringens*. Diarrhoea is the primary symptom in this case. Incubation period is 8–16 hours. The other toxin is a heat-stable, low molecular weight peptide that produces a severe emetic (vomiting) reaction, referred to as *B. cereus* emetic intoxication. Incubation period is 3–6 hours. Sources are fried rice, dried fruit, powder milk, etc. Diagnosis is through culture of contaminated food.

Clostridium perfringens

A Gram-positive, nonmotile, spore-forming, anaerobic rod-shaped, is a normal inhabitant of the human and animal large intestine. Spores of the organism persist in soil, dust and foods (raw meat, poultry, fish, vegetables, legumes, gravies, etc.). **I.P.:** 8–16 hours. **Pathogenesis:** Enterotoxin produced in food and in the gut causes hypersecretion in small intestine. **Symptoms:** Abrupt onset of profuse diarrhoea; vomiting occasionally. Recovery is usual in 1–4 days. Clostridia demonstrable in food and faeces.

Clostridium botulinum

The term **Botulism/Allantiasis** is derived from 'botulismus' meaning a sausage, since large outbreaks of the disease were first observed following ingestion of improperly cooked sausage. **I.P.:** 6 hours to 1 week. Neither the organism nor its spores are harmful, but the toxin, a heat-labile high molecular weight protein produced during growth of the organism under anaerobic conditions, is very lethal. Just a few nanograms of the toxin can cause illness. Toxin absorbed from gut blocks acetylcholine at neuro-muscular junction producing a bilaterally symmetrical descending motor paralysis (bulbar paralysis). Seven immmunogenic types (A to G) of the toxin have been identified. Types A, B and E toxins often cause human botulism, while types C and D cause animal botulism. **Sources:** Under-processed sausages, potted meats, tinned fish, canned acidic vegetables, fruits, etc. **Symptoms:** Diplopia, dysphagia, dry mouth, dysphonia, dysarthria, respiratory embarrassment, etc. **Treatment:** Clearing of the airways, ventilation and intravenous polyvalent antitoxin. (The toxin can be destroyed by heat at 80° C for 10 minutes and, therefore, adequate cooking gives protection against it.) Toxin can be demonstrated in food and serum.

Shigella

I.P.: 24–72 hours. **Sources:** Potato, egg, salad, lettuce, raw vegetables, fruits and milk, etc. **Pathogenesis:** Organisms grow in superficial gut epithelium and gut lumen, and produce enterotoxin and neurotoxin. Organisms invade epithelial cells. Blood, mucus and PMNs in stools. **Symptoms:** Abrupt onset of diarrhoea, often with blood and pus in stools. Cramps, tenesmus and lethargy. Stool cultures are positive. Therapy depends on sensitivity testing.

Salmonella

Sources include beef, poultry, eggs, dairy products. Forms of illnesses may be the following: (*i*) Gastroenteritis—Incubation period is 12–48 hours. Main feature is watery diarrhoea stained with blood or mucus. Stool culture positive. (*ii*) Bacteraemia (usually occurs in infants, old patients and patients with AIDS)—Main features include watery stools followed by septic arthritis, meningitis, etc. Stool culture positive. (*iii*) Enteric fever (Typhoid)—Incubation period is 1 week. Main features include fever, headache, myalgia, cough, etc. Culture of stool, urine and blood. Treatment consists of chloramphenicol, or trimethoprim-sulfamethoxazole, or ampicillin.

Vibrio parahaemolyticus

I.P.: 6–96 hours. **Sources:** Molluscs, crustaceans. **Pathogenesis:** Organisms grow in seafood and in gut, and produce toxin or invade. Hypersecretion in small intestine, stools may be bloody. **Symptoms:** Abrupt onset of diarrhoea in a number of people consuming the same food, especially crabs and other seafood. Recovery is usual. Food and stool cultures are positive.

Vibrio cholerae

I.P.: 24–72 hours. **Sources:** All foods, especially sea food like oysters and crabs. In India, it is usually a waterborne illness. **Pathogenesis:** Organisms grow in gut and produce toxin. Enterotoxin (choleragen) causes hypersecretion in small intestines. **Symptoms:** Explosive watery diarrhoea. Needs prompt replacement of fluids and electrolytes. Stool cultures positive.

Campylobacter jejuni

It is said to be one of the commonest causes of diarrhoea in the world. **I.P.:** 2–10 days. **Sources:** Water, milk and meat. **Pathogenesis:** Organisms grow in jejunum and ileum, and invade intestinal mucosa. Elaboration of enterotoxin. **Symptoms:** Watery or bloody diarrhoea, along with fever, abdominal pain and headache. Usually self-limiting. Special media needed for culture at 43° C. Dark field microscopy of stool. Culture of stool or blood.

Escherichia coli

It is a part of the normal flora of the intestinal tract of humans and other warm-blooded animals. Food-borne diarrhoeagenic *E. coli* are grouped into **four categories** according to virulence properties, clinical syndromes, differences in epidemiology and distinct O:H serogroups. They are enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enterohaemorrhagic *E. coli* (EHEC), and enteroinvasive *E. coli* (EIEC). The mechanism by which this micro-organism causes illness has not been

completely defined, but verotoxins produced by the organism and adhesion of the organism to intestinal cells have been associated with *E. coli's* virulence. Transmission of the illness is primarily due to food, but person-to-person transmission has also occurred in some *E. coli* outbreaks. **I.P.:** 1–3 days. **Sources:** Salads, cheese, meats, water, raw vegetables, apple juice, etc. **Pathogenesis:** Invasion of intestinal mucosa and elaboration of enterotoxin. Stool culture positive.

Traveller's Diarrhoea

Whenever a person travels from one place to another—particularly if the change involves a marked difference in climate, social conditions, or sanitation standards and facilities—diarrhoea may develop within a few days. Usual symptoms include abdominal cramps, nausea, occasionally vomiting and fever. The stools do not usually contain mucus or blood. The illness usually subsides of its own within a week or so. Bacteria like enterotoxigenic *E. coli*, Shigella species and *Campylobacter jejuni* are the most common pathogens in it. Contributory causes may at times include unusual food and drink, change in living habits, occasional viral infections (adenoviruses or rotaviruses), and change in bowel flora. In most individuals, the affliction is short-lived and symptomatic therapy with 'loperamide' is all that is required provided the patient is not systemically ill and does not have bloody stools. (In many cases, a specific diagnosis is not necessary or not available. One can proceed with the information obtained from the history, stool examination and evaluation of the severity of dehydration. **World Health Organisation** recommends a solution containing 3.5 gm sodium chloride, 2.5 gm sodium bicarbonate, 1.5 gm potassium chloride, and 20 gm glucose/40 gm sucrose per litre of water.)

Postmortem Appearances

An important feature in most cases (except botulism) is gastrointestinal congestion. In some cases, there may be ulceration of intestinal mucosa. Botulism fatalities are usually characterised by a lack of postmortem findings. Asphyxial signs may be present.

Medicolegal Aspects

Isolated cases of food poisoning may have medicolegal importance, for example, the deliberate addition of a culture of microorganisms in the food of an intended victim of homicide. Cases of mass food poisoning are not uncommon in India, and usually occur during functions or celebrations when food is served to a number of guests. Contamination may result from unhygienic measures during the preparation or storage of articles of food. The public health authorities must be contacted to take charge of the situation. Doctors coming across cases of food poisoning from public eateries such as hotels and canteens must report them to the public health authorities.

Botulism represents a special type of problem on account of its distinctive symptomatology and high mortality. Fortunately, it is a rare type of food poisoning in India since canned food (the main source of *Clostridium botulinum*) is relatively unpopular here.

Ptomaine Poisoning

Ptomaines are alkaloidal bodies produced by the action of saprophytic micro-organism upon nitrogenous material during decomposition. When these are formed in the dead tissues, they are known as **cadaveric alkaloids**. Alkaloids secreted by living cells during metabolism are called **leucomaines**, which are slightly toxic when injected into an animal but have no action when ingested. They are not bacterial poisons and are not derived from bacteria. Ptomaines closely resemble vegetable alkaloids but are nonpoisonous, except neurine and mydaleine, which are actively poisonous and produce symptoms resembling those of poisoning by atropine, muscarine and aconite. By the time food decomposes and they are developed, the food becomes so unpalatable that it is not likely to be eaten. They are, therefore, not common agents in the causation of food poisoning.

POISONOUS FOODS

By usual implication, this term excludes conventional food poisoning by bacteria and their toxins and is restricted to poisoning by articles of food that contain poison derived from plants, animals and inorganic chemicals. Metallic contamination of food or drink and migrants from packaging material may also be included in this entity.

Lathyrus sativus (Kesari Dal)

This is a drought-resistant pulse. It is a staple food for the low income groups in some areas of Central India. Consumption of seeds in quantities exceeding 30% of the total diet for more than six months has been known to cause paralysis (neurolathyrism). The active neurotoxic principle is B(N) oxalyl-amino-alanine, which is present as a free amino acid in the seed cotyledons to the extent of about 1% and has a predilection for the pyramidal tracts. The condition manifests as a spastic paralysis of the lower limbs due probably to a localised lesion of the pyramidal tracts. In mild cases, there is only stiffness and weakness of the legs.

Steeping the pulse in hot water and parboiling remove most of the toxic amino acid. Rich diet with exclusion of the pulse and massage, etc. are useful. Death in the acute stage is very rare. Sclerosis of the lateral columns is seen on postmortem examination.

Mushrooms

All mushrooms are not poisonous. *Amanita phalloides* and *Amanita muscaria* are the common varieties of poisonous fungi. The former is also known as **deadly agaric or death cap**. Active principles are phalloidins (only in Amanita species), amatoxins (most important toxic principle) and virotoxins.

Amatoxins are powerful inhibitors of cellular protein synthesis. Cells with high replication rates (liver, kidney, intestinal cells) develop necrosis. Liver usually shows centrilobular necrosis.

Symptoms

There is usually an incubation period of 6–12 hours. Three stages may be recognised: (*i*) Gastroenteritis stage: lasting for 24 hours and is characterised by cholera-like diarrhoea, abdominal pain, vomiting, fever, etc. (*ii*) Latent stage: lasting for a variable period during which most of the symptoms subside. However, a patient may return in 2–3 days in a moribund condition. (*iii*) Hepato-renal stage: showing jaundice, confusion, delirium, metabolic acidosis, coagulopathies, renal failure and coma.

Diagnosis

Amatoxins in the stools or gastric samples (or in the mushroom specimen itself) may be detected by **Meixner Test.**

Treatment

Treatment consists of gut decontamination, correction of dehydration, forced diuresis and other supportive measures.

Rye, Wheat, Oats, Barley and *Bajra*

These grains, when badly stored, allow growth of fungus *Claviceps purpurea*, consumption of which may cause ergot poisoning.

Argemone mexicana

Common names: prickly poppy, *pila dhatura*, *Ujarkanta*, *kutila*, *sial-kanta*. It is a herbaceous plant belonging to the family Papaveraceae and grows wild in the countryside all over India. It bears spiny leaves, yellow flowers, and oblong, prickly capsules containing a number of small black seeds, which resemble mustard seeds. Oil can be extracted from the seeds, which is known as **argemone oil** (katkar oil). About 1% of argemone oil as an adulterant is necessary to produce clinical epidemic dropsy. When the seeds are pressed on a slide, they burst with a report, whereas mustard seeds collapse silently.

Active Principles

Seeds contain berberine and protopine, while the oil contains sanguinarine and dihydrosanguinarine.

There is no legitimate use for this plant, but the seeds and oil are used illicitly to adulterate mustard seeds and oil. When such contaminated products are consumed over a period of time, it results in a condition called **epidemic dropsy**. This condition presents with vomiting, diarrhoea, oedema, especially of the feet and sometimes a generalised anasarca may be seen. Pleural and pericardial effusions, hepatomegaly, and congestive heart failure with breathlessness may also be noticed. Hair loss is noted in some cases. Death may result from myocardial damage. Treatment consists of withdrawal of contaminated seeds or oil, administration of diuretics and corticosteroids. Other symptomatic measures as necessary.

Medicolegal Aspects

Adulteration of mustard oil with argemone oil is common in certain parts of India. The recent outbreak of epidemic dropsy in Delhi and neighbouring states (1998) claimed nearly a hundred lives, and has projected the catastrophic consequences of edible oil adulteration.

Lolium temulentum (Darnel)

This weed grows in wheat fields. The grains are similar to wheat, but much smaller in size. The grains are attacked by a fungus that contains toxin, temuline. This toxin is contained between the seed coat and the endosperm. A few cases of poisoning, mostly nonfatal, have occurred from the use of bread made from seeds containing darnel. The symptoms are chiefly giddiness, headache, tremors, muscular weakness, dilatation of the pupils, gastrointestinal irritation, stupor and even coma. Death has not been recorded.

Paspalam scrobiculatum (Kodra)

This corn is often used by poor people as an article of food. The poison is supposed to reside in the husk of the grain and can be removed by boiling. The exact nature of the poisoning is not known. The symptoms of kodra poisoning are very similar to those of darnel. The poisoning may end fatally.

Stigmata maides (Maize)

This corn is cultivated everywhere in India. It causes pellagra when eaten due to lack of nicotinic acid. It is thought that maize is poor in the essential amino acid, tryptophan, from which nicotine acid can by synthesised in the body. Moreover, the greater part of nicotinic acid in maize is in a bound unabsorbable form.

Groundnuts

If stored under humid conditions, these are contaminated with metabolites of strains of *Aspergillus flavus* to which the collective name 'aflatoxins' is given. A wide range of domestic and laboratory animals are affected by the toxin. Hepatic damage occurs in almost all cases. With chronic exposure, the toxic agents have carcinogenic property.

Potato

It contains solanine 0.002–0.01%, mainly in the skin. Potatoes that are partly exposed above the soil and 'sunburned' (the skin of exposed part being green) contain considerable solanine and cause poisoning when not thoroughly cooked. Immature and sprouting potatoes contain up to 0.06% of solanine, and may cause severe or fatal poisoning.

Fish and Marine Animals

Ingestion of fish causes two types of poisoning. The first is due to bacterial growth in partially decomposed fish. The other is primary toxicity caused by the presence in certain fishes of a neurotoxin. Most cases of fish poisoning are **icthyosarcotoxic** (involving toxins from muscles, viscera, skin, gonads and mucous surfaces).

Metallic Contamination of Food

Arsenic contamination of iron pyrites, used to prepare sulphuric acid with which starch was converted to sugar, has been known to cause poisoning of beer, confectionery, and baking powder boosted with acid calcium phosphate. Enamels containing antimony have sometimes been attacked and dissolved by lemonade and other acid drinks with the production of illness. Copper is sometimes added to peas to preserve their colour and regular cooking in copper utensils may rarely cause harm. Cider, beer, lemonade or other plumbosolvent liquids stored in lead containers or bottled in siphons containing lead fittings may occasionally cause poisoning by this metal. Peaty waters may collect enough lead in passing through supply-pipes to do the same. Poisoning by tin usually results from solution of the metal from unlacquered tinplate containers by acid fruits or shell fish. Aluminium has no toxic effect, and for this reason it has become accepted as a standard luxury metal for cooking utensils. Zinc is rarely a cause of poisoning. Other examples of chemical food poisoning include illness due to the use of cutlery cleaned with cyanide plate powder, and the ginger paralysis due to the adulteration of fluid extract of ginger with triorthocresyl phosphate.

Migrants from Packaging Materials

With increased urbanisation and growth of the food industry, more and more foodstuffs are pre-packaged before reaching the public. Many problems are involved in packaging because almost any packaging material is subject to slow chemical or physical attack from the food or storage conditions. When some of the packaging material becomes a part of the food as consumed, it is termed as migrant and is classed as an incidental or unintentional additive. The packaging of foodstuffs in plastic materials presents complex problems. Although the plastic itself may be relatively insoluble, partially reacted polymers, plasticisers and contaminants can be dissolved and migrate into the food or become environmental problems. Phthalic acid esters (PAEs), which are extensively used as plasticisers in food wrap films, have been found in whole blood stored in plastic bags.

FOOD ALLERGY

This is due to sensitivity to certain articles of diet, usually proteinaceous in nature. It is followed by an illness characterised by nausea, vomiting, diarrhoea, fleeting joint pains, and urticaria. Oedema of the glottis and asthmatic seizures may also follow. Articles of food like shell fish, eggs, strawberries, mussels, dairy products may be implicated. In this, the individual factor plays a very important part. Antihistaminic drugs are of value.

Essential Metals—Potential for Toxicity Thereof

Metals generally accepted as essential are as follows: zinc, selenium, magnesium, manganese, molybdenum, iron, cobalt, copper and chromium. As advocated by WHO, the traditional criteria for nutritionally essential metals are that their deficiency produces either functional or structural abnormalities and that the abnormalities are related to or a consequence of specific biochemical changes that can be reversed by the presence of the essential metal. For essential trace elements, risk assessment requires consideration of both toxicity from excess exposures and health consequences as a result of deficiencies.

ZINC (Zn)

Zinc is a bluish white lustrous metal. It is used extensively for the coating of iron utensils. The toxic effects of zinc are confined mainly to the use of three salts of the metal, namely (*i*) the sulphate, the action of which is irritant; (*ii*) the chloride, which is corrosive; and (*iii*) the phosphide, which is used as a rodenticide. The sulphate, also known as **white vitriol**, closely resembles magnesium sulphate, and is employed to some extent in medicine, mainly as an astringent. Zinc chloride (butter of zinc) is used therapeutically as an astringent.

Zinc is ubiquitous in the environment. It is present in most foodstuffs, water, and air. Seafoods, meats, dairy products, grains, nuts and legumes are high in zinc.

Essentiality

Many metalloenzymes including oxidoreductases, transferases, hydrolases, isomerases, and ligases require zinc as a cofactor. Zinc is a functional component of several proteins that contribute to gene expression and regulation of genetic activity. As reported, zinc has a role in membrane stabilisation by binding ligands in membranes for maintenance of the normal structural geometry of the protein and liquid components. Zinc is essential for the development and normal function of the nervous system. It has a role in immune function and the cytokines, primarily interleukin 1 (IL-1 and IL-06). As documented, there is a reciprocal relationship between plasma levels of zinc and copper, i.e. massive zinc ingestion may produce copper deficiency and large doses of elemental zinc may result in negative copper balance in patients with Wilson disease.

About two-thirds of the zinc is bound to albumin, and most of the remainder is complexed with β_2-macroglobulin. Normal physiological requirement for absorbed zinc is 1.4 mg/day. Bile is the major route of excretion. Urinary excretion is low and not significantly influenced by dietary zinc. In the liver as well as

other tissues, zinc is bound to metallothionein. The greatest concentration of zinc in the body is in the prostate, probably related to the rich content of zinc-containing enzyme, acid phosphatase.

The most reliable index for status of zinc is the determination of zinc balance, i.e. the relationship between intake and excretion. An alternative approach is to identify a biomarker sensitive to changes in zinc status, like metallothionein, serum alkaline phosphatase, and erythrocyte superoxide dismutase.

Deficiency

Its deficiency leads to wide spectrum of effects depending upon age, stage of development and deficiencies of related metals. In humans, it was first reported by Prasad (1983) in adolescent Egyptian boys with growth failure and delayed sexual maturation accompanied by protein–calorie malnutrition, pellagra, and iron plus folate deficiency. Latent zinc deficiency is the most common syndrome, implying marginally adequate zinc status. This may be due to zinc-deficient dietary habits, especially among young children or the elderly, or may occur as a consequence of disease state. Because of its requirement as a cofactor for numerous enzymes and proteins, zinc has a role in various degenerative disease of the nervous system. It has been suggested that zinc modulates the solubility of β-amyloid in the brain and contributes to the formation of degenerative plaques in brains of patients with Alzheimer disease.

Toxicity

Acute toxicity from excessive ingestion is uncommon. However, gastrointestinal distress and diarrhoea have been reported following ingestion of beverages standing in galvanised cans or from the use of galvanised utensils.

Acute Poisoning

It occurs following ingestion of zinc or one of its salts, vomiting, diarrhoea, fever, lethargy, muscle pain and stiffness may occur, and profound anaemia has been reported. Metallic taste in the mouth, pain in the stomach and abdomen are usually present. Ingestion of zinc chloride has led to erosive pharyngitis, oesophagitis and haematemesis. Acute renal failure and pancreatitis have also been reported. Topical exposure to zinc chloride causes ulceration and dermatitis of the exposed skin. Zinc chloride is highly irritant to the eye. **Metal fume fever** (also called Smelter shakes/Brass chills/Monday morning fever, etc.) was described as early as 1669 in brass foundry workers as **'brass foundryman's ague'**. Symptoms of metal fume fever occur up to 24 hour after exposure to zinc oxide fumes. It presents as an influenza-like illness with headache, fever, sweating, chest tightness and discomfort, and joint pains. Typically, symptoms appear after the weekend, thereby giving rise to the term 'Monday morning fever'. The illness usually has good prognosis, and the symptoms often improve towards the end of the working week as some short-term immunity from previous symptoms usually develops. It has been shown that inhalation of zinc oxide fumes is associated with a dose-dependent marked inflammatory response in the lung even if clinical symptoms do not occur. The pathogenesis is not known, but is thought to result from endogenous pyrogen release due to cell lysis.

Other metals that produce this condition may include copper, magnesium, iron, chromium, cadmium, nickel, manganese, mercury, cobalt, lead antimony, silver and aluminium, etc. Exposure to zinc chloride ammunition bombs (hexite) has led to the development of fatal adult respiratory distress syndrome.

Fatal Dose and Fatal Period

Fatal dose is about 15 gm for zinc sulphate and 350–500 mg for zinc chloride. In poisoning by zinc chloride, death may occur within a few hours from shock, or the patient may recover and die after several weeks.

Treatment

The stomach should be washed out unless marked vomiting has removed the poison. There is no specific antidote. Sodium bicarbonate in tepid water may be administered freely. Other symptoms may be treated as they arise.

Postmortem Appearance

These are similar to those of other irritants in case of sulphate and corrosives in case of chloride. Characteristic findings in fatalities from zinc chloride or zinc fume inhalation are pulmonary oedema and damage to respiratory tract.

SELENIUM (Se)

Selenium occurs in nature and biological system as selenate (Se^{6+}), selenite (Se^{4+}), elemental selenium (Se^{0}), and selenide (Se^{22}). Its deficiency leads to cardiomyopathy in mammals, including humans (WHO, 1987). Foodstuffs and daily source of selenium include seafood, meat, milk product, grains, etc. Highest accumulation initially occurs in liver and kidneys. However, appreciable levels remain in the blood, brain, myocardium and skeletal muscle. It crosses the placental barrier. Its increased urinary levels provide a measure of exposure. Urinary selenium is usually less than 100 µg/L. Excretory products appear in sweat and expired air also. The latter may have a garlicky odour due to dimethyl selenide (WHO, 1987).

Essentiality

Selenophosphate is an anabolic form of selenium involved in the synthesis of selenoproteins and seleno-t RNAs. The most documented deficiency of selenium in humans is **Keshan disease** (an endemic cardiopathy first discovered in Keshan Country in China). It occurs most frequently in children under 15 years of age and in women of child-bearing age. Occurrence of disease was invariably associated with a lower content of selenium in the diet of maize and rice than that in grain grown areas. As reported, average selenium concentration in the hair of

residents of affected areas was 0.122±0.010 ppm versus 0.270±0.066 ppm in the hair of residents in the unaffected area.

Toxicity

It may occur where intake exceeds excretion, especially areas where the soil content is relatively rich in selenium, contributing to relatively high selenium in vegetation. Plants vary in their ability to accumulate selenium. Some plants that accumulate high levels of selenium are known as 'selenium accumulators'. These plants, however, usually grow in nonagricultural area and when consumed by livestock may lead to a syndrome known as **blind staggers** characterised by impaired vision, decreased appetite and a tendency to wander in circles. A more characteristic syndrome described in livestock and horses is **alkali disease**, characterised by loss of vitality, shedding of hoofs, loss of long hair, and erosion of joints of long bones.

MAGNESIUM (Mg)

Nuts, cereals, meats, and seafood are high dietary sources of magnesium. The drinking water content of magnesium increases with the hardness of water. Magnesium oxide, magnesium sulphate, magnesium hydroxide, and magnesium carbonate are widely used as antacids or cathartics. Magnesium hydroxide (milk of magnesia) is one of the constituents of the universal antidote.

Essentiality

It is a co-factor for many enzymes involved in intermediary metabolism (in the glycolytic cycle converting glucose to pyruvate, several key enzymes require Mg^{2+}. It is also involved in the citric acid cycle and in the beta-oxidation of fatty acids). In the plasma, about 65% magnesium is in the ionic form, while the remainder is bound to protein. Urine is its major route of excretion under normal conditions. Excretion also occurs in sweat and milk.

Toxicity

It can occur when magnesium-containing drugs (usually antacids) are ingested chronically by individuals with renal insufficiency. Hypermanganesaemia impairs neuromuscular junction transmission by decreasing acetylcholine release from the presynaptic membrane and by diminishing the depolarising action of acetylcholine at the postsynaptic junction. Magnesium oxide fumes can cause 'metal fume fever' analogous to the effect caused by zinc oxide.

MANGANESE (Mn)

It is a traditional metal and can exist in eleven oxidation states from –3 to +7. The most common valency in biological system is +2. It is the oxidative state of manganese in superoxide dismutase and is probably the form that reacts with Fe^{3+}. Manganese is a co-factor for a number of enzymatic reactions, especially those involved in phosphorylation. Vegetables, germinal portions of grains, fruits, nuts, and tea, etc. are usually considered rich sources of manganese.

Manganese is transported in plasma, bound to β_1-globulin and is widely distributed in the body. Manganese concentrates in mitochondria. Biological half-life of manganese in the body is around 37 days. It readily crosses the blood–brain barrier, and its half-life in the brain is longer than in the whole body. Principal route of excretion is with faeces.

Deficiency

Whether its deficiency has actually been reported in humans is questionable (WHO 1996). Deficiency in animals results in impaired growth, skeletal abnormalities and disturbed reproductive functions.

Toxicity

The most common form of toxicity is the result of chronic inhalation of airborne manganese in mines, steel mills and some chemical industries. It may lead to pneumonitis or neuropsychiatric disorder, so-called **manganese madness** (Locura manganica), speech disorders, hallucinations and compulsive behaviour. The outstanding case of manganese encephalopathy has been reported as severe selective damage to the subthalamic nucleus and pallidum. [Some manganese miners on an Australian island have been reported to be afflicted with a peculiar neurological disease characterised by upper motor neuron, cerebellar signs, and oculomotor symptoms **(Angurugu syndrome)**.]

MOLYBDENUM (Mo)

The most important mineral source of molybdenum is molybdenite (MoS_2). It may exist in multiple oxidation states, facilitating electron transfer. It is a component of xanthine oxidase, which has a role in purine metabolism. More than half of its amount is excreted in urine. The blood level is in association with the level in the red blood cells. Chronic exposure to excess molybdenum in humans is characterised by high uric acid levels in serum and urine, loss of appetite, diarrhoea and anaemia, etc.

IRON (Fe)

Absorption of iron is influenced by quantity and bioavailability of dietary iron, amount of stored iron, and rate of erythrocyte production. The best known enhancer is Vitamin C (ascorbic acid). Its absorption occurs in two steps, viz., (*i*) absorption of ferrous ions from the intestinal lumen into the mucosal cells and (*ii*) transfer from the mucosal cells to plasma, where it is bound to transferring (β_1-globulin, produced in the liver) for transfer to storage sites.

Essentiality

Human body usually carries 3–5 gm of iron. About two-thirds of its amount is bound to haemoglobin, 10% is bound to

myoglobin and iron-containing enzymes and the remainder is bound to the iron-storage proteins (ferritin and haemosiderin).

Deficiency

Iron deficiency has been reported to be most common worldwide, affecting infants, young children and women of childbearing age. The major manifestation is anaemia (microcytic hypochromic red blood cells). Iron status is determined by showing low serum ferritin [in some individuals with inborn errors, even normal dietary iron can cause toxic effects due to accumulation, for example, haemochromatosis (bronze diabetes)].

Toxicity

Severe toxicity occurs after the ingestion of >0.5 gm of iron. It is manifested by vomiting, metabolic acidosis and shock. Liver damage and coagulative defects may occur. Liver effects may include liver failure and hepatic cirrhosis. The factors operating for toxicity may include (*i*) mucosal cell damage leading to absorption of ferrous ions directly into the circulation and (*ii*) capillary cell damage in the liver. Inhalation of iron oxide fumes or dust by workers in metal industries may result in deposition of iron particles in lungs, producing an X-ray appearance resembling silicosis.

COBALT (Co)

It is a relatively rare metal produced primarily as a byproduct of other metals. Cobalt is an essential component of Vitamin B_{12} (in the form of cobalamin). Cobalamin is actually synthesised by intestinal flora, which is sufficient for human requirements. This consideration has made nutritionists not to regard cobalt as an essential element for humans. Cobalt salt is generally well-absorbed after oral ingestion. About 80% of the ingested cobalt is excreted in the urine, and about 15% is excreted in the faeces by an enterohepatic pathway. The milk and sweat are other secondary routes of excretion.

Total body burden has been estimated as 1.1 mg. Muscle contains the largest total fraction, but fat has the highest concentration. The normal levels in human urine and blood are about 1.10 and 0.18 µg/L, respectively. Epidemiological study suggests that incidence of goiter is higher in regions containing increased levels of cobalt in soil and water. Cardiomyopathy has been caused by an excessive intake of cobalt, especially from the drinking of bear to which cobalt is added to enhance its foaming qualities (referred as **bear drinker's heart**). Autopsy findings have been reported to show ten-fold increase in the cardiac levels of cobalt. Occupational inhalation of cobalt-containing dust may cause respiratory irritation at air concentrations of 0.002–0.01 mg/m^3. Higher concentrations may be the cause of 'hard-metal' pneumoconiosis, a progressive form of interstitial fibrosis.

[High photon energies (mega voltage range) became available with the development of ^{60}Co **teletherapy units and linear accelerators** (photon is a unit of energy of a light ray or other form of radiant energy). The cobalt teletherapy is a mega voltage machine that uses radioactive materials as the photon source. The ^{60}Co is prepared by irradiating stable cobalt ^{59}Co with neutrons in a reactor. The ^{60}Co source decays to nickel 60 with the emission of a β-particle and two γ rays of 1.17 and 1.33 MeV. Current evidence suggests that thyroid gland and breast are sensitive to cancer induction at relatively low doses of radiation; lymphoid tissue, lung, and liver require moderate doses; and bone requires the highest dose. However, distinguishing such neoplasms from those 'naturally' developing/occurring is beyond the scope of present knowledge. In future, it may be possible to identify specific genetic changes associated with radiation-induced malignancies through the science, the so-called molecular forensics.]

COPPER (Cu)

Copper is not poisonous in the metallic state, but some of its salts are poisonous. The two most commonly known salts are copper sulphate or **blue vitriol** (*nila tutia*) and the subacetate or **verdigris** (*Zangal*). Copper sulphate is a crystalline salt with blue colour and a metallic taste. In small doses, it acts as an emetic, but in larger doses, an irritant. Copper subacetate is a bluish green salt formed by the action of vegetable acids on copper cooking vessels that are not properly tinned.

It is widely distributed in nature and is a nutritionally essential element. Food, beverages, and drinking water are potential sources for excess exposure. Daily intake in adults varies between 0.9 and 2.2 mg. The provisional WHO guidelines for copper in drinking water is 2 mg/L (WHO 1993). Copper exposures in industry may occur in miners, or to smelting operators, welders etc.

Copper is transported in serum where it is initially bound to albumin and later more firmly to ceruloplasmin and transcuprein. The normal serum level of copper is 120–145 µg/L. The bile is the normal excretory pathway and plays a primary role in copper homeostasis. Major amount of copper is stored in liver and bone marrow. Tissue levels gradually decline up to about 10 years of life, remaining relatively constant thereafter. Brain levels, on the other hand, tend to almost double from infancy to adulthood.

Copper is a component of all living cells and an essential component of several metalloenzymes, including type A oxidases and type B monoamine oxidases (cytochrome C oxidase is probably the most important because of its involvement in energy metabolism). Further, copper/zinc superoxide dismutase is present in the cytosol of most cells. This enzyme scavengers superoxide radical by reducing the same to hydrogen peroxide.

Deficiency

The main age groups susceptible to its deficiency are low-birthweight infants and infants having malnourishment after birth. Deficiency is manifested clinically by hypochromic microcytic anaemia refractory to iron. Biomarkers of copper deficiency include ceruloplasmin and serum copper levels, levels of low-density lipoproteins, and cytochrome oxidase activity.

Toxicity

Ingestion of large amounts of copper salts may cause **acute poisoning**. Symptoms usually commence within a short period. There is metallic taste in the mouth with salivation and thirst. Sensation of burning with abdominal pain; vomiting and diarrhoea usually follow. The vomited matter is coloured green or blue and needs to be distinguished from bile or bilious vomit. Addition of ammonium hydroxide turns the copper vomit deep blue, while the bile remains unchanged. The stools are liquid and brown but not bloody. The urine is scanty and inky in appearance. It may contain albumin and casts. In severe cases, jaundice occurs due to both haemolysis as well as direct liver damage produced by copper sulphate. Renal failure may occur due to direct toxicity as well as intravascular haemolysis. Convulsions and coma usually precede death.

Fatal Dose and Fatal Period

Fatal dose is 10–20 gm of copper sulphate or of verdigris. Fatal period varies from 12 to 24 hours. It may be delayed for 3–7 days.

Diagnosis

Serum ceruloplasmin levels are reduced. A value of 35 mg% or less at 24 hours is associated with serious toxicity. Elevated blood copper level beyond 1.5 mg/100 ml exhibits likelihood of serious toxicity.

Management

Gastric lavage should be done with 1% solution of potassium ferrocyanide, which forms an insoluble compound, cupric ferrocyanide. Albumins form an insoluble albuminate of copper and are valuable. Penicillamine, BAL or calcium EDTA are helpful and may be given in usual dosage.

Postmortem Appearances

Greenish blue froth may be coming out of the mouth and nostrils. The striking appearance is the bluish or greenish colouration imparted to the gastric mucosa. The mucous membrane is congested and may show eroded patches. The intestinal mucous membrane may show the same appearances.

Chronic Poisoning

It can result from occupational exposure to copper fumes (during refining or welding) or to copper-containing dust, which causes 'metal-fume fever' with upper respiratory tract symptoms, headache, fever and myalgia. Chronic inhalation of copper sulphate spray used as an insecticide in vineyards can cause **vineyard sprayer's lung disease** characterised by a histocytic granulomatous lung. Chronic contact with swimming pool water containing algicidal copper chemicals can cause green hair discolouration.

Wilson disease is an inborn error of metabolism characterised by excessive accumulation of copper in the liver, brain, kidneys and cornea. D-penicillamine enhances copper chelation in this disease. The disease is characterised by the excessive accumulation of copper in liver, brain, kidneys, and cornea. Serum ceruloplasmin is low, and unbound serum copper is elevated. Urinary excretion of copper is high. The disorder is sometimes referred to as 'hepatolenticular degeneration' in reference to effects of copper accumulation in the brain. Menke's **kinky-hair syndrome** is a sex-linked trait characterised by peculiar hair, failure to thrive, mental retardation and death usually by 5 years of age. The gene responsible for this disease produces a cation transporting ATPase and has some homologies with the gene responsible for Wilson disease (Mercer et al., 1993). In children, another disorder characterised by progressive liver degeneration is known as **Indian childhood cirrhosis** (ICC). Reportedly, two distinguishing features of this disorder are (*i*) widespread brown orcein staining (copper) and (*ii*) intralobular fibrosis progressing to portal cirrhosis and inflammation. **Non-Indian childhood cirrhosis** (idiopathic copper toxicosis) is a similar disorder, reported with rarity in Western countries. The largest non-Indian series of cases are reported by Muller et al. (1996) from the Tyrol region of Australia.

Medicolegal Aspects

Copper sulphate is used as an antidote in phosphorus poisoning. Copper as a metal is not poisonous and swallowed copper coins are not known to cause poisoning. Copper sulphate has been used for suicidal purposes. It is not applicable to criminal administration because of colour and strong metallic taste of its salts. Accidental poisoning may occur when copper has been added in order to keep the green colour of vegetables. The formation of subacetate on copper vessels is an alleged cause of poisoning resulting from contamination of food stored in such vessels. Churches in some developing countries distribute 'spiritual green water' to devotees that contains copper sulphate. When this is ingested, serious toxicity can result. Cooking in copper or brass vessels can cause copper poisoning due to verdigris. Copper poisoning can also result from leaching of copper containers in which carbonated water, citrus fruit juices and other acidic beverages have been stored.

CHROMIUM (Cr III)

It is considered an essential trace nutrient serving as a component of the 'glucose tolerance factor'. It is thought to be a cofactor for insulin action and to have a role in the peripheral activities of this hormone by forming a ternary complex with insulin receptors, facilitating the attachment of insulin to these sites. It is also thought to be concerned with carbohydrate metabolism as suggested by epidemiological studies showing that chromium supplementation improved the efficiency of insulin effects on blood lipid profile.

1
ANNEXURE
Scientific Aids to Investigative Techniques

The convicts, under-trials, detenues and other persons in custody cannot be denied the right to live with human dignity except by placing such reasonable restrictions as are permitted by the law. A crime suspect can indeed be subjected to a sustained and scientific interrogation in accordance with law, but he cannot be tortured or subjected to third-degree methods or eliminated with a view to elicit information, extract confession or derive knowledge about his accomplices, weapons, etc. The extent to which this so-called 'negative privilege' of crude display of physical power by those operating under the shield of 'uniform' and 'authority' could be stretched was highlighted by NHRC in a case wherein death of an under-trial prisoner was attributed by the authorities to head injury resulting from convulsions arising out of high fever. To this, Commission observed, "this was indicative of the extent to which imagination, pressed into convenience, could stretch itself". Some scientific investigative techniques presently being used are as follows.

Brain Fingerprinting

This investigative technique is used to assess recognition of familiar stimuli by measuring electrical brain wave responses to words, phrases, pictures, acronyms, etc. that are presented/flashed on a computer screen. The basis for the test is that the suspect's reactions to the details of an event or activity will be displayed through changes in his brain waves if the suspect had prior knowledge of the event or activity. Brain fingerprinting was invented by Dr. Lawrence Farwell in 2001 while researching the P 300 (an electrical signal known as P 300 is emitted from an individual's brain approximately 300 milliseconds after it is confronted with a stimulus of special significance, for example, presenting a murder weapon on the screen, victim's face, etc.). Dr. Farwell also observed the response up to 800 milliseconds after the stimulus. He called this technique as MERMER i.e. 'memory and encoding related multifaceted electroencephalographic response'. One study showed that several different types of stimuli could be used to determine whether a subject was 'information present' or 'information absent' with respect to several different kinds of information. Brain fingerprinting has also been used as a medical tool that can be used to detect symptoms of Alzheimer disease and other forms of dementia before many of greater degenerative symptoms make their appearance. With early diagnosis, the progression of disease can be delayed through the use of medications and through dietary plus lifestyle changes. In November 2000, an Iowa District Court held that Dr. Farwell's brain fingerprinting 300 test results were admissible as scientific evidence as defined in Congress Ruling 702 and in Daubert vs. Marrell Dow Pharmaceutical.

Polygraph

A polygraph (commonly referred to a lie detector) is an instrument that measures and records several physiological responses such as blood pressure, pulse, respiration and skin conductivity while the subject is asked and answers a series of questions. The polygraph measures physiological changes caused by the Sympathetic Nervous System during questioning. The term 'polygraph' was used first time in 1908 by James MacKenzie in his invention, the 'ink polygraph' which was used for medical reasons. Marston nevertheless remained its primary advocate, endlessly lobbying for its use in the courts.

A typical polygraph test starts with a pre-test interview to gain some preliminary information, which will later be used for 'Control Questions' or C (control questions that most people will lie about may be like: have you ever stolen money? or have you ever committed such crime before also?, etc.). The tester will explain how the polygraph is supposed to work, emphasising that it can detect lies and that it is important to answer truthfully. Then a 'stim test' is often conducted: the subject is asked to deliberately lie and then the tester reports that he was able to detect this lie. This is followed by 'the actual test' wherein some of the questions asked are 'Irrelevant' or IR ('Is your name Rob/Tom?'), others are 'probable-lie'; and the remainder are the 'Relevant Questions' or R in which the tester is really interested in.

Narco-Analysis

Greek word 'narke' means 'anaesthesia' or 'torpor' numbness/apathy/dormancy, etc. The term 'analysis' has been used in Pierre Janet's sense of a process that means of partial dissolution of consciousness, undoes the complex syntheses of waking mental life and accesses mental content that is more automatic. During 20th century, physicians began to employ scopolamine along with morphine and chloroform to induce a state of 'twilight sleep' during childbirth. Scopolamine was known to produce sedation and drowsiness, confusion and disorientation, in-coordination and amnesia for events experienced during the effects of the drugs. It first reached the mainstream in 1922, when Robert House, a Texas obstetrician, used the drug scopolamine on two prisoners in the Dallas county jail. And both the prisoners were found 'not guilty' upon trial, i.e. consistent with the results of Robert's experiments.

Most commonly used drug, presently, is the sodium pentothal/thiopental sodium. (Barbiturates in which the oxygen at C2 is replaced by sulfur are called thiobarbiturates, making the compound more lipid-soluble. In general, structural changes that increase lipid solubility decrease duration of action, decrease latency to onset of activity, accelerate metabolic degradation, and increase hypnotic potency). It is used as an adjuvant to general anaesthetic for procedures of short duration and also, as anticonvulsant and for narco-analysis. Mechanism of action of the drug is by binding GABA(A) receptors in the brain and spinal cord. The site of action is either postsynaptic (as at cortical and cerebellar pyramidal cells and in the cuneate nucleus, substantia nigra, and thalamic relay neurons) or presynaptic (as in the spinal cord). Through controlled administration of drug, the individual is driven into a state of hypnotic trance, and the revelations made during this stage are recorded both in video and audio cassettes. And the person in his/her post-test interview can account the sequence of events by seeing the video recording at conscious level. This can help the individual from not becoming a criminal, which is the aim of *forensic rehabilitation*.

The procedure has been criticised at various platforms (critics voicing the technique as 'betrayal through medicine/pharmacological torture'), and in its latest ruling, the Apex Court held, "Compulsory administration of any of these techniques is an unjustified intrusion into the mental privacy of an individual. Invocations of a compelling public interest cannot justify the dilution of constitutional rights such as the right against self-incrimination guaranteed under Art 20 (3) of the Constitution". Concludingly, the Apex Court made it clear that eight-point guidelines issued by the NHRC in 2000 for conducting narco-analysis test should be strictly adhered to. Amongst the guidelines were (*i*) no lie detector tests should be administered except on the basis of consent of the accused; (*ii*) if the accused volunteers for a lie detector test, he should be given access to a lawyer and the physical, emotional and legal implications of such a test should be explained to him and his lawyer by the police and (*iii*) the consent should be recorded before a judicial magistrate.

2 ANNEXURE

Proforma for Age Certification

MLR No.: _____
DDR/FIR No.: _____ Dated _____ U/S _____ PS _____
Name: _____ S/O, W/O, D/O _____
Sex: _____ Marital status: _____
Address: _____
Occupation: _____
Brought by: _____
Date, place and time of examination: _____
Examined in presence of (in case of female subject): _____
Consent: I, _____ S/O, D/O, W/O _____
R/O _____, give my free and voluntary consent for my/my ward's _____ medicolegal examination including relevant investigations, the nature and consequences of which have been explained to me in the language that I understand. I certify that I have not/my ward has not been examined before for the said purpose.

Signature:

Examinee In presence of (if needed)

Identification marks:
1. _____
2. _____

Brief history (including alleged age): _____
_____
_____

Menstrual history (in case of females): _____

General physical examination:
- Vital signs:
- Mental status: Conscious/co-operative/oriented to time and place
- Height:
- Weight:
- Hair:
  - Moustache
  - Beard
  - Axillary hair
  - Pubic hair
  - Body hair
- Voice: High pitched/low pitched
- Genital examination:
  - Development
  - Deformity (if any)
    - Congenital
    - Acquired
- Breast examination (in females):
- Relevant systemic examination:

Dental examination:

S/8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8/S
S/8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8/S

✓ = Present X = Missing
= Present and fractured S = Space for 3rd molar

Radiological examination:

X-ray plate no.	Part X-rayed	Findings

OPINION: Based on the general physical, dental and radiological examination, the estimated age of the person is between _____ and _____ years.

Date: _____
Place: _____

Signature of the Doctor
(with name and designation)

3

ANNEXURE

Proforma for Medicolegal Examination of Injuries

MLR No.: _____

DDR/FIR No.: _____ Dated _____ U/S _____ PS _____

Name: _____

Age: _____ Years Sex: _____

Address: _____

Occupation: _____

Requested by: _____

Brought by: _____

Date, place and time of examination: _____

Examined in presence of (in case of female victims): _____

Consent: I, _____ S/O, D/O, W/O _____ R/O _____, give my free and voluntary consent for my/my ward's _____ medicolegal examination including relevant investigations, the nature and consequences of which have been explained to me in the language that I understand. I certify that I have not/my ward has not been examined before for the said purpose.

Signature:

Examinee In presence of (if needed)

Identification marks:

1. _____
2. _____

Brief history:

General physical examination:

1. Vital signs: _____
2. Build: _____
3. Weight: _____
4. Height: _____

Relevant systemic examination/referral:

Examination of the injuries:

Sr. no.	Type of injury	Location of injury	Dimensions of injury	Weapon	Duration since infliction/ sustenance	Nature of injury (simple/grievous dangerous)	Referral/ investigation(s), etc.

Note: The above information must include (*i*) type, situation and number, (*ii*) size, shape, depth and direction, (*iii*) condition of edges/margins, ends and floor, (*iv*) foreign body/material adherent or embedded (like metal, glass, hair, dirt, etc.), (*v*) extent of haemorrhage (if recordable), (*iv*) evidence of age of the wound (duration since infliction/sustenance). In case of firearm wounds, additional features like presence of abrasion collar and deposition of firearm residue (with nature and extent of distribution).

Date: _____ Signature of the Doctor
Place: _____ (with name and designation)

Note: It is prudent to conduct photography wherever warranted.

4
ANNEXURE

Proforma for Examination of a Victim of Sexual Assault

MLR No.: _____
DDR/FIR No.: _____ Dated _____ U/S _____ PS _____
Name: _____ S/O, W/O, D/O _____
Age: _____ Years Sex: _____ Marital status: _____
Address: _____
Occupation: _____
Requested by: _____
Brought by: _____
Date, place and time of examination: _____
Examined in presence of _____
Consent: I, _____ S/O, D/O, W/O _____ R/O _____, give my free and voluntary consent for my/my ward's _____ medicolegal examination including relevant investigations, the nature and consequences of which have been explained to me in the language that I understand. I certify that I have not/my ward has not been examined before for the said purpose.

Signature:

Examinee In presence of

Identification marks:
1. _____
2. _____

Brief history

a. History of the incident (in verbatim, in detail):
(with date, time and place of the alleged offence and names if known of the alleged offender/s)
b. Posture acquired during the act (i.e., whether standing, lying, sitting):
c. Whether the victim was menstruating at that time (menarche, LMP, etc.)
d. Use of any contraceptive: _____
e. Whether in senses or intoxicated:
f. Whether struggled/cried for help:
g. Did she experience pain during the act:
h. Was there any emission of semen:
i. Did the assailant wear the condom/use lubricant:
j. Post-coital complaints of pain/discomfort while walking:
k. Did she change the clothes after the incident?
l. Whether passed urine/stool/took bath since the alleged assault:
m. Whether suffering from any general/emotional disease and whether on some medication:
n. History of any past or present STDs:
o. Is she pregnant? If yes, the duration of pregnancy:
p. H/o previous pregnancy(ies), abortion(s) or delivery(ies), etc.

General physical examination:

- Height:
- Weight:
- Build:
- Mental status:
- Vital signs:
- Secondary sexual characters:
- Gait:
- Dental status:

Examination of the clothing:

(Look for tears/hair/foreign material/blood stains/seminal discharge/any other evidence)

Extragenital examination:

(Look for presence of abrasions/bruises/lacerations/stains/foreign body, especially over the face, breasts, back and inner aspects of thighs)

Genital examination:

- Pubic hair: Present/Absent
  Matted/Non-matted
- Length of pubic hair:
- Vulva (labia majora/minora):
- Fourchette and posterior commissure:
- Hymen: Intact/Torn (if torn, position/size, Fresh/old)
- Discharge if any:
- Digital examination (to be done after taking appropriate swabs):
  - Easy/painful
  - Areas of vaginal tenderness
  - Laxity of vaginal orifice
  - Elongation of posterior vaginal fornix
  - Signs of pregnancy (if any)
- Speculum examination: location of injuries on the vaginal wall, cervix, appearance of the cervical os, etc.
- Any other finding:

Samples for laboratory investigation:

1. Clothing (carrying some stain or other evidence)
2. Loose hairs/foreign material over the clothes
3. Matted pubic hair
4. Loose pubic hair
5. Nail scrapings/clippings
6. Swabs from over the breasts/cheeks/inguinal region/any other bite area
7. Vaginal swabs (ant., post., lateral fornices)
8. Vaginal aspirate from the posterior fornix
9. Urethral swab for venereal disease
10. Blood for grouping, toxicology or any other investigation
11. Urine for toxicology (if available)

Above marked samples has/have been labelled, sealed and handed over to Police _____ B. No. _____ of _____ Police station.

OPINION:

Date: _____
Place: _____

Signature of the Doctor
(with name and designation)

Note: It is prudent to conduct photography wherever warranted.

5

ANNEXURE

Proforma for Examination of an Accused of Sexual Offence

MLR No.: _____

DDR/FIR No.: _____ Dated _____ U/S _____ PS _____

Name: _____ S/O _____

Age: _____ Years Sex: Male Marital status: _____

Address: _____

Occupation: _____

Requested by: _____

Brought by: _____

Date, place and time of examination: _____

Examined in presence of (in case of female victims): _____

Consent: I, _____ S/O, D/O, W/O _____ R/O _____, give my free and voluntary consent for my/my ward's _____ medicolegal examination including relevant investigations, the nature and consequences of which have been explained to me in the language that I understand. I certify that I have not/my ward has not been examined before for the said purpose.

Signature:

Examinee In presence of (if needed)

Identification marks:

1. _____
2. _____

Brief history:

- History of the incident (in verbatim, in detail): (with date, time and place)
- Whether passed urine/stool/took bath since the alleged assault:
- Whether changed the clothes after the incident:
- History of any past or present STDs:

General physical examination:

- Height:
- Weight:
- Build:
- Mental status:
- Vital signs:
- Secondary sex characters:
- Dental status:

Examination of the clothing:

(Look for and give accurate description of tears/hair/foreign material/blood stains/seminal discharge/any other evidence on the clothes of the person)

Extragenital examination:

(Look for presence of abrasions/bruises/lacerations/stains/foreign body especially over the face, arms, inguinal region, etc.)

Genital examination:

- Pubic hair: Present/Absent
  Matted/Non-matted
- Penis:
  General development
  Injuries and their distribution (if any):
  Smegma — Present/Absent
  Prepuce — Circumcised/Retractile/Nonretractile
  Frenulum — Intact/Torn
  Discharge from the urethra — Present/Absent
  Any other evidence of STD

Samples for laboratory investigation:

1. Clothing (carrying some stain or other evidence)
2. Loose hair/Foreign material over the clothes
3. Matted pubic hair
4. Loose pubic hair
5. Nail scrapings/clippings
6. Swabs from buccal mucosa
7. Penile swab
8. Urethral swab
9. Blood for grouping, toxicology and any other investigation

Above marked samples has/have been labelled, sealed and handed over to Police_____ B. No. _____ of _____ Police station.

OPINION:

Date: _____
Place: _____

Signature of the Doctor
(with name and designation)

Note: It is prudent to conduct photography wherever warranted.

6
ANNEXURE

Issuing/Supplying Copies of Injury and/or Postmortem Reports (MLR and/or PMR)

MLR/PMR is obtained by the investigating officer during investigation of the case and forms a part of the police file. The report of the postmortem examination is to be prepared by the medical officer and submitted to the police in a prescribed form as per the provisions of Rule 25.35 of Chapter XXV of the Punjab Police Rules (PPRs). And, as per provisions of Rule 25.47, the duty is cast upon the medical officer to give his detailed opinion as to the cause of the death and related issues on a prescribed proforma. This rule clearly lays down that the report is to be kept with the police file of the case. The inquiry/investigation as contemplated under Section 174 CrPC is confined to find out the cause of death and not to be extended for the purpose of finding out the person who caused the death. The opinion of the medical officer is only to aid the investigating officer in the investigation and not to be made public so long as the investigation is in progress. The fact that the Insurance Companies or the victims or the dependents of the deceased can obtain copy of such documents during this period is in the spirit that the aggrieved or his representative, who is already victim of circumstances, should not be further victimised through undue delays in issuing/supplying copies of such documents. This concession has not been made available to the accused by the CrPC. However, in the event of its being relied upon by the prosecution (i.e., when the 'challan' is put up in the court), a copy of each is mandatorily to be supplied to the accused/defence counsel as per provisions of Section 207 of the CrPC (State vs. Gian Singh; Criminal Revision No. 197 of 1980, Delhi HC).

Another armamentarium housing the contention of non-supply of MLR and/or PMR copies to accused/defence counsel during the period wherein the investigation of the case is in progress is the Right to Information Act 2005. Section 8 of this Act deals with the 'exemption from disclosure of information' and clause (h) under this Section provides as "information that would impede the process of investigation or apprehension or prosecution of offenders".

7(a)

ANNEXURE

Penal Provisions Applicable to Medical Persons

Section of IPC	Context
118	Sections 118, 119 and 120 all contemplate the concealment of a design for commission of an offence by persons other than the accused. Under Section 107, such concealment constitutes an abetment. CrPC creates an obligation for the public (including doctors) in respect of several offences of serious nature (Section 39 and 40) to give information to the police.
174	Nonattendance in obedience to an order from the public servant (a doctor receiving summons from the court or from some other authority is duty bound to appear for such court or authority). Refusal or intentional omission to attend is punishable.
175	Omission to produce documents or electronic record to the court or public servant.
176	Intentional omission to give notice or information to public servant. It covers situations like information about commission of an offence, its prevention, or apprehension of an offender, etc.
177	Section 160 of CrPC reserves the right of police to require attendance of witnesses, and Section 161 deals with examination of a witness by the police through investigational interrogation (including those of doctors). Furnishing false information is punishable.
178	Refusing oath or affirmation when duly required by the public servant to make it.
179	Refusing to answer public servant authorised to question.
180	Refusing to sign the statement.
181	False statement on oath or affirmation to public servant or person authorised to administer an oath or affirmation.
191	Deals with false evidence and is based upon recognition of decline of moral values and erosion of sanctity of oath.
192	Fabricating false evidence. The wording of this Section is so general as to cover any species of crime that consists in the endeavour to injure another by supplying false data.
193	Punishment for giving or fabricating false evidence in judicial proceeding or in any other case.
197	Issuing or signing a certificate knowing or believing that the certificate is false has been put on the same footing as the offence of giving false evidence.
198	Using or attempting to use a certificate knowing or believing it to be false in some material point.
201	Causing disappearance of evidence of offence, or giving false information to screen the offender.
202	Intentional omission to give information of an offence to the magistrate or the police by person knowing or having reason to believe that the offence has been committed.
203	Giving false information respecting an offence committed.
204	Destruction of document or electronic record to prevent its production as evidence.
304A	Covers cases wherein a person causes death of another by such acts as are rash or negligent but there is no intention to cause death and no knowledge that the act will cause death (Under English Law, such cases are termed as manslaughter by negligence).
336–338	Rash or negligent acts that endanger human life, or the personal safety of others, are punishable under Section 336 even though no harm follows and are additionally punishable under 337 and 338 if they cause hurt or grievous hurt, respectively. The word 'rashly' means something more than mere inadvertence or inattentiveness. It implies an indifference to obvious consequences and to the rights of others.

7(b)

ANNEXURE

Penal Provisions Affording Protection to Medical Persons

Section of IPC	Context
87	Protects a person who causes injury to another person above 18 years of age by doing an act not intended or known to be likely to cause death or grievous hurt. It appears to proceed upon the maxim *volenti non fit injuria*, i.e. he who consents, suffers no injury.
88	Sanctions the infliction of any harm if the act by which it is caused is done in good faith and for the benefit of the person consenting to the act. Hence, a surgeon performing an operation for the benefit of the consenting person does not stand liable if it entails any harm to that person.
89	Empowers the guardian of a child under 12 years of age or an insane person to consent to the infliction of any harm to the child or the insane person provided the act by which the harm is caused is done in good faith and for the benefit of the child or insane person.
90	Instead of giving positive definition of 'consent', this Section defines it in negative terms. It goes to explain that a consent is not a free consent in the law and is no answer to a charge of crime, if it has been procured by putting a man under the fear of an injury, coercion, or under a misconception of fact, or the consent is given by a person who by reason of unsoundness of mind or intoxication or immaturity of age (a child under 12 years of age) is incapable of understanding the nature and consequences of the act to which the consent was accorded.
91	Excludes acts that are offences independently of harm caused. For example, causing a miscarriage is an offence independently of any harm that it may cause or be intended to cause to the woman. Consent of the woman or her guardian to the causing of such miscarriage does not justify the act.
92	Consent may be dispensed with when the circumstances are such as to render consent impossible or when, in the case of person incapable of assenting, there is no one at hand whose consent can be substituted. This Section sanctions emergency action taken by a medical man on his own initiative acting in good faith in the interest of the individuals.
93	No communication made in good faith for the benefit of the person is an offence by reason of any harm to the person to whom it is made. A doctor communicating to the patient about his/her serious condition sending some feeling of shock to the patient may not be considered to commit any offence. However, ethics may be questionable.

8
ANNEXURE

Standard Weights/Measures/Dimensions of Organs/Tissues

Organ/structure	Male	Female
Heart		
Weight	300–350 gm	250–300 gm
Thickness of walls		
Atria	1–2 mm	1–2 mm
Rt. ventricle	3–5 mm	3–5 mm
Lt. ventricle	10–15 mm	10–15 mm
Circumference of valves		
Aortic	6–7.5 cm	6–7.5 cm
Pulmonary	7–9 cm	7–9 cm
Mitral	8–10 cm	8–10 cm
Tricuspid	10–12.5 cm	10–12.5 cm
Lung		
Rt. lung	360–570 gm	360–570 gm
Lt. lung	325–480 gm	325–480 gm
Stomach		
Length	25–30 cm	25–30 cm
Capacity	100–1200 ml	100–1200 ml
Small intestine	550–650 cm	550–650 cm
Large intestine	150–170 cm	150–170 cm
Liver	1400–1500 gm	1400–1500 gm
Spleen	150–200 gm	150–200 gm
Kidney	130–160 mg	120–150 gm
Pancreas	90–120 gm	90–120 gm
Uterus		
Weight		
Nulliparous	–	40–50 gm
Parous	–	80–100 gm
Dimension		
Nulliparous	–	$7 \times 5 \times 2\ cm^3$
Parous	–	$10 \times 6 \times 2.5\ cm^3$
Vagina		
Anterior wall	–	7.8 cm
Posterior wall	–	9–10 cm
Ovary	–	5–7 gm
Testis	20–25 gm	–
Brain	1400–1450 gm	1250–1350 gm
Spinal cord	28–30 gm/45 cm	28–30 gm/45 cm

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