Textbook of Forensic Medicine and Toxicology

Book · January 2014

DOI: 10.13140/2.1.2458.0800

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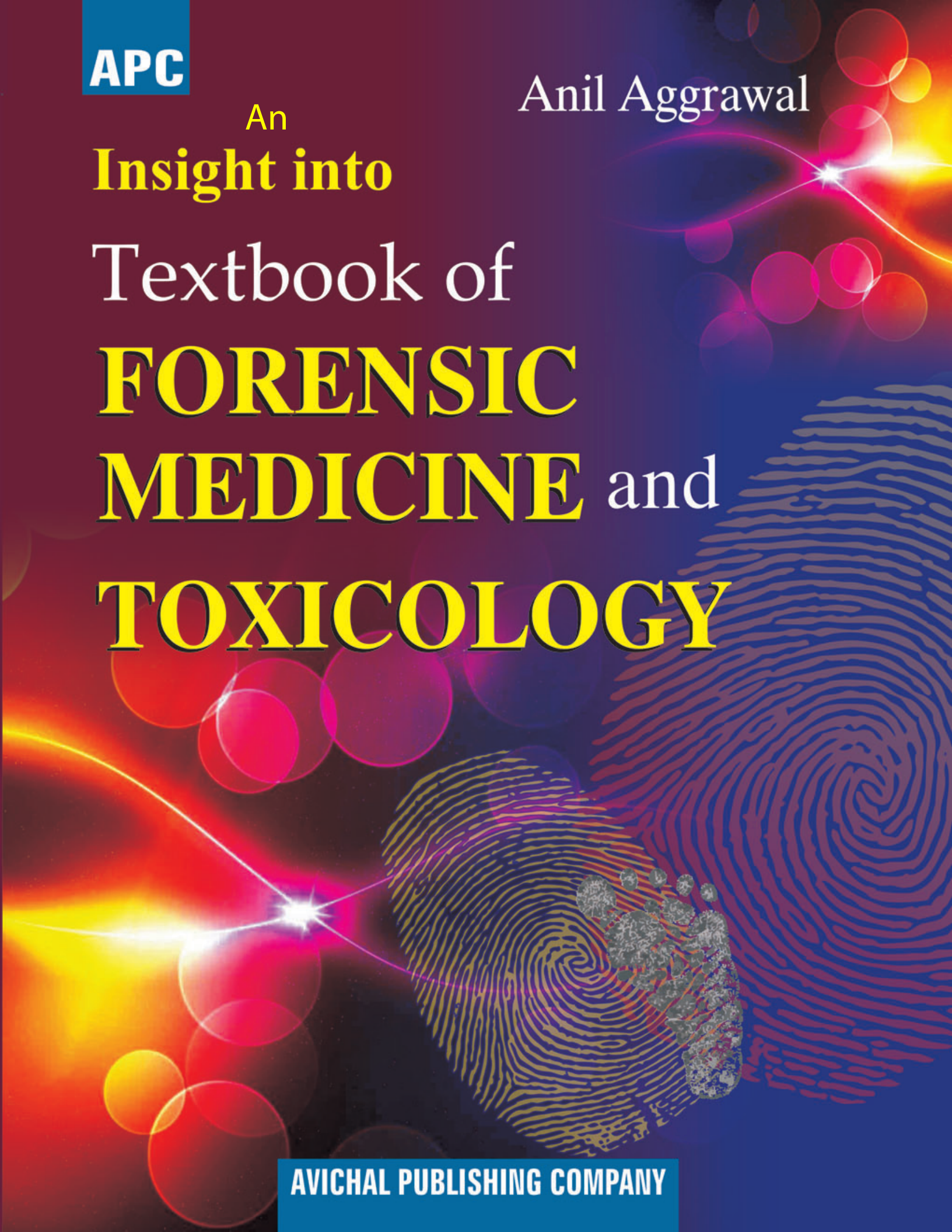
An

Insight into

Textbook of FORENSIC MEDICINE and TOXICOLOGY



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Contents

How this Book is Organized

This book is written primarily for students. Emphasis is on presenting information in a logical, systematic, clear and concise manner for better, easier and faster memorization. Text is presented at two levels – regular information is given in normal font; extra information including conceptual and historical information and case studies has been rendered in small type. This enables the student to quickly differentiate between what is essential for exam, and what can be read at leisure for building of concepts. In addition, the book presents following essential features: court cases, appendices and an extensive index have been included.

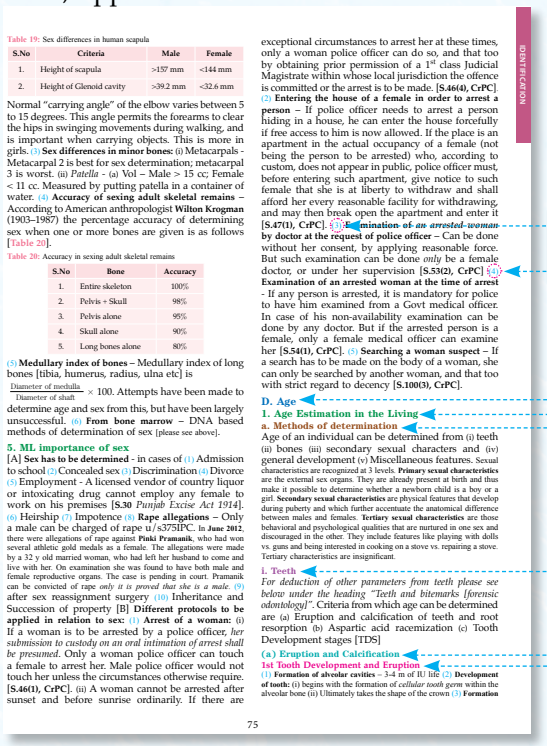

Table 19: Sex differences in human scapula

S.No	Criteria	Male	Female
1.	Height of scapula	>157 mm	<144 mm
2.	Height of Glenoid cavity	>39.2 mm	<32.6 mm

Normal "carrying angle" of the elbow varies between 5 to 15 degrees. This angle permits the forearms to clear the hips in swinging movements during walking, and is important when carrying objects. This is more in girls. (3) **Sex differences in minor bones**: (i) Metacarpals - Metacarpal 2 is best for sex determination; metacarpal 3 is worst. (ii) *Patella* - (a) Vol – Male > 15 cc; Female < 11 cc. Measured by putting patella in a container of water. (4) **Accuracy of sexing adult skeletal remains** – According to American anthropologist **Wilton Krogman** (1903–1987) the percentage accuracy of determining sex when one or more bones are given is as follows [Table 20].

Table 20: Accuracy in sexing adult skeletal remains

S.No	Bone	Accuracy
1.	Entire skeleton	100%
2.	Pelvis + Skull	98%
3.	Pelvis alone	95%
4.	Skull alone	90%
5.	Long bones alone	80%

(5) **Medullary index of bones** – Medullary index of long bones [tibia, humerus, radius, ulna etc] is

$$\frac{\text{Diameter of medulla}}{\text{Diameter of shaft}} \times 100$$

. Attempts have been made to determine age and sex from this, but have been largely unsuccessful. (6) **From bone marrow** – DNA based methods of determination of sex [please see above].

5. ML importance of sex

[A] **Sex has to be determined** - in cases of (1) Admission to school (2) Concealed sex (3) Discrimination (4) Divorce (5) Employment - A licensed vendor of country liquor or intoxicating drug cannot employ any female to work on his premises [**S.30** *Punjab Excise Act 1914*]. (6) Heirship (7) Impotence (8) **Rape allegations** – Only a male can be charged of rape u/s375IPC. In **June 2012**, there were allegations of rape against **Pinki Pramanik**, who had won several athletic gold medals as a female. The allegations were made by a 32 y old married woman, who had left her husband to come and live with her. On examination she was found to have both male and female reproductive organs. The case is pending in court. Pramanik can be convicted of rape only *if it is proved that she is a male*. (9) after sex reassignment surgery (10) Inheritance and Succession of property [B] **Different protocols to be applied in relation to sex:** (1) **Arrest of a woman:** (i) If a woman is to be arrested by a police officer, *her submission to custody on an oral intimation of arrest shall be presumed*. Only a woman police officer can touch a female to arrest her. Male police officer would not touch her unless the circumstances otherwise require. **[S.46(1), CrPC]**. (ii) A woman cannot be arrested after sunset and before sunrise ordinarily. If there are exceptional circumstances to arrest her at these times, only a woman police officer can do so, and that too by obtaining prior permission of a 1st class Judicial Magistrate within whose local jurisdiction the offence is committed or the arrest is to be made. **[S.46(4), CrPC]**. (2) **Entering the house of a female in order to arrest a person** – If police officer needs to arrest a person hiding in a house, he can enter the house forcefully if free access to him is now allowed. If the place is an apartment in the actual occupancy of a female (not being the person to be arrested) who, according to custom, does not appear in public, police officer must, before entering such apartment, give notice to such female that she is at liberty to withdraw and shall afford her every reasonable facility for withdrawing, and may then break open the apartment and enter it **[S.47(1), CrPC]**. (3) ***Examination of an arrested woman*** **by doctor at the request of police officer** – Can be done without her consent, by applying reasonable force. But such examination can be done *only* be a female doctor, or under her supervision **[S.53(2), CrPC]** (4) **Examination of an arrested woman at the time of arrest** - If any person is arrested, it is mandatory for police to have him examined from a Govt medical officer. In case of his non-availability examination can be done by any doctor. But if the arrested person is a female, only a female medical officer can examine her **[S.54(1), CrPC]**. (5) **Searching a woman suspect** – If a search has to be made on the body of a woman, she can only be searched by another woman, and that too with strict regard to decency **[S.100(3), CrPC]**.

D. Age

1. Age Estimation in the Living

a. Methods of determination

Age of an individual can be determined from (i) teeth (ii) bones (iii) secondary sexual characters and (iv) general development (v) Miscellaneous features. Sexual characteristics are recognized at 3 levels. **Primary sexual characteristics** are the external sex organs. They are already present at birth and thus make it possible to determine whether a newborn child is a boy or a girl. **Secondary sexual characteristics** are physical features that develop during puberty and which further accentuate the anatomical difference between males and females. **Tertiary sexual characteristics** are those behavioral and psychological qualities that are nurtured in one sex and discouraged in the other. They include features like playing with dolls vs. guns and being interested in cooking on a stove vs. repairing a stove. Tertiary characteristics are insignificant.

i. Teeth

For deduction of other parameters from teeth please see below under the heading "Teeth and bitemarks [forensic odontology]". Criteria from which age can be determined are (a) Eruption and calcification of teeth and root resorption (b) Aspartic acid racemization (c) Tooth Development stages [TDS]

(a) Eruption and Calcification

1st Tooth Development and Eruption

(1) **Formation of alveolar cavities** – 3-4 m of IU life (2) **Development of teeth**: (i) begins with the formation of *cellular tooth germ* within the alveolar bone (ii) Ultimately takes the shape of the crown (3) **Formation**

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Information presented in points for easy memorization.

The points are in blue color, so student can easily search all points.

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Heading 3

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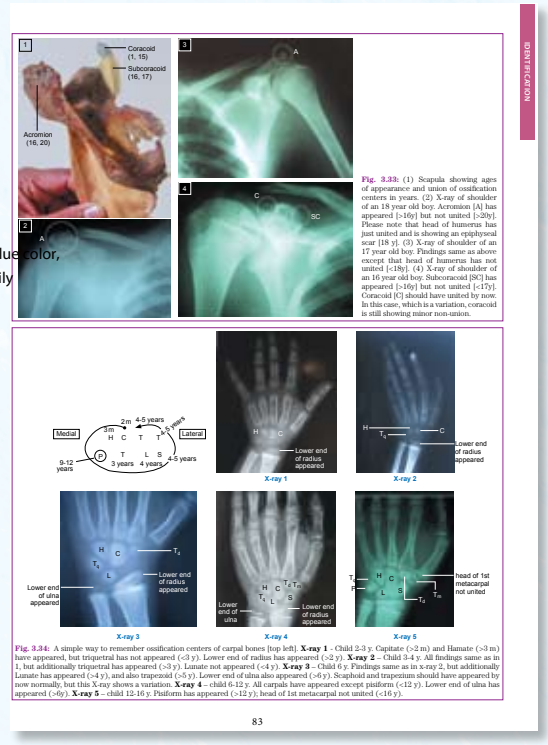

Fig. 3.33: (1) Scapula showing ages of appearance and union of ossification centers in years. (2) X-ray of shoulder of an 18 year old boy. Acromion [A] has appeared [>16y] but not united [>20y]. Please note that head of humerus has just united and is showing an epiphyseal scar [18 y]. (3) X-ray of shoulder of an 17 year old boy. Findings same as above except that head of humerus has not united [<18y]. (4) X-ray of shoulder of an 16 year old boy. Subcoracoid [SC] has appeared [>16y] but not united [<17y]. Coracoid [C] should have united by now. In this case, which is a variation, coracoid is still showing minor non-union.

Fig. 3.34: A simple way to remember ossification centers of carpal bones [top left]. **X-ray 1** - Child 2-3 y. Capitate (>2 m) and Hamate (>3 m) have appeared, but triquetral has not appeared (<3 y). Lower end of radius has appeared (>2 y). **X-ray 2** - Child 3-4 y. All findings same as in 1, but additionally triquetral has appeared (>3 y). Lunate not appeared (<4 y). **X-ray 3** – Child 6 y. Findings same as in x-ray 2, but additionally Lunate has appeared (>4 y), and also trapezoid (>5 y). Lower end of ulna also appeared (>6 y). Scaphoid and trapezium should have appeared by now normally, but this X-ray shows a variation. **X-ray 4** - child 6-12 y. All carpals have appeared except pisiform (<12 y). Lower end of ulna has appeared (>6y). **X-ray 5** – child 12-16 y. Pisiform has appeared (>12 y); head of 1st metacarpal not united (<16 y).

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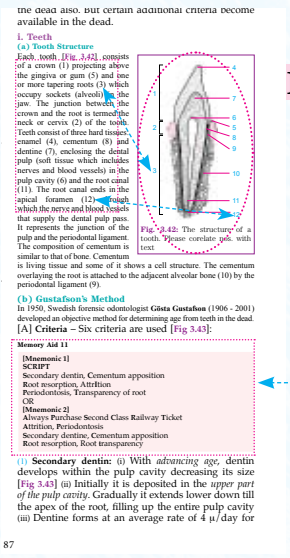
This book presents data under seven levels of headings, each heading being a subset of its predecessor. For ease of differentiation, the colour of each level has been made different.

Full of illustrative color photographs and X-rays all of which are fully labeled for better comprehension.



the dead also. But certain additional criteria become available in the dead.

i. Teeth

(a) Tooth Structure

Each tooth [Fig. 3.42] consists of a crown (1) projecting above the gingiva or gum (5) and one or more tapering roots (3) which occupy sockets (alveoli) in the jaw. The junction between the crown and the root is termed the neck or cervix (2) of the tooth. Teeth consist of three hard tissues – enamel (4), cementum (8) and dentine (7), enclosing the dental pulp (soft tissue which includes nerves and blood vessels) in the pulp cavity (6) and the root canal (11). The root canal ends in the apical foramen (12) through which the nerve and blood vessels that supply the dental pulp pass. It represents the junction of the pulp and the periodontal ligament. The composition of cementum is similar to that of bone. Cementum is living tissue and some of it shows a cell structure. The cementum overlaying the root is attached to the adjacent alveolar bone (10) by the periodontal ligament (9).

Fig. 3.42: The structure of a tooth. Please corelate nos. with text

(b) Gustafson's Method

In 1950, Swedish forensic odontologist **Gösta Gustafson** (1906 - 2001) developed an objective method for determining age from teeth in the dead. [A] **Criteria** – Six criteria are used [Fig 3.43]:

Memory Aid 11

[Mnemonic 1]
SCRIPT
Secondary dentin, Cementum apposition
Root resorption, Attrition
Periodontosis, Transparency of root
OR
[Mnemonic 2]
Always Purchase Second Class Railway Ticket
Attrition, Periodontosis
Secondary dentine, Cementum apposition
Root resorption, Root transparency

(1) **Secondary dentin:** (i) With *advancing age*, dentin develops within the pulp cavity decreasing its size [Fig 3.43] (ii) Initially it is deposited in the *upper part of the pulp cavity*. Gradually it extends lower down till the apex of the root, filling up the entire pulp cavity (iii) Dentine forms at an average rate of 4 μ/day for

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Heading 1

Memory Aids

I. Introduction

(1) **Epidemiology:** (i) *Age* - Most injuries are commonly seen between 20–35 y (ii) *Sex* – More frequent in males.

II. Head Injuries

A ***head injury*** *is any trauma that leads to injury of the* ***scalp***, ***skull***, *or* ***brain***. The injuries can range from a minor swelling on the scalp to serious brain injury. No case of head injury should be taken lightly. All cases of head injury should be **hospitalized for at least 24 hours** for observation. **Head injuries** have been described since antiquity. In the **Bible**, **Ahimelech**, an unprincipled, ambitious ruler, often engaged in war with his own subjects, received head injury when he was engaged in war with the town of **Thebez**, which had revolted against him. He was **struck on the head by a mill-stone**, thrown by the hand of a woman from the wall above. Realizing that the wound was mortal, he ordered his armor-bearer to thrust him through with his sword, so that it might not be said he had perished by the hand of a woman. **David** also killed **Goliath** by inflicting head injury on him; "He put his hand into the pouch, took out a stone and slung it and struck the Philistine [Goliath] on his forehead. The stone sank into his forehead and he fell down on the ground. So David prevailed over the Philistine with a sling and a stone. He struck the Philistine down and killed him, but there was no sword in David's hand." **Sisera**, the commander in chief in King Yabin's army, fled on foot following the complete rout of his troops by the Israelites under the leadership of Deborah and Barak. He made his way to the land of the **Kenites**, a peaceful, neighboring people. There he was greeted and invited into the tent of **Jael**, Heber's wife. After quenching Sisera's thirst with a bottle of milk, Jael covered him with a blanket. Sisera then asked Jael to say that no one was in the tent, if she were questioned by his pursuers. Then Jael took the tent pin and a hammer in her hand and softly went to him. She struck the pin into his head and it penetrated through into the ground, for he was asleep from exhaustion [Fig. 17.1]. The blow was so forceful that the peg pinned his head to the ground.

Fig. 17.1: Jael killing Sisera by producing head injury

A. Epidemiology

Approximately 1.6% of emergency department [ED] visits are for a head injury; up to 90% of these are for Mild Traumatic Brain Injury [mTBI; Table 1]. Risk factors for intracranial injury include male sex, increasing age, and alcohol intoxication.

B. Classification

(1) **Primary head injuries** - Damage directly related to a traumatic event, such as fractures, lacerations, or hematomas. (2) **Secondary head injuries** - Damage incurred as a result of subsequent ischemia, edema, or inflammation. Secondary injury contributes greatly to overall morbidity and mortality in traumatic brain injury [TBI] [Please see (1) primary and secondary intracranial hemorrhages below also (2) cf; **ch 11**→Primary and secondary hemorrhage].

C. Scalp

Scalp injuries are usually the result of direct impact but may not be readily apparent. May manifest as abrasion, bruising, laceration, subcutaneous hemorrhage or edema (**caput succedaneum**), subgaleal hemorrhage or a **subperiosteal hemorrhage (cephalhematoma)**.

1. Anatomy

(1) **Thickness of scalp** – Variable in an adult. Ranges from a few mm to about 1 cm. (2) Layers – Five in all.

Memory Aid 1

S - Skin
C - Connective tissue & cutaneous vessels & nerves.
A - Aponeurosis (epicranial aponeurosis or Galea aponeurotica)
L - Loose areolar tissue ("Danger Zone")
P - Pericranium (periosteum of skull bones)

2. Injuries

Most injuries to the scalp are caused by blunt force [eg blows or falls]

a. Bruising of scalp

(1) **Causes:** (i) Blunt force *without* fracture of skull (ii) *Fractures of the vault of skull* – blood may extravasate into scalp from ruptured diploic veins. (2) **Site** – May occur in (i) connective tissue (2nd layer), (ii) in the temporalis muscle or (iii) in loose areolar tissue (4th layer, **subgaleal hemorrhage**). (3) **Associated features** - Usually associated with prominent edema. (4) **Extent:** (i) Bruises in connective tissue (2nd layer) appear as localized swellings. They are limited in size because of the dense fibro-fatty nature of connective tissue. (ii) Bruises in loose areolar tissue (4th layer, **subgaleal hemorrhage**) are usually very extensive and may spread beneath the galea over most of the skull. Generally referred to as "hematoma of the scalp". This may be very difficult to see with naked eye, and is better felt than seen. In the living, MRI may reveal it. In the dead, becomes visible when the scalp is dissected and reflected. **Caution** - Its firm edges may feel like the edges of a depressed fracture of the skull. (5) Several blows can give rise to different bruises. They can join

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Numerical co-relation of labeling with text. What is given in text can be co-related with numerals in accompanying diagrams and photographs for better comprehension. Several memory aids provided for quick memorization and revision.

Historical information wherever necessary for better understanding. Historical information is always given in small type.

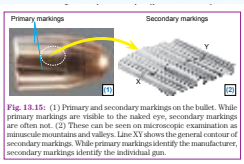
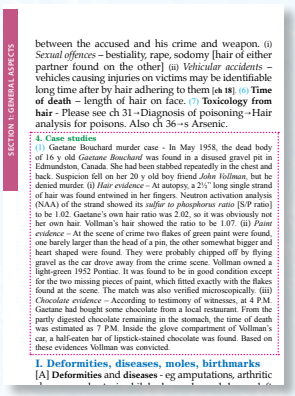


Fig. 13.15: (1) Primary and secondary markings on the bullet. While primary markings are visible to the naked eye, secondary markings are often not. (2) These can be seen on microscopic examination as minuscule mountains and valleys. Line XY shows the general contour of secondary markings. While primary markings identify the manufacturer, secondary markings identify the individual gun.

Microscopic features shown enlarged by "lens technique" wherever required.



between the accused and his crime and weapon. (i) *Sexual offences* – bestiality, rape, sodomy [hair of either partner found on the other] (ii) *Vehicular accidents* – vehicles causing injuries on victims may be identifiable long time after by hair adhering to them [**ch 18**] (6) **Time of death** – length of hair on face. (7) **Toxicology from hair** - Please see ch 31→Diagnosis of poisoning→Hair analysis for poisons. Also ch 36→s Arsenic.

4. Case studies

(1) Gaetane Bouchard murder case - In May 1958, the dead body of 16 y old *Gaetane Bouchard* was found in a disused gravel pit in Edmundston, Canada. She had been stabbed repeatedly in the chest and back. Suspicion fell on her 20 y old boy friend *John Vollman*, but he denied murder. (i) *Hair evidence* - At autopsy, a 2½" long single strand of hair was found entwined in her fingers. Neutron activation analysis (NAA) of the strand showed its *sulfur to phosphorus ratio* [S/P ratio] to be 1.02. Gaetane's own hair ratio was 2.02, so it was obviously not her own hair. Vollman's hair showed the ratio to be 1.07. (ii) *Paint evidence* – At the scene of crime two flakes of green paint were found, one barely larger than the head of a pin, the other somewhat bigger and heart shaped were found. They were probably chipped off by flying gravel as the car drove away from the crime scene. Vollman owned a light-green 1952 Pontiac. It was found to be in good condition except for the two missing pieces of paint, which fitted exactly with the flakes found at the scene. The match was also verified microscopically. (iii) *Chocolate evidence* – According to testimony of witnesses, at 4 P.M. Gaetane had bought some chocolate from a local restaurant. From the partly digested chocolate remaining in the stomach, the time of death was estimated as 7 P.M. Inside the glove compartment of Vollman's car, a half-eaten bar of lipstick-stained chocolate was found. Based on these evidences Vollman was convicted.

I. Deformities, diseases, moles, birthmarks

[A] **Deformities** and **diseases** - eg amputations, arthritic

Case studies for better grasp of subject. All case studies are given in small type.

persons of high social status avoid tattoos. (2) **Country** of origin (3) **Perversions** – drawing of indecent figures (4) May reflect **travel history, involvement in wars, sex interests** etc. (5) May cause hypersensitivity reactions.

L. Occupation marks

Occupational marks *or* ***stigmata*** *are special marks on the body of a person, produced as a result of his profession or habits.* These may sometimes be helpful in identifying a person. Sherlock Holmes often impressed his colleague Watson by telling the occupation of his clients simply by observing occupational marks. Occupational marks may be temporary or permanent.

[A] **Recent and temporary:** (1) **Bakers and millers** – flour (2) **Dye workers** – dyes (3) **Engineers and mechanics** – grease (4) **Painters** – paint spots. In dead bodies, examine microscopically dust and debris (i) on clothing (ii) in pockets (iii) trouser turn-ups at the lower end (iv) under fingernails and (v) in the ear wax. This would often tell the occupation of the dead person.

[B] **Permanent** – If marks are due to constant friction between body part and some object of work, the marks are termed **frictional occupational dermatosis** [eg callosities on manual workers]. These are relatively permanent; may disappear a few months or years after the profession is discontinued. (1) **Athletes** – Black discoloration of toenail (Tennis Toe, Jogger's Toe). Due to subungual hematoma resulting from repeated minor trauma to the nail plate. Related to quick stops or slamming of the toe into the toe-box of the shoe. Occur under the large toenails of tennis, squash, and racquetball players; the second and third toes of soccer and football players, and in the fourth and fifth toes of runners and joggers. (2) **Basketball, racquetball, and tennis players** - Irregular, black macular lesion on the heel (Black Heel, Calcaneal Petechiae, Talon Noir). Caused by hemorrhage due to sudden stopping and starting (3) **Battery Charger** - Contact with sulphuric acid in the batteries Blackens the skin which will not wash off. In addition there is some roughening and thickening of the skin on the outer side of the tip of the index finger due to screwing on terminals. (4) **Boxers** - Fibromatous nodules overlying the finger joints (*knuckle pads* or *Garrod's pads* because first described by Garrod in 1893). (5) **Blacksmiths** – Scars on hands, caused by burns from hot fragments (6) **Bricklayers** – Flattening of the thumb and index finger of left hand due to constant picking up of bricks (7) **Butchers** – Gripping the knife in the right hand thickens the skin on the right palm (8) **Carpenters** – Callosities on thumb, index finger and palms. One shoulder is usually higher than the other (9) **Clerks** – Callosities on fingers, especially on the last joint of the right middle finger (*pen callosities*). Was seen especially in olden days, when clerks used to write with rough pens grasped between fingers (10) **Coal miners** - Blue scar over the hands and face, caused by coal-dust entering small lacerations. (11) **Coconut tree climbers** - Forearms on the flexural aspect are hyperpigmented and lichenified with linear striations, due to constant friction with coconut tree bark. (12) **Guitarists** - Inflamed nipples because the edge of the guitar constantly presses against their chests (*guitar nipple*) (13) **Long distance runners:** (i) A small area of ecchymosis in the gluteal cleft [*runner's rump*] (ii) Crusted, erythematous, painful, fissured erosions on nipples that can occasionally bleed (*Jogger's Nipples*). Caused by constant friction from hard fabrics against the unprotected nipple and areola. Women suffer less due to wearing of bras. (14) **Manual workers** - callosities, cuts, hyperkeratosis and scars on the hands (15) **Opticians** – Small cuts on the tips of index finger and thumb inflicted during preparation of glass lenses. Glass dust is found under and around the nails. (16) **Rowers:** (i) Painful and pruritic macules, plaques, and vesicles on hands due to mechanical trauma from rowing (*Pulling boat hands*). (ii) Gluteal callosities from prolonged rowing on unpadded seats (*rower's rump*) (17) **Shoemaker** - A depression on the sternum due to the constant pressure of the "shoemaker's last" against it. (18) **Tailors**- multiple needle prick marks on hands. (19) **Violinist:** (i) Hardened tips on the fingers of the left hand. (ii) Thick, leathery, bark-like, discolored neck skin, because of constant contact with violin (*Fiddler's neck*) (20) **Workers involved with chemicals** – have discolored skin, hair and fingernails (21) Some occupation marks are seen in teeth – please see Forensic odontology below.

M. Anthropometry (Bertillon system, Bertillonage)

Anthropometry *(Gk anthropos, man; metron, measure→literally "measurement of man") refers to the measurement of* ***eleven key dimensions*** *of the individual for the purposes of identification.* **Salient features:** (1) **Earliest** system of criminal identification (2) Started by **Alphonse Bertillon** (1853 –1914) in the year **1883**. (3) **Principle** - Based on the two facts (i) Dimensions of the skeleton *do not change after the age of 21 years* (ii) *Quetelet's law* [please see above under "Stature"]. According to **Quetelet's law**, there is *one in four chance* of two persons having exactly same height. Bertillon reasoned that if one more measurement was added - for example the length of the trunk - the chance of finding the both measurements exactly the same decreased to one in sixteen. By extending the number of measurements to eleven, the cumulative probability of two criminals having exactly the same measurements would be 4^{11} or over 4 million to one. (4) The system included (i) **Descriptive data** – e.g. (a) *color* - of hair, eye and skin [complexion], (b) *shape* - of ears, nose and chin (ii) **Body marks** – e.g. tattoos, scars, moles (iii) **Body measurements** - 11 parameters were used by Bertillon. No individual has all 11 parameters exactly the same. The parameters used were: (1) Height: Standing (2) Height: Sitting (3) Reach: Arms outstretched (middle finger-tip of one hand to middle finger tip of other) (4) Length of head (5) Width of head (6) Length of right ear (7) Width of right ear (8) Length of left foot (9) Length of left forearm (10) Length of left middle finger (11) Length of left little finger (d) Front and side photographs. Also called *portrait parlé* (Fr. The Speaking Portrait), because it was like making a speaking portrait of a person. (5) **Disadvantages** - (a) Measurement requires use of instruments and trained operators. Otherwise two operators may give different values for same person (b) applicable only to adults (c) Calculation errors are there (d) photographs are not reliable (e) Like fingerprinting, anthropometric data is not left at the scene of crime. Presence of criminal at the scene cannot be established by anthropometry (6) **Current status** - This system has now been replaced by dactylography.

N. Dactylography

Dactylography **(Gk** *daktylos*, **finger;** *graphein*, **to write) [syn.** Fingerprinting, dermatoglyphics, Galton system**]** *is a method of identification, based on unique epidermal ridge pattern on the tips of fingers.* **Salient features:** (1) It is the *most important, accurate* and *unfailing* method of positive identification. (2) It is a better modified method of identification than even DNA profiling. *DNA profiling would be same in identical twins, but fingerprints are different even in identical twins.* (3) **Paternity by fingerprints** – Patterns are not inherited and paternity *cannot be proved through fingerprint patterns.* (4) ***Epidermal ridges*** *(also called* ***friction ridges****) are raised portions of the epidermis on the fingers* (also seen on toes, palms, soles). Their function is to amplify vibrations and to assist in gripping rough surfaces and wet surfaces. Sweat gland pores open on their top [Fig 3.61]→Responsible for fingerprints. A subsidiary ridge [syn incipient, nascent

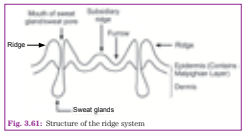


Fig. 3.61: Structure of the ridge system

thigh and the lower part of a leg. Other parts of the body including more than 150 bones and fragments were also recovered.[B] **How body was identified:** (1) Mrs. Parkman identified the body as her husband's from markings near the penis and on the lower back. (2) His brother-in-law said that he had seen the extreme hairiness of Parkman's body and confirmed that the body was his. (3) At his arrest, Webster had claimed that the body parts were from a cadaver used by medical students. Dr. **Jeffries Wyman** along with a team of experts worked with the bone fragments, and concluded that (i) the remains were human, (ii) belonged to a male, (iii) whose height was 5'10" and (iv) who was between 50-60 y of age. All details exactly matched George Parkman. (v) The level of decomposition equaled 7-8 days. This coincided with the length of time Parkman had been missing. (vi) Furthermore there were no traces of embalming fluid, which should have been present, had the body belonged to an anatomy dissection hall. (4) Dr **Nathan Keep**, who had constructed the denture for Parkman identified him from fragments of mineral teeth fused to his gold denture base. (5) Dr. **Lester Noble**, Dr. Keep's assistant, had in his possession, some study models of Parman's lower jaw, which he had made. Recovered portion of porcelain denture fitted accurately the model. [C] **Outcome** – Webster was found guilty of murder and hanged on Aug 30th, 1850. [D] **Significance** – This was the first time that evidence of dental identification was presented in an American court. Such was the interest generated in Boston, that spectators in the court gallery were only allowed to remain for 10 min before relinquishing their places to those waiting outside.

b. Acid Bath Murder Case (1949)

Please see **ch 6**

T. Clothes, ornaments and personal articles

(1) Indicate social status [texture, value of clothes] (2) People of different nationality and races may wear different types of clothes (kurta, pajama, sherwani, tie, necktie, hat, caps). Helpful only rarely. (3) The clothing may bear laundry marks or tailors' tabs (4) quality and condition of the garments may assist in establishing the social status (5) Identity card, ration card, driver's license, cell phone, peculiar rings, shoes etc (6) Forensic examination of clothings - **ch 5**

U. Molecular Methods

Telomere Shortening: (i) There is continuous shortening of telomeres during cell division [50–100 base pairs/cell division]. Thus telomere length decreases with age, mostly in somatic cells. Based on this fact, attempts have been made to predict age of individual from his DNA left at crime scene. (ii) **Weaknesses** – There are other factors which regulate telomere length [eg heritability (telomere length varies among individuals at birth), gender (males lose telomere length more rapidly than females), diet etc.]

V. Miscellaneous methods of identification

1. Biometric methods

Biometrics (Gk *bios*, life; *metros*, measure) *is the science of identification based on a person's unique physical or behavioral traits.* **Salient features:** (1) Most physical and behavioral traits can be accurately captured using sensors and can be compared using computers. (2) **Divisions of biometric methods:** (i) *Physical characteristics* - related to the shape (or other physical characteristics) of the body. **Ex** (a) body odor (b) face recognition (c) Fingerprint (d) hand geometry (e) iris scan (f) Palm print (g) retina scan etc (ii) *Behavioral characteristics* [*behaviometrics*] - related to the behavior of a person. **Ex** (a) gait (b) signature (c) voice etc.

a. Physical characteristics

i. Bone comparisons

If X-ray records of some bone anomalies are available, bones may be compared with antemortem x-rays for identification. (1) **Trabecular meshwork** - of the bones is unique in each individual. (2) **Congenital** - abnormalities, cervical rib, deformities [for *skull deformities*, please see above under "skull"], malunion (3) **Transitional vertebrae:** (i) Developmental variants of the spine. (ii) Found in app 20% individuals (iii) Often involve the sacrococcygeal and lumbosacral junctions. The L5 vertebra can be incorporated into the sacrum ("*sacralized*") or the S1 vertebra can be incorporated into the lumbar spine ("*lumbarized*") [**Fig 3.81**]. (iv) Transitional vertebrae are usually incidental findings during radiologic evaluation. (4) **Implants** [nails, plates etc]. Even if implants are subsequently removed, intra-bone metal particles can still be detected by histopathology and scanning electron microscope-energy dispersive electroscopy (5) **Foreign bodies in the bones** [souvenir bullets, pellets]. (6) **Wormian bones** [syn, extra sutural bones]: (i) Extra bone pieces that occur within a suture in the cranium. Named after Danish anatomist **Ole Worm** [1558–1654] who first described them. (ii) Occur most frequently in the course of the lambdoid suture. Less frequently within sagittal and coronal sutures. (iii) A large wormian bone at lambda is known as an Inca bone (*Os Incae*), due to the relatively high frequency of occurrence in Peruvian mummies. (iv) *Pterion ossicle* [syn, epipteric bone, Flower's bone] – Wormian bone seen between sphenoidal angle of the parietal bone and the great wing of the sphenoid bone. (7) **Miscellaneous** - double articular facets, Ununited sutures [eg metopic suture]

Fig. 3.81: Transitional vertebra – Lumbarization of sacral vertebra

ii. DNA profiling

Please see **ch 29**

iii. Frontal sinus pattern

Besides fingerprints, the ***frontal sinus pattern*** *is another major identification criteria, that is different even in identical twins.* **Salient features:** (1) **History** - The idea of establishing identity by frontal sinus patterns was first suggested in **1921** by **Schuller**. In 1931 Poole corroborated the idea by showing that the pattern was different even in identical twins. (2) **Anatomy and Development:** (i) Appear in **2nd year** of life. (ii) keep growing in size during the first two decades. (iii) Good for identification after they have stabilized, especially in cases where the bodies have been badly burnt or mutilated [eg air disasters]. Antemortem X-rays may be obtained from deceased person's medical records/physicians. (iv) absent in 5% (v) unilateral in 1% (3) **Method of use** - The scalloped upper margins of the sinuses are used. These are smaller and more numerous in the females. Several different techniques are used for comparison (i) tracing out the patterns and compare. (ii) tracing out the patterns on a cardboard, cut it out and then weigh it. Weight derived from both antemortem and postmortem films must be same (iii) computed tomography of frontal sinuses.

iv. Iris scans

Iris patterns of all people are different. In 1987, Leonard Flom and Aram Safir patented the use of the iris as a personal identifier. In 1994 John Daugman at Cambridge University of England, developed the technology for iris scanning [**Fig 3.82**].

Fig. 3.82: The principle behind iris scan

v. Nails

(1) **Nails are helpful in:** (i) decomposed and dismembered bodies (ii) disaster victims (iii) Broken or clipped nails may be present at the scene of crime, or may be found in some hidden place (iv) In kidnapping cases, clipped nails of kidnapped persons might be available from the place where they were held for hostage. (2) **Information from nails:** (i) *identification*

Extra information is given in small type. May profitably be read to get extra credit.

Extensive cross-references to avoid duplication of text.

2. MLI of algor mortis

(1) Helps to estimate TSD. Sometimes two dead bodies lying side by side may show remarkable diff in rectal temp. This may be because of individual body variations, and also because one died earlier. In one case brought before the author bodies of a couple were found inside a closed room. Female's body was significantly cooler than the husband's indicating that it had died earlier. It turned out that the husband had killed wife first, than after some time committed suicide.

3. PM Caloricity

PM caloricity *is a condition when the temperature of body ↑es after death instead of ↓ing.* **Salient features:** (1) **Conditions when it occurs:** (i) **Dead body is lying in the open in hot summers** – Most common [body temp is 37°C, while in hot summers environmental temp may be as high as 45°C] (ii) **Infections** – like cholera, malaria, septicemia, tetanus, typhoid etc. *Cause* - (a) ↑ in temp may already be ↑ed at the time of death (b) Bacterial and other microorganism metabolism continuing after death] (iii) **Other Conditions**- (a) **Sunstroke** - It is due to body lying in the open [sunstroke occurs in open sun]; also the temp of victim is ↑ed at the time of death itself, which may be wrongly taken as pm caloricity. (b) **Strychnine** – Due to excessive convulsions, the temp may be ↑ed at the time of death itself. There is no physiological reason why it should occur in sunstroke and strychnine poisoning.

D. Postmortem lividity (Livor mortis)

Postmortem lividity **(syn**: cadaveric lividity, darkening of death, hypostasis, livor mortis *L. Livor*, blueness; *mortis*, death**)**, livores, postmortem hypostasis, postmortem lividity, Postmortem staining of the cutaneous hypostasis, suggillation) *is the* (i) *bluish-purple or purplish-red discoloration* (ii) *which appears after death,* (iii) *on the most dependent parts of the skin* (iv) *due to collection of blood in the capillaries and small veins (rete mucosam) of the most superficials of the dermis* (v) *due to gravity* [Fig 9.1]. **Salient features:** Cause: (i) Stoppage of circulation (ii) tendency of blood to sink by gravity to the most dependent parts; its collection in toneless BV and capillaries (iii) Backward flow – of venous blood to the capillaries from the venular end [adds to the blueness of pm staining] (2) **Color:** (i) *Hue* – **Bluish purple**, but may vary in different parts of the body (ii) *Intensity* – depends upon the amount of reduced Hb in the blood. Large amounts of reduced Hb produce deep purplish coloration (iii) Portions drained of blood – become correspondingly pale (iv) may depend on cause of death [Table 1]. (3) **Place:** (i) In general – well marked in lobes of ears and in tissues underneath fingernails (ii) back of chest and abdomen in supine position. *Most common* (iii) front of chest and abdomen in prone position; (iv) lower legs and hands in hanging (v) Face in drowning [in drowning, face is the most dependent part]. On the contrary, antigravity regions of the body are drained of blood and become **pale**. (4) **Visibility:** (i) *More clearly seen in* (a) fair skinned people (b) young having good nutrition and normal hemogram (ii) *Less clearly appreciated in* (a) dark colored people (b) old, anemic persons (5) **PM staining and congestion:** (i) *True congestion* - Table 3, (ii) *Hypostatic congestion* – may occur even a few hours before death in persons dying slowly with circulatory failure, eg, asphyxia, CCF, cholera, coma [deep], poisonings [barbiturate (barbiturate blisters also appear because of same mechanism [**ch 40**]), morphine], tuberculosis, typhus and uremia. It is not true congestion, but only because of passive stasis of blood. In such cases, pm staining is quicker to develop. (6) **PM staining and cyanosis** – It is not possible to distinguish between the two [both are bluish purple]. Many pathologists refuse, because, asserting that it cannot reflect the true ante-mortem situation. (7) **Vibices** [syn, **Postmortem Ecchymoses**, **Death Spots**]: (i) *Appearance* - Vibices are tiny, spot-like, sometimes confluent, oval-to-round, bluish-blackish hemorrhages of postmortem origin exclusively limited to areas of livor mortis. (ii) *Cause* - (a) ↑in intravascular hydrostatic pressure [IVHP] due to pooling of blood → PM mechanical rupture of s/c capillaries and smaller vessels [predominantly veins]. (b) IVHP is itself dependent upon (i) total amount of the circulating blood volume [vibices more common in obese] and (ii) posture of the body after death [more common in legs in hanging, where hydrostatic pressure develops too much] (iii) *Microscopy* – RBCs are intact within vibices in the early PM interval. As putrefaction starts, vibices diminish in number and intensity due to the breakdown of erythrocyte membranes with subsequent hemolysis and diffusion of hemoglobin and its derivates into the surrounding tissue. (iv) *D/d* – May be confused with AM petechial bleeding due to asphyxia or DIC. Vibices are however confined only to areas of PM staining.

Fig. 9.6: PM staining. (1) shows a fresh death, where PM staining has not yet appeared. (2) shows a body lying on the back for about 12 hours. Full pm staining has appeared. Note that pressure areas, including those caused by clothes are devoid of pm staining. (3)PM Staining in a person dead for 18 h. Note intense PM staining, and especially its absence on pressure areas, which is typical "butterfly-like" over the scapular region. A peculiar tattoo is also visible on right side of back and right arm.

Numbering at 4 levels (1) → (i) → (a) → (I)

Conceptual information

Topic → definition in italics → salient features

Every topic starts with a definition in italics, followed by salient features. Synonyms of each term are given in brackets. Numbering at 4 levels helps to remember salient features easily. Every term is followed by all possible synonyms in brackets. Etymologies are explained wherever they help to grasp the subject better. Conceptual information is given in small type and wherever necessary, is followed by appropriate examples.

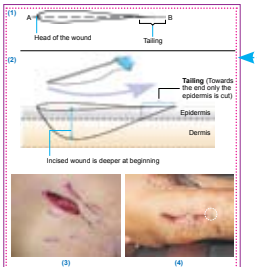


Fig. 12.33: Incised wound is deeper at the beginning and less deep towards the end (1). (2) is a vertical section of the incised wound along the broken line "AB" showing its depth at various points. (3) two incised wounds showing tailing. Sometimes a stitched wound may have to be opened to reveal tailing (4). White dotted circle highlights tailing.

D. Age of incised wounds

1. Gross

(1) **Fresh** – Hematoma formation (2) **12 h** – (i) Edges red, swollen, adherent with blood and lymph (ii) Leukocytic infiltration. (3) **24 h** – Dried clot in the form of crust or scab

2. Histopathology

Following changes are for uninfected wounds and healing by first intention. Figures are considerably different when infection sets in [healing by second intention]. In the latter the inflammatory reaction is more intense and much larger amounts of granulation tissue are formed. Scarring is more. (1) **Few min** – (i) Reactive changes in tissue histiocytes (ii) Dilatation of capillaries (iii) Swelling of vascular endothelium (iv) Margination and emigration of neutrophils (2) **12 h** – (i) Reactive changes in fibroblasts (ii) Monocytes appear in exudate (3) **16 h** – Mitotic divisions begin in monocytes (4) **24 h** – (i) Epithelium begins to grow at the edges (ii) A continuous layer of endothelial cells cover the surface (iii) Vascular buds begin to form (5) **72 h** – Formation of vascularized granulation tissue (6) **4-5 d** – New fibrils are formed (7) **1 wk** – scar formation if wound is small.

E. MLI of incised wounds

(1) **Reconstruction of crime** – Incised wounds help to reconstruct the crime and to corroborate or refute the story of the victim by enabling to determine (a) *the nature of the weapon* – sharp edged (b) *Age of injury* and (c) *Direction of force* (2) **Decomposed body** - Differentiation between incised and lacerated body is difficult (3) **Body with incised wounds immersed in water soon after death**

Fig. 12.35: Homicidal cut throat

an angle >90° with the skin surface (**beveled edge**). The other edge (under mined edge) makes an angle <90° with the skin surface. It mostly lies submerged under the surface. (3) **Nearly horizontal** – If weapon enters nearly horizontally [Fig 12.34(3)], a wound with a flap is produced. [C] **Manner** – Beveling usually indicates homicide, and may indicate relative position of the assailant and victim.

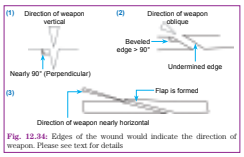


Fig. 12.34: Edges of the wound would indicate the direction of weapon. Please see text for details

7. Hemorrhage

(1) Hemorrhage is **more** as the vessels are cut cleanly. (2) Spurting will occur if an artery has been cut.

C. Cut throat wounds

1. Suicidal and homicidal

2. Cause of death

Death depends upon extent and severity of wounds. Causes are (1) hemorrhage from jugular veins (2) choking due to blood if trachea or larynx are cut (3) air embolism.

Photographs with corresponding line diagrams

Co-relation of photographs with corresponding line diagrams for better comprehension of concepts.

Memory Aids and Mnemonics Index

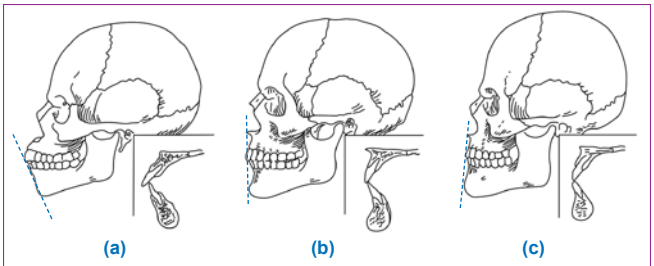


Fig. 3.5: (a) Shape of the negroid skull with prognathism and no nasal sill [floor of nasal opening] (b) Shape of a caucasoid skull with a nasal sill and receding maxillary bones (c) Slope of a Mongoloid skull with projecting maxillary bones and an edge to edge bite in the incisor region

Memory Aid - 1

Cusp of Carabelli is present in Caucasoids.

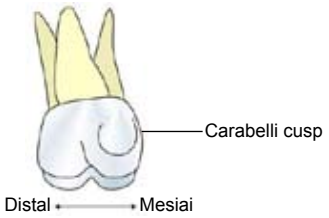


Fig. 3.6: Cusp of Carabelli - a small additional cusp at the mesiopalatal angle of maxillary first molars

a. Skull

i. Cephalic index

Cephalic index *(CI) [syn* ***index of breadth****] is the ratio of the maximum width* (B) *of the head divided by its maximum length (L), multiplied by 100.* CI as a criterion of race was introduced in **1842** by **Anders Retzius** (1796 – 1860). It is now known that besides race, *local environmental conditions also influence the CI.* Its validity in race determination has been challenged.

$$\text{C.I.} = \frac{\text{Maximum breadth of skull (B)}}{\text{Maximum length of skull (L)}} \times 100$$

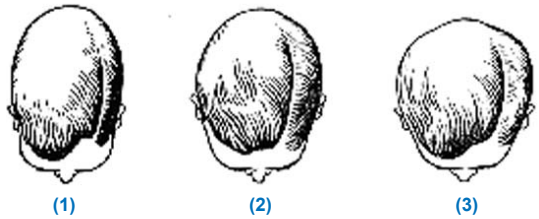


Fig. 3.7: Skulls as seen from the top (1) dolichocephalic (long headed); (2) mesaticephalic (medium headed); (3) brachycephalic (short headed)

Salient features: (1) **Measurements:** (i) Maximum length of skull = Summit of glabella to furthest occipital point.

(ii) Maximum breadth of skull = greatest breadth, at right angles to sagittal plane. (iii) measured by sliding calipers. If not available, use osteometric board (2) As seen from the top, a longer skull is dolichocephalic and a more rounded skull is brachycephalic [Fig 3.7]. Brachycephaly can occur in any other race also if the coronal suture fuses prematurely [flat head syndrome]. (3) Races can be determined to some extent by CI [Table 3]. However as can be seen in the "race" column, there is no sharp racial demarcation. An average Indian has a **mesaticephalic skull** [Strictly speaking *it is wrong to speak of Indians as a single biological group based on race*, because they comprise of a huge admixture of populations].

Table 3: Racial differences in cephalic index

S.No	Type of skull	Cephalic index	Race
1.	Dolichocephalic (long headed)	70-75	Caucasoids (some, eg. Pure Aryans), Negroids [Aborigines and Negroes]
2.	Mesaticephalic (medium headed)	75-80	Caucasoids (some, eg Europeans), Mongoloids (some, e.g. Chinese)
3.	Brachycephalic (short headed)	80-85	Mongoloids (some, e.g. Mongolian)
4.	Hyperbrachycephalic	85-90	Kyushu of Japan

Memory Aid - 2

DuMB Horse

ii. Deformities of skull

If one or more cranial sutures fuse prematurely (craniosynostosis) it may result in an abnormal head shape [Reference source not found.]. These do not help in determination of race, yet useful in identification, because of their rare occurrence. (1) **Scaphocephaly – [**Gk *scaphos*, boat; *kephale*, head] - Boat shaped skull. Due to early fusion of the sagittal suture. Most common craniosynostosis. (2) **Plagiocephaly (anterior)** [Gk, *plagios*, askew]: Early fusion of 1 coronal suture (3) **Plagiocephaly (posterior)**: Early closure of 1 lambdoid suture (4) **Trigonocephaly** [Gk *trigonon*, triangle] - Congenital condition due to premature fusion of the metopic suture leading to a triangular shaped forehead.

b. Long bones

(1) **Brachial index** [Latin *Brachium*, arm]

[syn, radio-humeral index] $= \frac{\text{Length of radius}}{\text{Length of humerus}} \times 100$

(2) **Crural index** [Latin *crus*, leg] [syn, tibio-femoral index] $= \frac{\text{Length of tibia}}{\text{Length of femur}} \times 100$

(3) **Intermembral index** $= \frac{\text{Length of (humerus + radius)}}{\text{Length of (femur + tibia)}} \times 100$

(4) **Humerofemoral index** $= \frac{\text{Length of humerus}}{\text{Length of femur}} \times 100$

Table 4: Long bone Indices in different races

S.No	Indices	Indians	Caucasoids (Europeans)	Negroids
1.	Brachial Index	76.49	74.5	78.5
2.	Crural Index	86.49	83.3	86.2
3.	Intermembral Index	67.27	70.4	70.3
4.	Humerofemoral Index	71.11	69.0	72.4

6. Soft tissues

(1) **Gums** – often brown and mottled in negroids. (2) **Brain** – In negroids there is a dark gray deposit of melanin (i) in the arachnoid membrane around the medulla oblongata and (ii) in the area of the olfactory nerves at the base of frontal lobes. (3) **Retina** – shows dark deposit of melanin in most non-white races.

B. Religion

(1) Hindu males are not circumcised and wear a sacred thread. (2) Muslim males are circumcised, have corns and callosities on lateral aspects of knees and feet and have a black spot on their forehead [From making *sujood*,

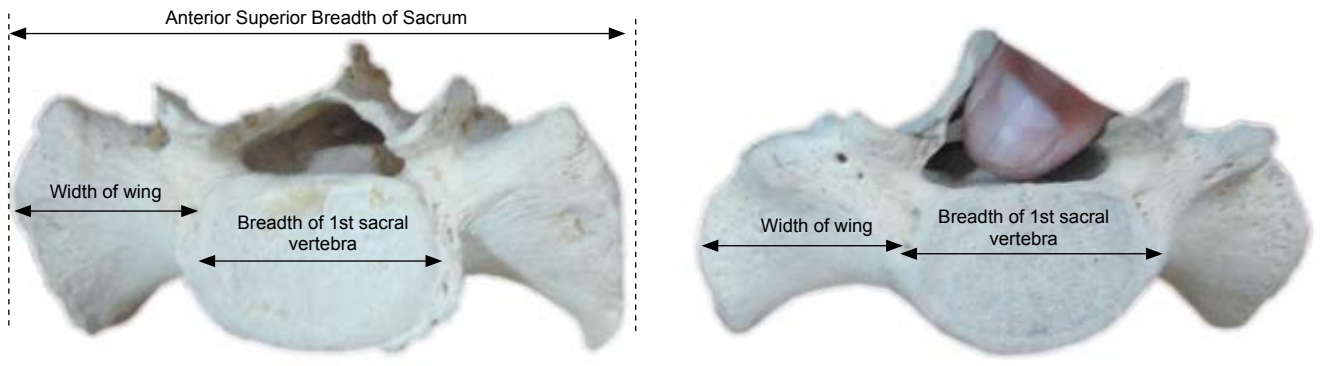


Fig. 3.15: Base-wing index and corporobasal index (please correlate with Table 13)

M.emory Aid - 6 [for 3 indices relating to sacrum]

State **B**ank **C**orporation - **S**acral, **B**ase-**W**ing, **C**orporobasal. For males, figures are in a neat sequence - **105, 65, 45**. Female indices are greater for first 2 and lesser for 3rd. The differences are **10**, **15** and **5** - another easily remembered sequence.

Table 14: Sex differences in the human sternum (Please correlate with **Fig 3.16**)

S.No	Criteria	Male	Female
1.	**Ashley's rule**	Total length>149 mm [also sometimes known as Ashley's "149 rule". Enunciated by Ashley in 1956]	Total length<149 mm
2.	**Hyrtl's law**	Body is longer and more than twice the length of manubrium	Body is shorter and less than twice the length of manubrium
3.	Breadth	more	Less
4.	Length of the mesosternum [Middle piece or body of the sternum]	>95 mm	<80mm
5.	Level of Upper margin	in level with lower part of body of T2	in level with lower part of body of T3
6.	Sternal [manubrio-corpus] index $\frac{\text{Length of manubrium}}{\text{Length of body}} \times 100$	46.2	54.3
7.	width of the 1st sternebra at its waist	27 mm [approx]	24 mm [approx]
8.	width of the 3rd sternebra at its waist	32 mm [approx]	29 mm [approx]
9.	Sternal foramen	Twice as common as in females	Less common

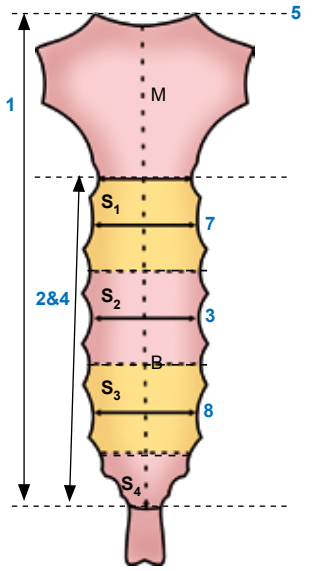


Fig. 3.16: Major differences in male and female sternum (please correlate numbers with Table 14; no 6 not shown)

Table 15: Sex differences in human ribs

S.No	Criteria	Male	Female
1.	Composition	Thicker	Thinner
2.	Curvature	Lesser	Greater
3.	Obliquity [cranio–caudal inclination]	Less oblique. cranio–caudal inclination of ribs is ↓	More oblique. cranio–caudal inclination of ribs is ↑
4.	Length of ribs in relation of body height	Less [ribs are relatively shorter]	More [ribs are relatively longer – better accommodate abdominal distension during pregnancy]

Table 16: Sex differences in human thoracic bones

S.No	Criteria	Male	Female
1.	Shape of thorax	Longer and narrower	Shorter and wider
2.	Volume	More	Less [about 10% less than that of males having the same height]
3.	Clavicle	Longer, broader, heavier, less curved	Smaller, narrower, lighter, more curved

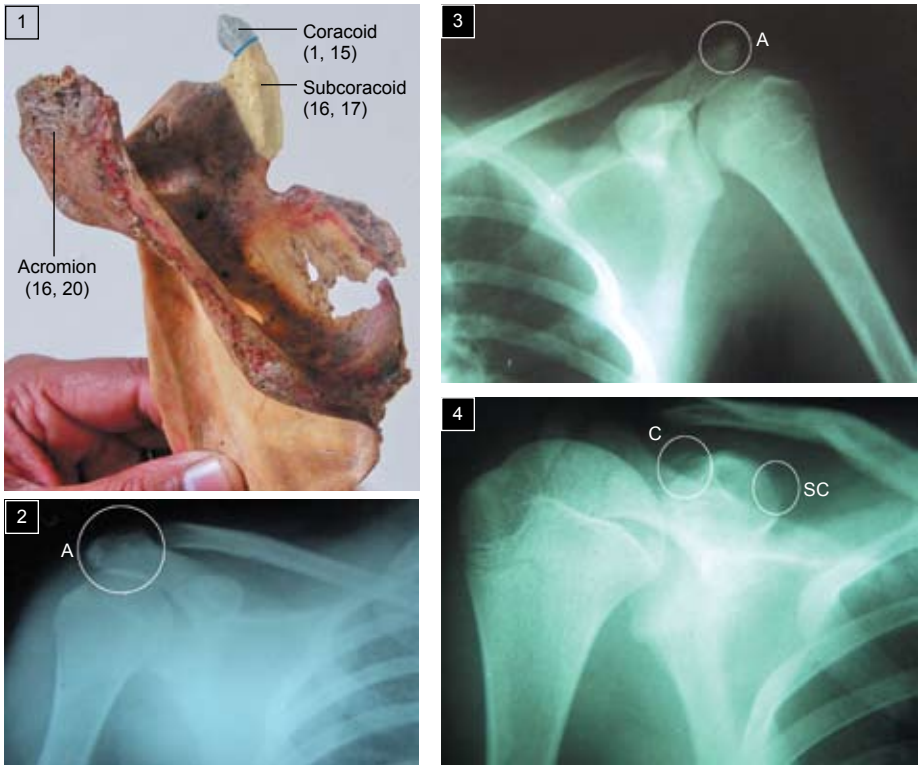


Fig. 3.33: (1) Scapula showing ages of appearance and union of ossification centers in years. (2) X-ray of shoulder of an 18 year old boy. Acromion [A] has appeared [>16y] but not united [>20y]. Please note that head of humerus has just united and is showing an epiphyseal scar [18 y]. (3) X-ray of shoulder of an 17 year old boy. Findings same as above except that head of humerus has not united [<18y]. (4) X-ray of shoulder of an 16 year old boy. Subcoracoid [SC] has appeared [>16y] but not united [<17y]. Coracoid [C] should have united by now. In this case, which is a variation, coracoid is still showing minor non-union.

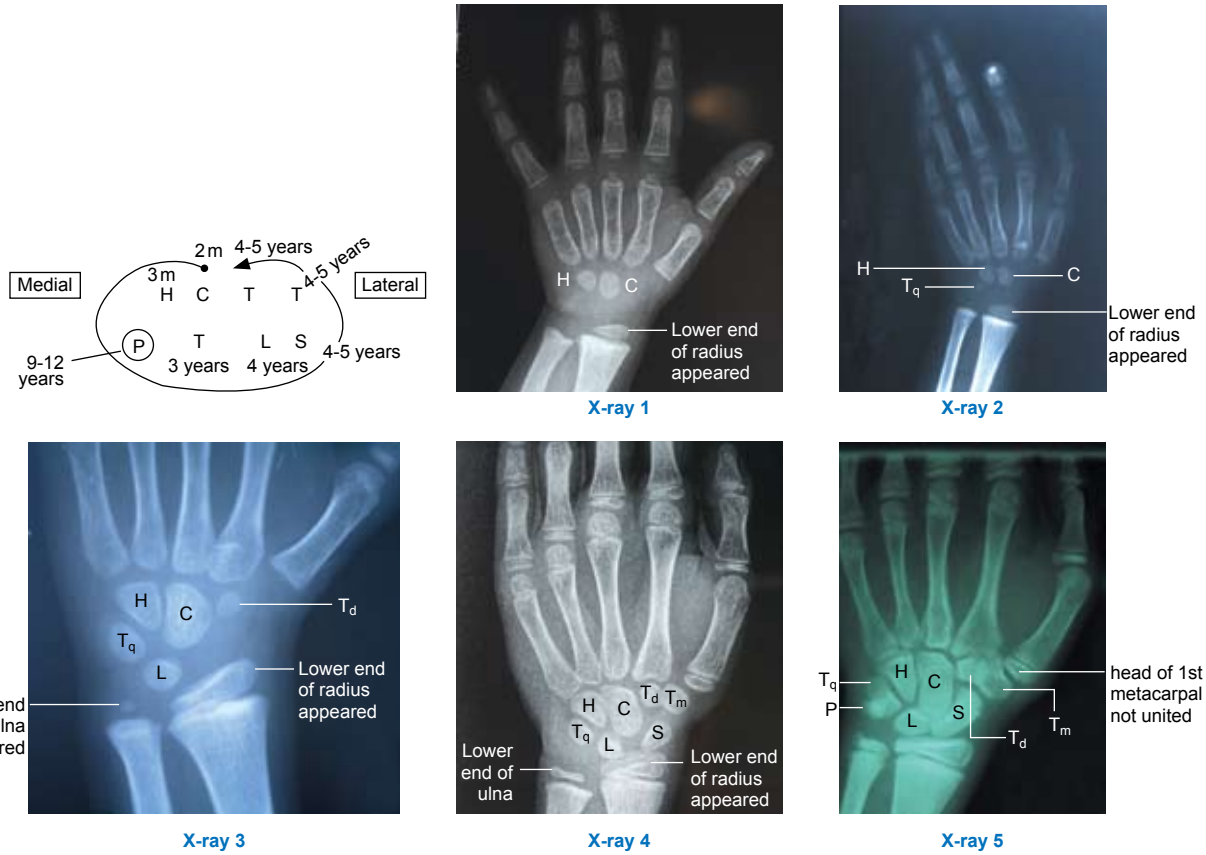


Fig. 3.34: A simple way to remember ossification centers of carpal bones [top left]. **X-ray 1** - Child 2-3 y. Capitate (>2 m) and Hamate (>3 m) have appeared, but triquetral has not appeared (<3 y). Lower end of radius has appeared (>2 y). **X-ray 2** – Child 3-4 y. All findings same as in 1, but additionally triquetral has appeared (>3 y). Lunate not appeared (<4 y). **X-ray 3** – Child 6 y. Findings same as in x-ray 2, but additionally Lunate has appeared (>4 y), and also trapezoid (>5 y). Lower end of ulna also appeared (>6 y). Scaphoid and trapezium should have appeared by now normally, but this X-ray shows a variation. **X-ray 4** – child 6-12 y. All carpals have appeared except pisiform (<12 y). Lower end of ulna has appeared (>6y). **X-ray 5** – child 12-16 y. Pisiform has appeared (>12 y); head of 1st metacarpal not united (<16 y).

5. Examination

(1) **Good lighting** and **magnifying glass** – Essential (because old scars may often be very faint and unrecognizable) (2) **Description** - should include, their number, size, shape, location, nature of surface (whether smooth or irregular), whether fixed or free, its level in relation to the body surface (whether raised above the surface or depressed), whether glistening or not, and its tenderness (3) **Ends** - rounded or tapering (can often help determine the nature of the weapon used to inflict the wounds) (4) **Probable direction** – of the original wound (note tailing). (5) **If the scar is faint** - can be made more visible by the application of heat, UV light or surface friction (6) **Demonstration of scar in a dead body** – can be done by microscopy (scars lack elastic tissue). Elastic tissue is however present in *striae gravidarum*.

6. Erasure

Scars can be erased by (1) Skin grafting and (2) Excision with suturing of edges of excised area. This results in another scar, but which is less prominent that the earlier one.

7. MLI

(1) Identification (2) Shape of scar – Can reveal nature of offending weapon (3) Age of scar – if it corresponds to the date of attack, it is a good corroboratory evidence (4) *Linea albicantes* – indicate previous pregnancy.

K. Tattoos

Tattoos *(Tahitian tatau, a mark) are special marks, designs, pictorial diagrams (e.g. of Gods and Goddesses), or alphabetical messages (e.g. name of self, husband, or a friend etc) made or written permanently on the skin of the body* [Fig 3.58]. Tattooing has been practiced since ancient times. The body of "Ötzi the Iceman", dated 3300 BC, bears 57 tattoos. Several Egyptian mummies have been found to have tattoos. **Salient features:** (1) **Site** - Tattoos can be found on just about any part of the body, though the *commonest site is front of forearm*. Other common sites are upper and lower limb, back of neck, abdomen, breasts and vulva (prostitutes), penis (homosexuals), buttocks. Even facial tattoos are known. (2) **Method of examination** – Record exact size, shape, design, color, site. Photography is better.

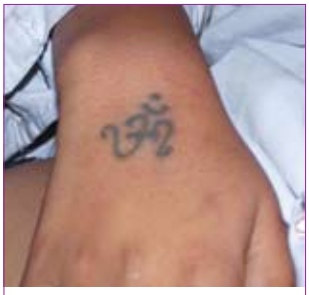


Fig. 3.58: A common tattoo used in India, with name of deity

1. Technique

Coloring matter (dye) is injected deep in the dermis with sharp needles, or an electric vibrator, so that the mark becomes indelible. An inaccurate technique would deposit the pigment in superficial layers of dermis only, which would cause obliteration of the tattoo mark eventually.

2. Dyes

(1) Henna and Mehndi in India (2) **Salts of heavy metals**

Fig. 3.59: Tattoo made with a UV reactive ink. Left - In normal light; Right – under UV light.

- Aluminum (green, violet); barium (white); cadmium (red, orange, yellow); chromium (green); cobalt (blue); copper (blue, green); iron (brown, red, black); lead (yellow, green, white); mercury (red); nickel (black); titanium (white), ultramarine [double silicate of aluminum and sodium; blue] and zinc (yellow, white). **Metal oxides** used are ferrocyanide and ferricyanide (yellow, red, green, blue). (3) **Organic chemicals** - Azo-chemicals (orange, brown, yellow, green, violet) and naphtha-derived chemicals (red). (4) **Homemade or traditional tattoo inks** - Made from pen ink, soot, dirt or blood. (5) **Glow In The Dark Tattoo Ink** - These work on the principle of *phosphorescence* and contain phosphors (a substance that radiates visible light after having absorbed energy earlier). Not approved by the FDA (Food and Drug Administration, US) as phosphors are known carcinogens. Sometimes radioactive materials are also added. Very dangerous to use. (6) **UV (Blacklight) Reactive Tattoo Ink** - Blacklight tattoos will not glow in the dark but glow under UV light [Fig 3.59]. These work on the principle of *fluorescence*; the inks are made of *florescent* material.

3. Permanency

(1) Once imprinted, tattoos remain on the body **almost indefinitely**, until and unless they are removed by specialized procedures. (2) **Rate** at which the tattoos fade depends upon (a)**composition** of dye (b) **depth** up to which the dye is inserted - Dye should be injected to the *right depth*. Dye injected *superficially* gradually fades in some years. Dye injected *too deep* is removed by phagocytes (c) **Site:** (i) Parts protected by clothing retain tattoos much longer than those which are exposed to sunlight (ii) Tattoos on hand disappear early due to constant friction

4. Faded tattoos

Faded tattoos may be made visible by (1) UV lamp (2) Infrared photography (Can also reveal old tattoos superimposed by new tattoos) [please see ch 30, under the heading photography] (3) Removal of epidermis: (i) makes the tattoo very clearly visible on dermis (ii) In **decomposed** or **burnt bodies** – can be done easily (Fig 3.60). (iii) In **fresh bodies** – Apply heat on suspected tattooed area (a) By placing a spirit soaked burning cotton over the area. (i) Examination of lymph nodes nearest to faded tattoo [eg in case of forearm tattoos, axillary LN] – Incise

1. Bistouri

2. Bone nipper

3. Bone rongeur

4(i). Brain knife (sharp point)

4(ii). Brain knife (round point)

5. Brunetti chisel

6. Cartilage knife

7. Catlin knife

8. Chisel

9. Councilman blade saw

10. Director grooved

11. Disecting knife

12. Dissecting scissors

13. Dura stripper

14. Enterotome

15. Hammer

16. Langenbeck Metacarpal Saw

17. Large ampulation knife

18. Large needle with twine

19. Large scissors

20. Metacarpal Saw

21. Myelotome

22. Plastic visor

23. PM40 Knife

24. Postmortem stapler

25. Probe

26. Rib knife

27. Richter Dissecting scissors

28. Scalpel

29(i). Rachiotomy saw

29(ii). Spinal knife

30. Spencer Wells forceps

Fig. 5.1: Usual instruments required for postmortem examination. Please see text for details. Instrument number in figure corresponds with the number in text

8
Thanatology, Death and Its Causes

I. Introduction

A. Thanatology

Thanatology *(Gk* ***Thanatos****, the God of Death) is that branch of science that studies death in all its aspects.* In Greek mythology *Thanatos* is the son of *Nyx* (Night) and *Erebos* (Darkness) and twin of *Hypnos* (sleep) after whom word "hypnotism" is coined [**ch 28**].

B. Forensic Thanatology

Study of death from a ***medico-legal angle*** *(e.g., when did a person die, how did he die).*

II. Stages of Death

Human body consists of about **60 trillion cells**, of which the brain contains **20 billion** (**10 billion nerve cells** in the cerebrum and **10 billion** in cerebellum). Brain stem contains still fewer cells (**2 billion** cells). When the **brain stem cells die, the individual dies**, but many of his body cells may still be alive (e.g. leucocytes may retain ameboid movements, spermatozoa may be motile). These may take some time to die. Thus **death occurs in two stages:** (i) First, when all the cells of the brain stem die. This is the time when the individual dies as a whole. At this time several of his body cells may still be alive (**somatic death**) (ii) Second, when all his body cells have died (**molecular death**).

A. Somatic Death

Somatic death *is the permanent, irreversible death of an organism as a whole.* Historically the concept of when somatic death occurs has kept changing. Traditionally the concept was that it occurs when there is irreversible cessation of heart, lungs and brain [**Bichat's criteria** - please see below]. The concept was gradually changed to **brain death** and eventually to **brain stem death**. With modern technology, bizarre situations can arise. Circulation and respiration of a brain stem dead individual can be maintained artificially. By classical criteria, such an individual would be somatically alive, which is an absurd situation.

Salient features: (1) According to modern concept, somatic death coincides with the death of brain stem. It involves **complete and irreversible stoppage** of vital brain stem functions. (2) *Legally a person is dead after somatic death.* (3) *It is death as a common man understands it.* (4) **Behavior of cells and tissues immediately after somatic death** - Several cells of the body keep functioning **after somatic death** till the onset of molecular death. (i) **Peripheral nerves** die within 5 minutes. (ii) **Skeletal muscles** continue to respond to electrical and mechanical stimuli for up to **2-3 hours** after death (Fig 8.1). (iii) **Bowel** – continue peristaltic movements (iv) **Cilia** - continue their movements (v) **Neurons** - Discharges from gradually dying neurons→focal muscular twitchings (vi) S/c inj of **pilocarpine** or **acetylcholine induces sweating**. (vii) Injection of **atropine** or **adrenaline** in the anterior chamber of the eye causes **dilatation of the pupil**, and injection of **pilocarpine** or **physostigmine** causes constriction. (viii) **WBC** retain their phagocytic movements. [if India ink particles are put in their vicinity, they are phagocytosed] (ix) **Spermatozoa remain motile** in the epididymis. (x) **Anaerobic chemical processes continue** - (a) liver cells continue dehydrating ethyl alcohol [if present] to acetic acid (b) muscle cells continue metabolism. Except for the rare situation of death occurring in a nuclear holocaust, all 60 trillion cells of the body never die at the same time. Even fragmentation of a body by a bomb does not kill all the body cells instantaneously. It has very aptly been remarked, **"we die in bits and pieces!"**

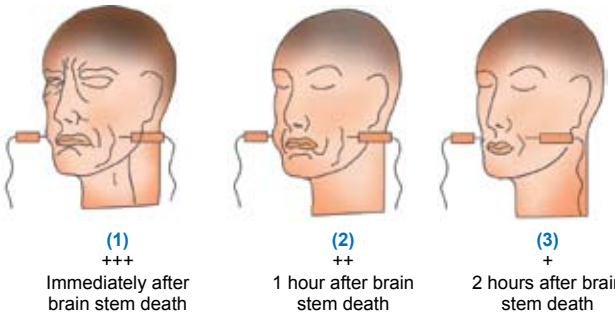


Fig. 8.1: Immediately after somatic death, muscles can be made to contract by electrical stimulation. Here current is applied to a recently dead individual. Note how muscles can contract on application of electrical current, because ATP molecules are still available in individual muscle cells. **(1)** Application of current immediately after death; **(2)** after one hour; **(3)** after two hours. The extent and strength of these contractions can be used to estimate TSD [Fig 8.3].

B. Molecular Death

Molecular death *is the death of all individual cells within the body.* All biochemical (molecular) activity within the cells comes to a stop. It comes **2-3 hours after somatic death** (Fig 8.2).

1. Distinction

Practical importance of the distinction between somatic and molecular death (1) *Legally speaking a person is dead when* ***somatic death*** *has occurred.* A death certificate can be issued and disposal of the body by cremation etc may be done after somatic death. *One does not have to wait for molecular death to occur.* (2) The **organs for transplantation**

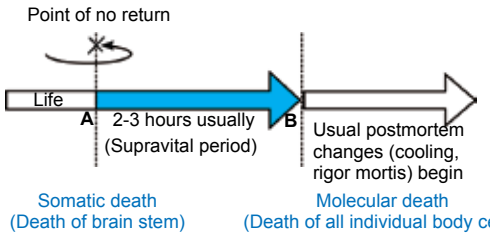


Fig. 8.2: A simplified conceptual view of somatic and molecular death. When brain stem dies, the patient cannot be revived by any means. He is said to have reached the "point of no return" (A). This is the brain stem death. 2-3 hours later, all individual cells die (B). This is molecular death. The period between A and B is known as the supravital period, and is best for harvesting of organs for transplantation. Usual postmortem changes, e.g. cooling of the body, rigor mortis start from B onwards.

with ferrous iron from Hb to form black **ferrous sulfide**]. (2) **Marbling** - *Marbling is greenish brown staining of superficial veins* [Fig 9.12, Fig 9.16]. (i) **Mechanism** – Putrefactive bacteria spread most easily in fluid medium, and thus tend to colonize the venous system. Hemolysis of RBCs releases Hb, which combines with H_2S released by bacteria to form sulfhemoglobin, which stains the walls of veins [esp over neck, shoulders, chest, roots of limbs, sides of abdomen and thighs]. This gives a **marbled appearance** [linear branching pattern, resembling branches of a tree] to the body (ii) **Time**→36-48 h. (3) **Skin:** (i) Macerated like appearance; Reddish-green [36-48 h]. (ii) In 3-4 days dark green or almost black. May give the appearance of gangrene, but can be differentiated easily [Table 5]. (4) **Poisoning:** (i) *Antimony poisoning→orange* [**ch 36**].

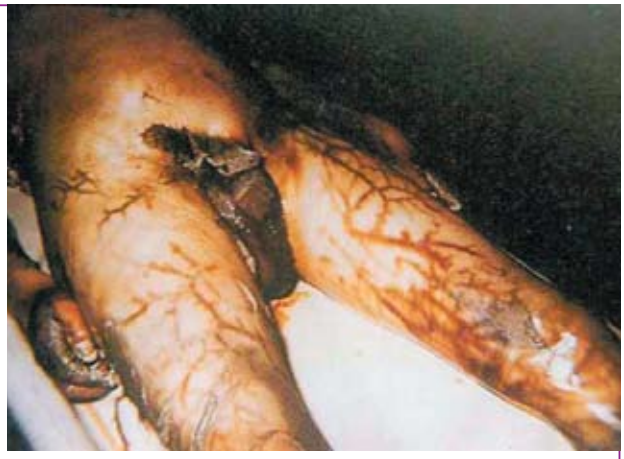

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Fig. 9.12: Marbling.

Table 5: Differences between Putrefaction and gangrene

S.No.	Criteria	Putrefaction	Gangrene
1.	Pus	Not formed	Usually formed
2.	Phenomenon	Postmortem	Antemortem
3.	Inflammation and repair	-	+
4.	Line of demarcation between living and dead tissue	None. All tissue is dead	Present
5.	Area	All over	Localized

vii. Development of foul smelling compounds

(A) **Chemical processes** in putrefaction are those of **reduction**. Proteins and carbohydrates are reduced to simpler compounds (amino acids, CH_4, CO, CO_2, H_2, H_2S, mercaptans, NH_3 and PH_3). Many of these gases are inflammable, and they can be ignited [Fig 9.13] [these gases may give rise to **preternatural combustion** (Ch 14)]. (b) **Foul smell** – Foul smell from the cadaver is mainly due to decarboxylation of the amino acids, ornithine and lysine, which yields carbon dioxide and the foul-smelling ptomaines **putrescine** [$NH_2(CH_2)_4NH_2$] and **cadaverine** [$NH_2(CH_2)_5NH_2$]. This breakdown occurs also in living people under certain circumstances as in *bad breath* and *bacterial vaginosis*. Cadaver dogs [**ch 1**] discover hidden bodies by smelling these ptomaines [for more on ptomaines please see

Fig. 9.13: Demonstration of flammability of putrefactive gases. In this body who had been dead for three days, putrefactive gases had collected in scrotum. A small syringe needle was inserted in the scrotum and the other end ignited. The clear blue flame is due to methane, hydrogen and H_2S.

ch 39]. (c) **Effects of gases:** (1) **Blisters** – (i) Form because of collection of gases between epidermis and dermis [**postmortem blisters**, Fig 9.14]. Fluids and liquid fat may also be pushed in the blisters (ii) Must be differentiated from antemortem blisters [**ch 14**]. (iii) Blisters form first on areas where tissues contain more plasma due to hypostatic edema [back, lower surfaces of trunk, thighs (Fig 9.15)] (iv) **Time of development**: Summers-

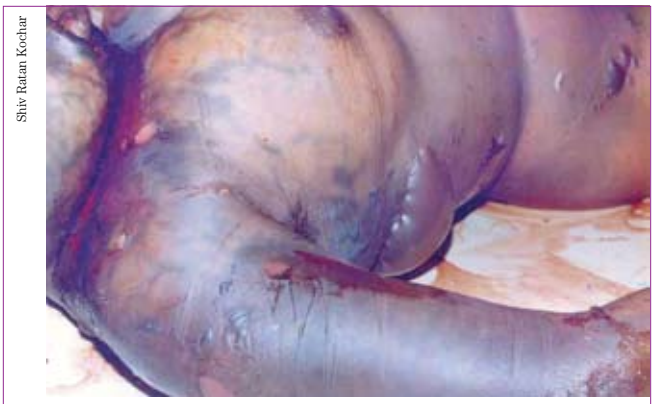
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Fig. 9.14: PM blister on chest.

Fig. 9.15: Several small PM blister on lower trunk can be seen. A large blister has been punctured and spread on the table by the pathologist to demonstrate its size. Greenish discoloration may be seen on lower trunk.

body at the time of discharge. (ii) In all contact wounds, soot, powder, carbon monoxide [carboxyhemoglobin, COHb], and vaporized metals from the bullet, primer, and cartridge case are deposited in and along the wound tract. Their concentration ↓es with ↑ing depth. (2) **Classification** - Contact wounds may be (i) hard (ii) loose (iii) angled or (iv) incomplete [a variation of angled].

i. Hard contact

(1) **Characteristics:** (i) Muzzle of the weapon is jammed "hard" against the skin, indenting it, so that the skin envelops the muzzle; *contact is so hard that gases cannot escape out* (ii) immediate edges of the entrance are seared by the hot gases of combustion and blackened by the soot (iii) This soot [along with unburnt powder] is embedded in the seared skin and cannot be completely removed either by washing or by vigorous scrubbing of the wound [Fig 13.61]. *This is thus a special case, where burnt powder [smoke] also produces tattooing.*

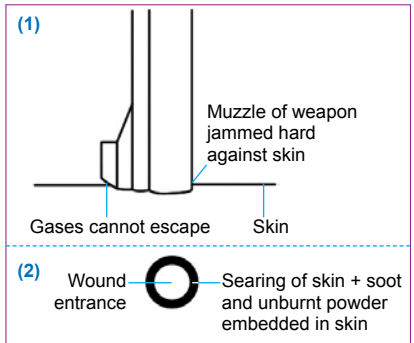

Fig. 13.61: Hard contact wound with blackened seared margins. (1) Position of the gun (2) wound produced by it.

ii. Loose contact

(1) **Characteristics:** (i) The muzzle, while in complete contact with the skin, is held lightly against it. (ii) Gas preceding the bullet, as well as the bullet itself, indents the skin, creating a *temporary gap* between the skin and the muzzle through which gas can escape. This *temporary gap cannot be produced* in hard contact wounds. (iii) Soot carried by the gas is deposited in a zone around the entrance (iv) This soot can be easily wiped away. (v) A few unburnt grains of powder may also escape out this gap and be deposited on the skin around the zone of soot [Fig 13.62].

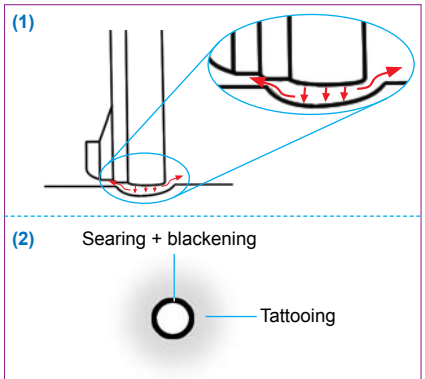

Fig. 13.62: loose contact wound with soot deposited in zone around entrance. (1) Position of the gun. Note that there is some gap through which gases can escape. (2) wound produced by it. Please compare with Fig 13.61.

iii. Angled contact

(1) **Characteristics:** (i) The *barrel is held at an acute angle to the skin* so that the complete circumference of the muzzle is not in contact with it. (ii) Gas and soot escaping from the gap, where contact is not complete, radiate outward from the muzzle, producing an eccentrically arranged pattern of soot. (iii) The soot is arranged in two different zones. (a) *More noticeable zone* – [often the only one seen] is a smaller blackened seared area of skin or cloth having a pear, circular, or oval configuration [Fig 13.63] (b) *Less noticeable zone* - is a larger fan-shaped zone of light-gray soot that radiates outward from the gap. On the skin (the less noticeable zone with respect to soot) this light zone is usually washed away, or obscured by bleeding or removed in cleaning the wound for examination. (iv) A few unburnt grains of powder may also be deposited in these zones. (v) The entrance wound is normally present at the base of the seared blackened zone. All or at least the majority of the seared blackened zone will be on the opposite site of where the muzzle was in contact with skin, and thus "points" the way the gun was directed. (vi) As the angle between the barrel and the skin ↑es, i.e., the barrel moves toward a perpendicular position to the skin, the entrance hole will be found more toward the center of the zone. (vii) If the angle between the barrel and the skin ↓es, the gap between the muzzle and skin becomes larger, and more material can escape through the gap. (viii) At some point, the gap becomes sufficiently large that unburnt grains of powder escaping through the gap will skim over the zone of seared skin, fanning out from the entrance, impacting distal to the entrance wound in a fan shaped pattern of powder tattooing.

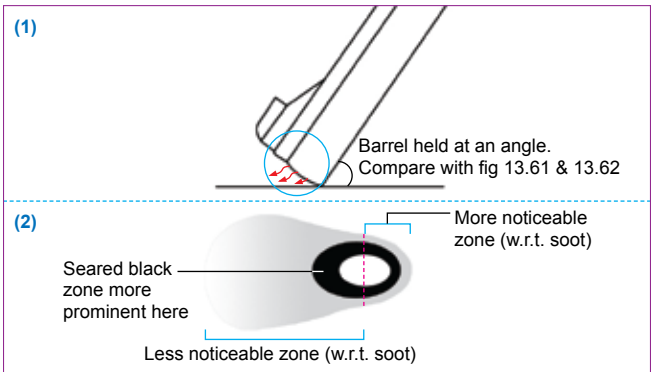

Fig. 13.63: Angled-contact wound (1) Position of the gun. (2) While more prominent seared zone is seen in the direction of the gun, more prominent blackening is seen in the opposite direction.

iv. Incomplete contact

(1) **Characteristics:** (i) This is a variation of angled-contact wound. (ii) muzzle of the weapon is held against the skin, but because the body surface is not completely flat, there is a gap between the muzzle and the skin [Fig 13.64]. (iii) *Difference with angled contact* – In angled contact, the

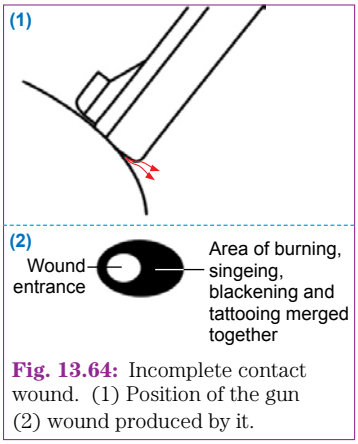

Fig. 13.64: Incomplete contact wound. (1) Position of the gun (2) wound produced by it.

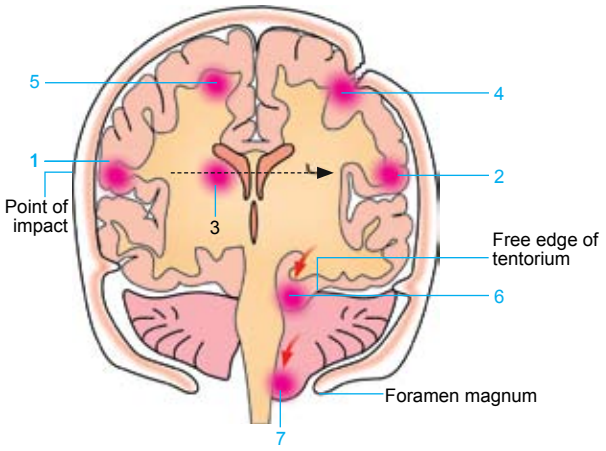

Fig. 17.18: Six types of contusions. Black dotted arrow depicts line of impact. 1. Coup contusion; 2. Counter coup contusion; 3. Intermediary coup contusion; 4. Fracture contusion (fracture occurring away from point of impact); 5. Gliding contusion [at junction of gray and white matter]; 6. Herniation contusion (temporal lobe herniating through free edge of tentorium. This also produces uncal grooving); 7. Herniation contusion (cerebellum herniating through foramen magnum (see text for details).

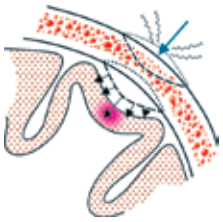

Fig. 17.19: Mechanism of production of coup contusions

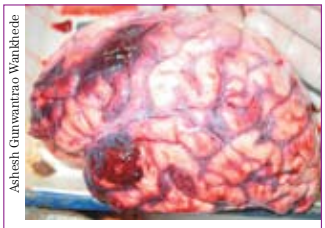

Fig. 17.20: Coup contusions of brain.

contusions. (5) Generally **more severe** than coup lesions. (6) Both coup and countercoup contusions are present over the **crests of gyri**.

Table 3: Coup and Countercoup Contusions

(1) A **blow to the head** produces **coup contusions** (no or minor countercoup contusions) – **head is accelerated**. *Impact occurs first -> followed by motion*. In general, **coup lesions** predominate if head is **accelerated** after impact.

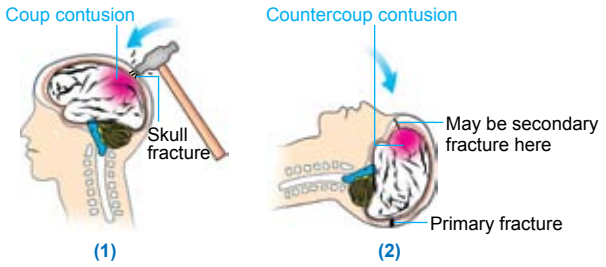

Fig. 17.21: (1) Coup contusions are caused when force strikes a fixed head (2) Countercoup contusions are caused when a moving head strikes a hard surface (see text for details)

(2) A **fall on the head** produces **countercoup contusions** (no or minor coup contusions) - **head is decelerated**. *Motion occurs first -> followed by impact*. In general, **contrecoup lesions** predominate if head is **decelerated** after impact.

Table 4: Theories of Countercoup Injuries

Following **theories** have been advocated to explain **countercoup injuries**.

(1) **Positive pressure theory** - Advocated by **Lindenberg**. Its salient features are as follows (Fig 17.22).

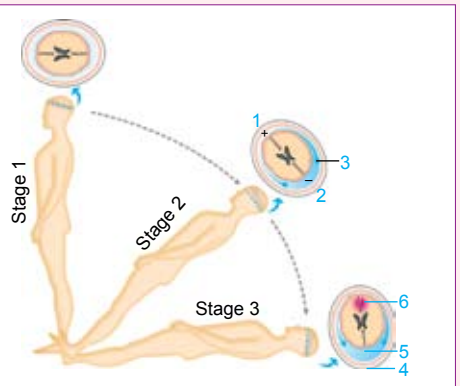

Fig. 17.22: Positive pressure theory of **Lindenberg**. (1) Trailing skull surface; (2) Leading skull surface; (3) CSF shifting towards leading skull surface; (4) Point of impact; (5) More CSF collecting here protects brain; (6) Counter coup contusion. See text for details

(i) At the beginning of fall, skull is accelerated. **Brain lags behind due to inertia** (stage 1 in Fig 17.22).

(ii) As victim continues to fall, brain lags far and far behind. This creates a **negative pressure** at the ***leading skull surface*** (the surface that is going to hit the surface), and a **positive pressure** at the ***trailing skull surface*** (stage 2 in figure).

(iii) As CSF continues to move towards the leading skull surface, very little CSF remains at the trailing skull surface, by the time impact occurs. Thus there is **no cushioning effect** of CSF there. Furthermore a **very great +ve pressure** has been built up at the trailing skin surface (stage 3 in Fig 17.22). Both factors combine to produce **countercoup lesions**.

(2) **Negative pressure theory** - Advocated by **Russel**. Also known as the **cavitation theory**. Russel agrees with the first three stages described by Lindenberg, but asserts that countercoup lesions do not occur at the time of impact, but a fraction of a second after. His salient features are:

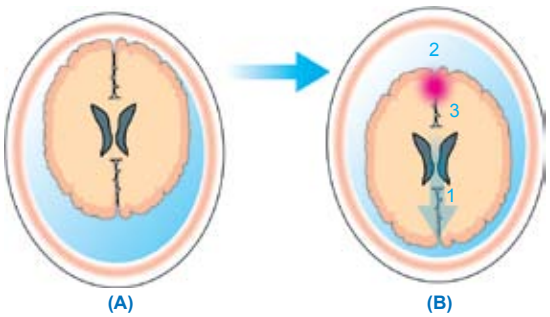

Fig. 17.23: Negative pressure theory of **Russel**. **(A)** Depicts stage 3 of fall as described by Lindenberg. According to Lindenberg, countercoup injury to brain occurs at this stage itself due to +ve pressure [Fig 17.22], but according to Russel, it occurs a fraction of a second later **(B)**. (1) Brain continues to move, while skull stops suddenly, producing -ve pressure at trailing skull surface; (2) Tensile stress produced in brain by -ve pressure; (3) Counter coup contusion produced by this tensile stress. Pl see text for details.

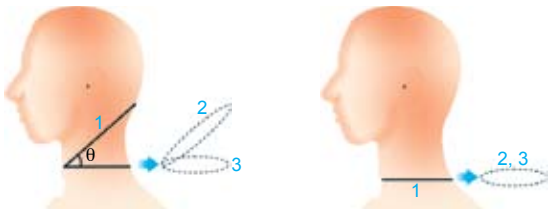

Fig. 19.4: (A) Hanging **(B)** Strangulation. (1) Position of ligature. If circumference of the ligature (2) is MORE than the circumference of the neck (3), the ligature must have been oblique (as in hanging). If it is SAME, the ligature must have been horizontal (as in strangulation)

the deepest groove would be at the back (with neck tilted backwards). (d) Knot and small adjacent lengths of ligature may be stretched away from the body, and may not even be in contact with the skin. Ligature mark will be absent in this area and impression from knot *not* found (13) **Padding** – a piece of towel may sometimes be found between the skin and ligature [placed by victim in order to reduce pain]. Found usually in accidental hanging, especially sexual asphyxias [please see below]. (14) **spontaneous breakage** - Sometimes the ligature may break spontaneously and body may be found lying on the ground. In such cases, it becomes imperative to determine if the death was due to hanging or strangulation. It can be done by (a) examining the other end of ligature which must be found tied to the suspension point (eg fan, tree etc)(b) Broken ends of ligature must coincide (15) **Knot** – (a) Record the location and type of knot. (b) **Location** – whether at occiput, below the chin, below either ear (subaural) or any other specific location. In all cases, it is always *above* the rest of the ligature. This produces an inverted 'V' shaped ligature mark, the apex of 'V' corresponding with the site of the knot. (c) **Type** - It may be fixed or running (Fig 19.5). Granny, reef [Syn, square], or other special types of knots are exceedingly rare and may sometimes give away the profession, predilection or hobby of the person, eg a sailor may use a reef knot; a scout, square knot etc. A complicated knot outside the victim's knowledge and experience may indicate the involvement of another person.

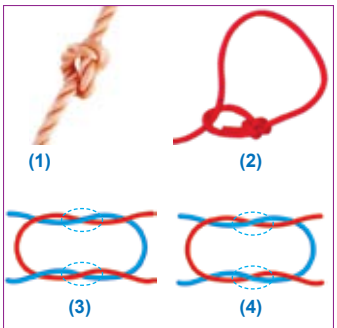

Fig. 19.5: (1) Fixed knot and **(2)** slip (running) knot. Less common are **(3)** Granny knot and **(4)** Reef (syn, square) knot. In granny knot, the crossings are opposite, while in reef knot they are same. Pl. see dotted circles.

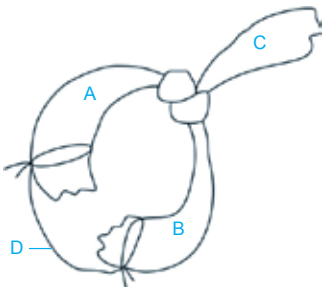

Fig. 19.6: Method of securing the knot. Limbs A and B form the actual noose around the neck. C is the free end of the ligature. If A and B are not secured with a string D, it would be difficult to demonstrate in court that A and B indeed formed the noose. Any two of the three limbs would appear to consist of original noose.

(d) **Removal of ligature** – Knot should never be opened. (i) *Photography* – First step is to take photographs [at close range] before removal of ligature (ii) The ligature should now be cut *opposite the knot*, and the two cut ends secured with a string (**Fig 19.6, Fig 19.7**) [for more on ***Forensic knot analysis***, please see **chapter 30** – Forensic Science Laboratory].

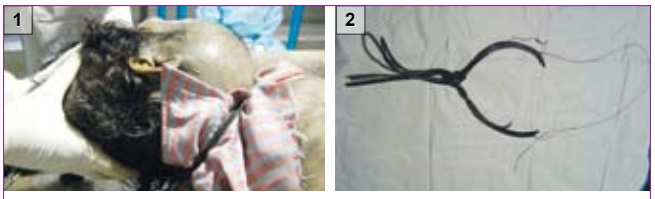

Fig. 19.7: Method of securing the knot. **(1)** This man - an electrician by profession - had hanged himself with an electric wire, wound twice round the neck. Portions of his shirt had been caught between the ligature and the neck. The knot is towards the lower right side. **(2)** A string was tied to intact ligature opposite the knot, and then the ligature severed. The entire loop was removed via top of the head.

b. Ligature mark

Ligature mark *is a type of pressure abrasion due to continued pressure by ligature on the neck.* **Salient features:** (1) It is seen both in hanging and strangulation [Table 3]. Strangulation mark is described later. (2) **Appearance** - It is usually seen as a *furrow or groove in the tissue which is soft and pale initially, but as the skin dries up, becomes hard (parchment like) and dark brown*. It runs from midpoint of the neck upwards, outwards and backwards [from either side of the neck] to reach behind the neck where it is deficient [Fig 19.8] (3) *It is the* ***most important*** *and* ***specific*** *sign of death from hanging*. (4) Following things must be noted (i) **Its position** - Usually it is at the front of neck, but rarely at the back of neck also [Fig 19.9]. (ii) **If in front its**

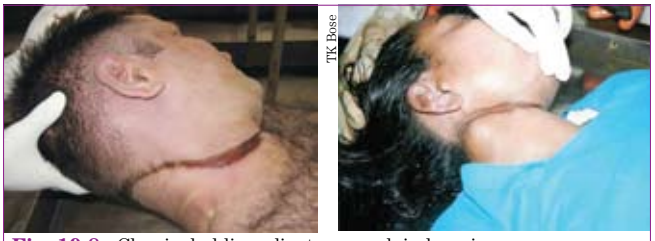

Fig. 19.8: Classical oblique ligature mark in hanging.

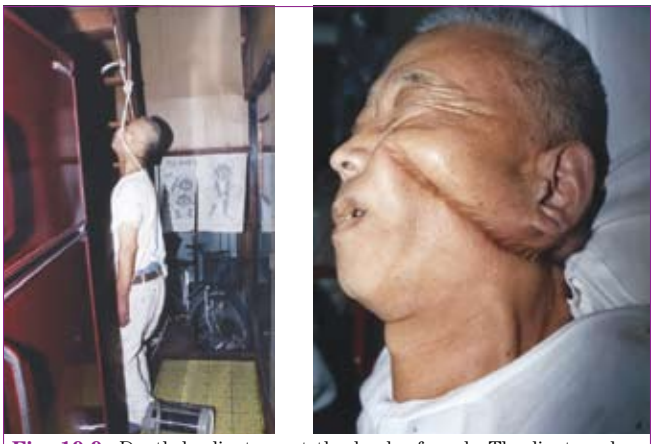

Fig. 19.9: Death by ligature at the back of neck. The ligature has slipped up and has formed a patterned abrasion on the left cheek. Ligature mark at the face is an exception rather than the rule.

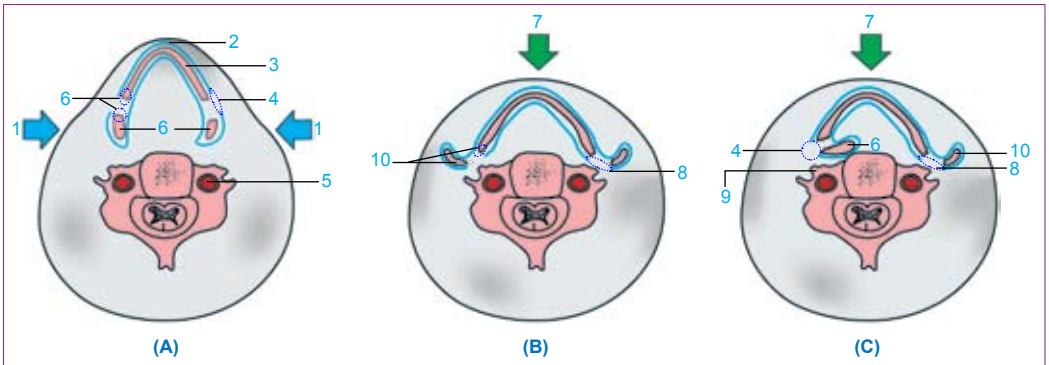

Fig. 19.36: Types of hyoid fractures. **(A) Inward compression fractures** [*seen in throttling*] **(B) Outward compression fractures** [*seen in hanging*] **(C)** One cornu **fractured inwards** and the **other outwards** [*seen in hanging, when one end is caught up against a bony ridge or sides of the vertebrae*].(1) Inward compressive force as occurs in throttling (2) periosteum over the hyoid (3) hyoid (4) periosteum torn on the outside (5) vertebral artery (6) Inward fractures of greater cornu of the hyoid (7) Backward force on hyoid as occurs in hanging due to ligature (8) periosteum torn on the inside (9) Bony ridge of the vertebra, in which greater cornu may get caught causing inward fracture (10) Outward fractures of greater cornu of the hyoid

b. Outward (Anteroposterior) compression fractures

(1) **Outward fracture** - In case of **hanging**, as ligature presses on the neck, the hyoid is forced directly backwards →↑es divergence of greater horns→Outward displacement of posterior fragment occurs, *with rupture of periosteum on the inside* [Fig 19.36(b)]. (i) Outward fracture may also occur in (a) **Ligature strangulation** - especially if the ligature was pulled backwards by the assailant standing at the back. This causes the hyoid to push back against the vertebra causing outward displacement of lateral ends. (b) **run over motor vehicle accidents** - multiple fractures of other structures are also found (c) **Blows on front of neck**. In all cases mechanism is same - hyoid is pushed backwards against the vertebra. (ii) Rarely one greater horn is fractured inwards and the other outwards [Fig 19.36(c)]. Since hyoid moves backwards and also sideways, one end may get caught up against a bony ridge or sides of the vertebrae. As the hyoid is pushed further back, this end would fracture inwards, while the other end would fracture outwards as usual. (2) **Nature of fracture** - (i) Like inward compression fractures, it may be unilateral or bilateral, and like it may occur at 2 places. Similarly complete detachment of smaller fragment from the hyoid may occur if the compression is severe. The smaller fragment may seen to be *lying laterally* to the rest of the bone.

2. According to Mechanism of fracture

a. Direct pressure fractures

When pressure is exerted directly on the hyoid. These are most commonly seen in hanging and strangulation.

b. Avulsion fractures

(1) There is no direct pressure on the hyoid. (2) Instead the fracture is caused by *muscular overactivity*. (3) **Mechanism** - in some forms of violence, hyoid is drawn up and held rigid by the powerful muscles attached to its upper and anterior surface→If there is associated violent downward or lateral movement of the thyroid cartilage or pressure between hyoid and thyroid, traction occurs on hyoid through thyrohyoid ligament→Avulsion fracture of hyoid (4) These are also known as "**tug**" or "**traction**" fractures. (5) Avulsion fractures can be both *inward compression* or *outward compression*, depending upon the type of muscular overactivity. 6.It is very difficult to ascertain at the time of PM if fractures were caused by direct pressure of they were avulsion fractures. But if fractures are present in absence of soft tissue injuries, they are likely to be avulsion fractures.

C. Demonstration of fractures

(1) **Palpatory method:** (i) Hyoid body is grasped in one hand (ii) distal fragment [tip] is held between the finger and thumb of other hand (iii) a gentle attempt is made of bend the distal fragment both on the inside and outside (iv) In case of **inward compression fracture**, it can be easily bent in an inward direction [because of torn periosteum outside]; but outward movement is limited to the normal position only [because of intact periosteum on inside]. Exactly the opposite happens in **anteroposterior compression fractures**. In cases of advanced putrefaction and maceration, when the periosteum is completely destroyed, the tip can be easily displaced on either side→becomes difficult to opine if the tip was fractured inwards or outwards. (2) **Toluidine blue with stereomicroscopy** - 1% Toluidine blue sol^{n} is applied to suspect areas, left for 15 s and cleaned off with pure water. Examine bone with a stereomicroscope [5-18 times magnification]. fractured sites would appear bluish [toluidine blue is also used to detect microinjuries in sexual assaults – **ch 25**]. A very effective method of discovering hyoid fracture. (3) **X-ray and CT** - *before start of autopsy are the best and most certain* method to ascertain fractures. If for some reason, could not be done before start of autopsy, the thyrohyoid complex should be carefully dissected out and x-rayed separately. The x-ray or CT plates should be preserved as evidence.

VI. Dissection of neck in hanging and strangulation cases

Please see **ch 5.**

VII. Suffocation

Suffocation *is exclusion of air from lungs from any means other than ligature [Fig 19.37].*

A. Environmental suffocation

Environmental suffocation *is deprivation of oxygen due to lack of oxygen in the environment*. **Salient features:** (1) There may be deficiency of O_2 in the environment, or it may be replaced by an inert gas as N_2. Replacement of O_2 by a poisonous gas such as Cl_2 or H_2S would not cause death primarily by asphyxia, but by poisonous effects. Mixing of O_2 with the poisonous gas would still cause death, while mixing of O_2 with an inert gas would prevent it. (2) Conc of O_2 in air is 21%. Its fall to 16% or less is dangerous. With 5% conc consciousness is lost rapidly and death occurs within a few minutes.

1. Causes

(1) **During play** - children may get locked in large boxes, trunks, or old disused refrigerators (2) **Glue sniffing** [inhalant abuse]- [**ch 46**]. Smothering may also be involved [please

Fig. 19.41: Postural asphyxia in a drunk person who collapsed on the backseat of his car. He had been drinking, waiting for a friend to arrive. White arrows depict weight of abdominal organs acting upon lungs preventing their expansion.

Fig. 19.42: Elephant man who died of positional asphyxia due to excessive weight of his head.

He had an extremely heavy and deformed head, and was exhibited as a human curiosity. Because of excessive weight of his head [Normal weight of head is 4.5-5 kg, constituting approx 8% of the body weight], he had to sleep sitting up. On 11 April 1890, aged 27, he decided to sleep lying down, to "be like other people". The weight of his head asphyxiated him. **Sir Frederick Treves**, who conducted an autopsy on him found his neck to be dislocated. (2) **Circumstances favoring positional asphyxia** - Victim is incapacitated due to some reason so that he cannot voluntarily make efforts to respire (i) strong intoxication (ii) Unconsciousness due to trauma, stroke or other natural disease.

1. PM Findings

(1) Same as that in traumatic asphyxia, except fractures and blunt injuries.

2. MLI

(1) Almost always accidental. But may be homicidal, if assailant "doubles up" victims body in a jack-knife position.

VIII. Drowning

Drowning *is a form of asphyxia caused by aspiration of fluid into air-passages, caused by complete or partial submersion in water or other fluid*. **Salient features:** (1) Sp gr of body is **1.08.** Other sp gr in increasing order are (i) **Fat**-0.92 (ii) **Brain**-1.04 (iii) **Soft organs**-1.05 (iv) **Muscle**-1.08 (v) **Bone**-2.01. Natural tendency of the body is thus to sink down. (2) Complete submersion is *not necessary*; submersion of only nose and mouth can cause drowning, as is seen in alcoholics passing out in shallow streams and getting drowned. (3) **Current concepts** - According to current thinking the term "drowning" should include *both fatal and nonfatal drowning*. Accordingly a new definition of drowning has been suggested. It states, "***Drowning*** *is the process of experiencing respiratory impairment from submersion/immersion in liquid*." The definition includes cases of drowning in all kinds of liquid, *except body fluids* [amniotic fluid, milk, saliva, vomitus]

A. Epidemiology

(1) **Incidence** - The World Health Organization (WHO) estimates the annual worldwide incidence of death by drowning to be about 400,000.

(2) **Global mortality rate** - 6.8 per 100,000 person-years. This places drowning as the second leading cause of death from unintentional injury, after road traffic injuries. (3) **Age** - Over half of global mortality occurs among children <15 y. (4) **Countries** - 97% of all drowning deaths occur in low and middle-income countries.

B. The Stages and Mechanism of Drowning

Dogs have been experimentally drowned to study the **stages of drowning**. Before drowning, dogs were prepared as follows: (i) Dog's four limbs were tied to a **wooden board**, and a weight of lead was fixed to this board (ii) A **cannula** was introduced into the femoral artery to measure BP and heart beats (iii) A **pneumograph** was attached to the epigastrium to record the respiratory movements. The wooden board (with dog tied to it) was then lowered into a tub filled with water, till the dog was submerged completely in water. On the basis of subsequent observations, drowning was divided into 5 stages: (1) **The stage of surprise** – *Lasted for 5-10 seconds*. Dog inspired once or twice, but was otherwise inactive (2) **The first stage of respiratory arrest** – *Lasted for 1 minute*. The dog was violently agitated. Struggled against its bonds, and tried to reach surface. It shut its mouth and did not breathe. (3) **The stage of deep respiration** – *Lasted for 1 minute*. Dog inspired deeply (when unable to stop breathing any longer). White foam formed, which rose to the surface. General agitation ceased. Mouth and eyes were open. A few swallowing movements were observed. (4) **The second stage of respiratory arrest** - *Lasted for 1 minute*. Respiration stopped. Thoracic movements stopped completely. No corneal reflex. Pupils markedly dilated. (5) **The stage of terminal gasps**- *Lasted for 30 seconds*. Respiration restored again but in the form of 3-4 terminal gasps. *Acute lung injury and hypoxemia develops because of disruption of surfactant*. Lung dysfunction leads to alveolar collapse and acute respiratory distress syndrome (**ARDS**). Fibrillary contractions were seen in the lip and jaw muscles. The entire process of drowning took 3½ to 4 minutes.

C. Classification

1. Wet Drowning

Wet drowning *[syn, classical drowning, primary drowning] involves inhalation of water into lungs*. **Salient features:** (1) Enormous quantities of liquid is inhaled into lungs (2) The victim suffers from severe chest pain. (3) **Fatal period** - Death occurs within minutes of submersion. (4) **Cause of death** – (i) Cardiac arrest (ii) ventricular fibrillation (iii) Hyperkalemia

2. Dry Drowning

In ***dry drowning****, there is no significant presence of liquid in the lungs*. **Salient features:** (1) **Mechanism:** (i) *Laryngospasm* - Just a few drops of liquids enter the larynx→elicit a violent laryngospasm→death due to asphyxia without entry of water in lungs. (ii) *Absorption of liquid in circulation* – victim brought out of water→resuscitated→water absorbed in hypertonic plasma (2) **Incidence** - About 10-15% of all drownings. (3) **Victimology** – Commonly seen in (i) children (ii) adults under the influence of alcohol, sedatives or

20 Starvation

I. Introduction

Starvation *is a severe reduction in nutrient, vitamin and energy intake that occurs either from withholding of food or from administration of unsuitable food.* **Salient features:** (1) **Types:** (i) **Acute starvation** - withholding of food is *sudden and complete.* (ii) **Chronic starvation** - withholding of food is *gradual*. Chronic malnutrition, as occurs in poor, deprived sections of society. (2) **Inanition** - *Symptoms and effects of starvation.*

II. Causes

A. According to etiology

1. Circumstantial causes

(1) Poverty [most common cause in India] (2) Fasting (3) **Accidental:** (i) earthquakes (ii) famine [failure of crops, overpopulation, war] (iii) landslides (iv) lost in desert or jungle (v) marooned on island (vi) shipwrecks (vii) trapped in mines and pits etc.

2. Medical causes

(1) Ankylosis of jaw (2) Alcohol and drug addicts [partial starvation only. Food is ignored due to overwhelming desire for drug. In alcoholics calories are supplied by alcohol, so no food intake → protein malnutrition] (3) Anorexia nervosa (4) Bulimia nervosa (5) cancer and stricture of esophagus (6) Coma (7) Diabetes mellitus (8) Digestive diseases (9) Mental illness [Major depressive disorder, paranoid schizophrenia, senile dementia].

3. Miscellaneous

(1) *Eccentrics* may refuse to eat food for no reason. (2) *Ignorance, witchcraft etc* – parents either do not provide food, or do not provide food of right kind.

B. According to manner

1. Accidental starvation

Same as mentioned above [circumstantial causes].

2. Suicidal starvation

(i) **Fasting** - (a) political reasons [fast unto death] (b) purely exhibition. (ii) **Mentally ill and hysterical persons** - often do not take food.

3. Homicidal starvation

Withholding of food from unwanted children, step children, illegitimate children, child abuse, elderly people, feeble minded, jail inmates etc.

III. Symptoms and signs

A. Acute Starvation

Starvation is an acute severe form of primary PEU [Protein-Energy Undernutrition].

1. Clinical

a. 30-48 hours

(1) Feeling of hunger (2) Pain in epigastrium, which is relieved by pressure.

b. 4-5 days

(1) **General:** (i) *Temp* – subnormal (ii) *Voice* – weak, whispering (iii) *Absorption of s/c fat* and *emaciation* – (a) *Cheeks* - sunken (b) Eyes – sunken, glistening. Pupils dilated (c) Bony prominences - become visible (d) Chest – ribs prominent, concavities in intercostal spaces, [Fig 20.1] supraclavicular fossae sunken (e) *Abdomen* - concave and scaphoid [boat shaped] [Fig 20.1] (f) *Limbs* – thin, flaccid, loss of muscular power (g) Muscular weakness – progressive, severe (h) Loss of wt – Marked. (iv) Odor – offensive, especially towards death. (2) **Dermatologic:** (i) *Skin* - dry, inelastic, pigmented, rough, thin, wrinkled. Shows follicular hyperkeratosis and trophic skin changes (ii) *Hair and nails* – brittle, dry, lusterless, hair loss. (3) **CVS:**

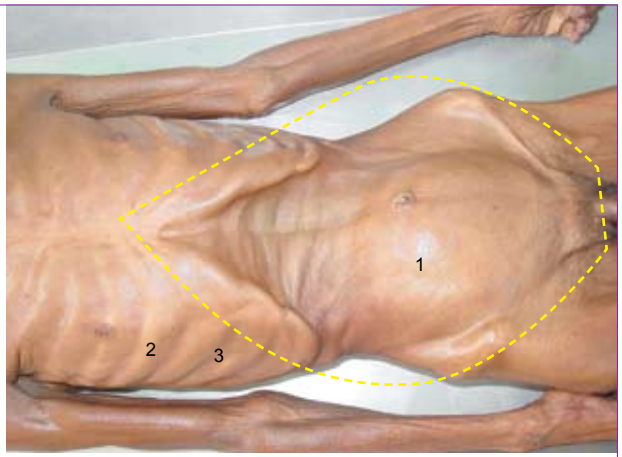

Fig. 20.1: PM appearances in starvation. Please note (1) scaphoid abdomen (2) prominent ribs and (3) concavities within intercostal spaces.

24 Virginity, Pregnancy and Delivery

I. Virginity

A. Definitions

(1) *A **virgin** (virgo intacta) [Latin virgo, "sexually inexperienced woman"] is a female who has not experienced sexual intercourse*. (2) **Defloration** [Latin *de*, away; *flore*, flower. A poetic likening of the rupture of hymen by sexual intercourse to the plucking of flowers] means loss of virginity *with associated rupture of hymen*. Such a woman is called a **deflorate woman**. (3) Loss of virginity *without associated rupture of hymen* results not in a deflorate woman, but a *false virgin*. (4) ***Apta viro*** [please *aptae viris*] is a woman who is "fit for a husband" or "a woman who has reached marriageable years".

B. Normal Female Genital Anatomy

1. External

The **labia majora** (1) [L. *labia*, lip] [Fig 24.1] are two elongated folds of skin projecting downwards and backwards from **mons pubis** (2) [Latin, "pubic mound". Also known as **mons veneris** ["mound of Venus"] or simply the **mons**]. It is a pad of fat lying in front of pubis. They meet in front at **anterior commissure** (3) and at the back at **posterior commissure** (4) in front of the **anus** (5) In a virgin the labia majora are thick, firm, elastic and rounded and lie in apposition so as to completely close the **vaginal orifice** (6). The **labia minora** (8) are about 4 cm long; they are two soft, small, thin, pink and sensitive folds just within the labia majora. The lower portions of labia minora fuse in the midline and form a fold called **fourchette** (9) [Fr, "little fork"]. The depression between fourchette and the vaginal orifice is called **fossa navicularis** (10) [L *fossa*, depression; *navicula*, boat; a boat shaped depression]. **Clitoris** (11) [Gk, *kleis*, key; in reference to it being key to female genitals] is a small button-like organ located near the anterior junction of the labia minora, above the **urethral opening** (12) and vagina. It is covered with a fold of skin that surrounds and protects it [**clitoral prepuce**, clitoral hood, or *preputium clitoridis*). It develops as part of the labia minora and is homologous with the foreskin (also called prepuce) in male genitals. The **frenulum** (13) [also known as the *Crus glandis clitoridis*] is a small fold of tissue on the under surface of clitoris, created by the two medial parts of the labia minora. Urethral opening is 2.5 cm behind the clitoris and immediately in front of the vaginal orifice. **Vestibule** (14) is a narrow triangular area which extends from the clitoris above to the anterior margin of the hymen below, and laterally to the labia minora.

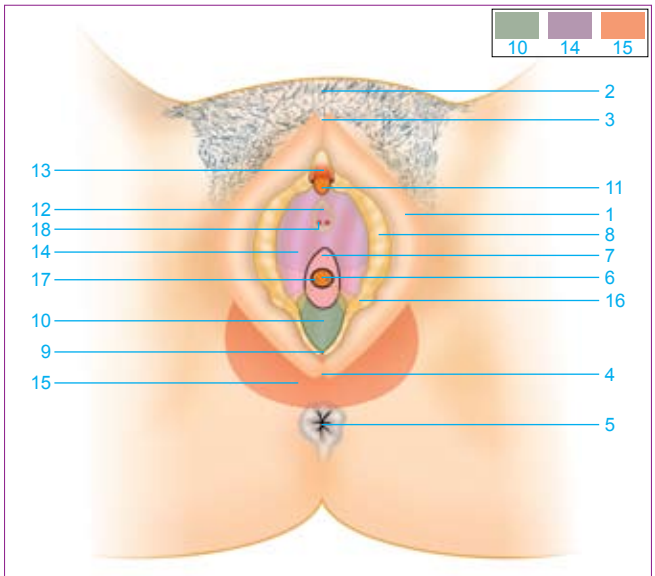

Fig. 24.1: Normal female genital anatomy (External). Please correlate nos. with text.

Memory Aid 1

F ossa navicularis is in F ront of F ourchette; V estibule is V entral to V agina.

It usually remains concealed by the labia. **Vulva** includes mons veneris [which forms its anterior portion], labia major, labia minora, clitoris, vestibule, hymen and urethral opening. The **perineum** (15) is a wedge shaped area between the lower end of the posterior wall of vagina and the anterior anal wall. The cervical canal is nearly at right angles to the vagina when bladder and rectum are empty. **Bartholin's glands** (16) [named after the Danish anatomist **Caspar Bartholin** (1655–1738) who first described them; syn **Greater vestibular glands**] are a pair of small pea-sized glands that lie just next to the lower part of the entrance to the vagina. Unless diseased or infected, they cannot be seen or felt. Each gland makes a small amount of mucus-like fluid, which drains down **Bartholin's duct**, about 2 cm long. It comes out through a small openings (17) towards the lower part of the entrance to the vagina. The fluid helps to keep the entrance to the vagina moist. **Skene's glands** (syn **female prostate**, **lesser vestibular glands**, **paraurethral glands**) are located on the upper wall of the vagina, around the lower end of the urethra. They drain into the urethra and near the **urethral opening** (18). They are the source of female ejaculation. Just as in the male, they are the principal source of PSA.

a. Hymen

***Hymen** [Fig 24.1, (7)] [Gk Hymenaios, God of marriage] is a fold of mucus membrane about 1 mm thick, situated at the vaginal outlet*. **Salient features:** (1) The average adult hymen

27 Infant Deaths Including Battered Baby Syndrome

I. Introduction

A. Infanticide

Infanticide *is unlawful destruction of a child under the age of one year by anyone*. Infanticide *does not include* the death of fetus during labor, or when it is destroyed by craniotomy or decapitation.

1. Investigation of a case of infanticide

Includes examination of both mother and fetus (1) **Examination of mother** – note (i) signs of recent delivery (ii) her psychiatric condition (2) **Examination of infant** – Points to be noted are (i) Whether the child was stillborn or deadborn (ii) Whether the infant had attained viability or not (iii) Whether the child was born alive (iv) If born alive, how long did he live and (v) What was the cause of death?

B. Filicide

Filicide *[syn,* ***prolicide****] is killing of a child older than 24 hours by its own parents*. **Salient features:** (1) **Types:** (i) **Maternal filicide** - (a) *Definition - Unlawful destruction of a child by* ***its own mother.*** (b) *MLI* - (I) If her mind was disturbed due to consequences of childbirth, or lactation, eg puerperal psychosis, she is charged under a lesser offence of **manslaughter** (and not homicide) **[s1, Infanticide Act of England (1938)]**. *This defense is not available to anyone else.* (II) **In India** - such a law does not exist. The mother would be charged **u/s 302** for murder, unless she can show that she was incapable of knowing the nature of the act (**s 84 IPC**). (c) Sometimes "maternal filicide" and "infanticide" are taken as synonymous, but the term "maternal filicide" is more appropriate in cases of killing by mother. (ii) **Paternal filicide** - *Unlawful destruction of a child by* ***its own father.*** Very few cases of paternal neonaticide are known. Maternal filicide is much more common.

C. Feticide

Feticide *is the killing of fetus at any time prior to birth* (**Main instances** – MTP, prenatal sex determination followed by selective abortion of female fetuses).

D. Neonaticide

Neonaticide *is killing of an infant within* ***24 hours*** *of birth.* The term was first coined by **Resnick** in 1970. The term is paradoxical, because although the term "neonate" in obstetrics is used to denote *an infant up to 28 days after birth, the term neonaticide in psychiatry and forensic medicine is used for killing of an infant within 24 hours of birth.*

II. Stillbirth

A ***stillborn child*** *is one which is born after 28th week of pregnancy and which did not breathe or show any other signs of life at any time after being completely born* [WHO]. In the UK, according to the *Still-Birth (Definition) Act 1992*, this period is reduced to 24 weeks. **Salient features:** (1) Stillbirth has been called the **Sudden Antenatal Death Syndrome** or **SADS**. (2) The child was alive *in utero* [(i) its heart sounds could be heard by the examining doctor; (ii) movements could be felt by mother; (iii) echocardiography showed beating heart], but dies during the process of birth [eg by aspiration, strangulation by cord etc]. (3) Stillbirths occur more frequently among illegitimate and immature male children in primiparae (4) **incidence** – 5% (5) **Causes** – (i) Anoxia (ii) Birth trauma [especially intracranial hemorrhage due to excessive moulding] (iii) Congenital defects (iv) Erythroblastosis fetalis (v) Placental abnormalities (vi) Prematurity (vii) Toxemia of pregnancy.

III. Dead birth

A ***deadborn child*** *is one, which has died in utero, and shows one of the following signs after it is completely born* (1) *maceration* (2) *rigor mortis* (3) *putrefaction* (4) *Adipocere* (5) *mummification* [Table 1].

A. Maceration

Maceration *(Latin macerare,* to soften by soaking*) is degenerative change occurring in a fetus retained* in utero *after death. It occurs due to the softening effect of soaking on solid tissues* (**pulpy fetus**). **Salient features:** (1) The **earliest sign** of maceration is **skin slippage** (**12 hours after IUD**) [Fig 27.1]. The epidermis can be easily separated from the dermis by applying oblique pressure. Loss of the epidermis exposes a red, shiny, moist dermal surface, particularly noticeable over bony prominences. (2) **24 hours**→fluid-filled bullae are formed between dermis and epidermis [Skin blebs]. (3) **48 hours**→(i) Sweetish, disagreeable odor (ii) internal fetal organs and connective tissue show increasing purple discoloration due to hemolysis and breakdown of red cells. (iii) Dark, red-stained fluid accumulates in the serous cavities. This should be distinguished from serous effusions acquired antemortem. (iv) Proteolytic digestion by

Fig. 27.1: Maceration. Please note skin slippage.

ii. Fallacies

There are **two major fallacies** of this test.

(a) Child respired after birth yet lungs sink

Causes: (1) **Absorption of air** – Circulation continued after stoppage of respiration for sometime→air from lungs is absorbed in circulation→No air remains in lungs. (2) **Atelectasis** (non expansion) of lungs. Sequence of events is ⇨ Feeble respiration→Air does not enter lungs, but remains up to tracheal and bronchial level→Oxygenation of blood occurs through tracheal and bronchial mucosa→Lungs remains unexpanded, but children was alive. (3) **Alveolar duct membrane** - Causing obstruction to entry of air in alveoli (4) **Diseases** - (i) Acute edema (ii) Congenital syphilis (iii) Pneumonia (5) **Feeble respiration** – More air is expelled from the lungs during expiration, than what is inhaled during inspiration (6) **Only a small portion of lung expanded during respiration** – This may enable the child to survive for up to 1-2 days. Lungs will sink as a whole, but when cut to pieces, the respired portions will float. Thus during performance of hydrostatic test, sectioning of the lungs is an important step.

(b) Child did not respire after birth yet lungs float

Causes: (1) **Artificial respiration** - given via a tube, catheter or cannula passed into the trachea or by mouth to mouth respiration. Lungs are inflated partially. In case of mouth to mouth respiration, air may be found in stomach too. (2) **Putrefaction** - Putrefactive gases will make the lungs float. Distinctive features (i) *Body* - shows signs of decomposition (ii) *Lungs* - (a) soft and greenish (b) Bubbles of gas - (I) large bubbles seen on lung surface. (II) Unequal in size. (III) Project considerably from the surface. (IV) Gas within them can be pushed readily from place to place. (V) Collapse on pricking. (3) **Respiration within the womb [vagitus uterinus]**- Fetus may respire within the womb if membranes have ruptured, but may die from natural causes within the birth canal and may not breathe once completely born. (4) **Respiration within the Vagina [vagitus vaginalis]** - Similar to above. Respiration occurs within vagina, but not when completely born. Both **vagitus uterinus** and **vagitus vaginalis** can cause tremendous joy among relatives waiting outside the labor room. Later if they are told that the infant was born dead, they would think that child was indeed born alive, and death was caused by medical negligence. (5) **Respiration in a head protruding from the outlet** - but not when child is completely born.

iii. Hydrostatic test is not necessary in following conditions

(1) **One is sure fetus was born dead** - Fetus is (i) born before age of viability [28 wks] (ii) macerated or mummified (iii) monster [eg anencephalic] (iv) Bruising on lungs - indicating efforts at artificial respiration. (2) **One is sure fetus was born alive** - (i) *stomach* - contains milk (ii) *Umbilical cord* - has separated and a scar has formed.

i. Radiography of lungs

May reveal air within them.

j. Microscopic Examination of the Lungs

(1) **Value** - is limited. Cannot provide clear evidence of extrauterine respiration, if child has lived only for a few minutes. (2) **Procedure** - (i) *Removal* - Thoracic contents are removed intact by cuts with a scalpel by a **"no touch" technique** first described by **Osborn** [Pathologist and Medical Superintendent, Derbyshire Royal Infirmary] in 1953. The technique aims to eliminate artifacts. (ii) *Fixation* - for 48 h (iii) *Sections* - taken from the whole lung (3) **Appearances** - (i) *at 4 months pregnancy* - Parenchyma of the lung shows gland like structure with cuboidal or columnar lining (ii) *at 5th month* - air cells are filled with amniotic fluid (iii) *at full term* - (a) thin walled adult type alveolus is formed.

It was earlier thought that as soon as respiration occurs, cuboidal or columnar [gland like] lining changes to squamous type, but this is not correct. Squamous type lining is simply an indicator of full term. Conversely presence of gland like lining does not indicate lack of respiration; it only indicates prematurity. (b) Completely atelectatic, but many of its terminal bronchioles and vesicles are partly expanded by amniotic fluid. (c) Fetus makes some respiratory movements near full term, which fill its alveoli with amniotic fluid. This material is not stained with H&E giving a spurious impression that air is present in lungs. As respiration takes place, further expansion of alveoli occurs. Fluid present in alveoli is partly absorbed back into the pulmonary circulation and partly expelled through the bronchi. (d) Tardieu's spots - are no indication of live birth. They may be seen in stillbirth, live birth and in bronchopneumonia.

k. Tests for fetal lung maturity

Presence of creatine, lecithin and fat cells.

4. Changes in stomach and intestines

Breslau in the year **1865**, drew attention to the fact that the stomach and intestines in still-born infants sink when placed in water, whilst in infants which were born alive a sufficient amount of air is present within them to render them buoyant. In **1886**, **Ungar** began to advocate the

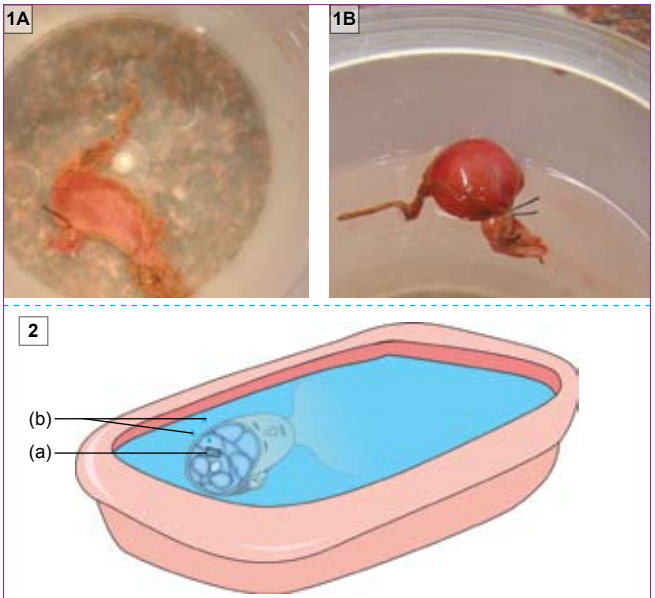

Fig. 27.3: (1A&1B) Breslau's test: (1A) Stomach and intestines from a dead born baby sink **(1B)** The same complex from a live born floats. **(2) Wreden's test:** On removal of roof of middle ear (a), bubbles of air (b) would escaped if the child was born alive.

(f) Crossed-over immunoelectrophoresis

Crossed-over immunoelectrophoresis [syn, *counterimmunoelectrophoresis*] is same as Ouchterlony method, except that here voltage is applied to facilitate movement [Fig 29.9]. Hb carrying a -ve charge, migrates toward the anode. In contrast, the antibody migrates toward the cathode. In a positive reaction, a precipitate line forms.

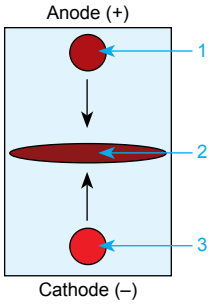

Fig. 29.9: Principle of **crossed-over electrophoresis**. (1) Anti-human antibody (2) Precipitation line (3) Human blood

b. Enzymological

(1) Blood in all animals have similar enzymes, but in different molecular forms [**isoenzymes**]. For example peroxidase [Px], Lactate dehydrogenase (LDH), malate dehydrogenase [MDH] and esterases [Es] have different molecular structures in all species. (2) Because of this difference they travel to different distances during electrophoresis and produce different electrophoretic patterns.

c. DNA Based methods

(1) **Principle** - A sample can be determined to be blood of human origin by reaction with a probe specific for human DNA. (2) **Steps:** (i) *Step 1* - Unknown sample→Probes complementary to **primate specific DNA sequences** [eg those at the locus **D17Z1**], are used to determine the amount of human DNA present in the sample. (ii) *Step 2* - DNA extracted from the sample is spotted on a membrane along with known standards [with known concentrations of human DNA]. (iii) *Step 3* - Human-specific probe is added. Is sample is from human, it must stick to the DNA. (iv) *Step 4* - A color reagent [or any other similar technique] is used to display attached DNA. (v) *Step 5* - Signal intensity obtained from the unknown sample is compared with the signal intensity of the known standards. If it is similar, unknown sample belongs to humans. (3) **Sensitivity** - 0.15-0.20 ng when a color reagent detection method is employed. (4) **Disadvantage** - Human DNA from any tissue or cells will produce a +ve reaction. Thus, it is first necessary to determine that the DNA obtained is from blood by using one of the heme identification techniques discussed earlier.

3. If it belongs to a human, what is the blood group

Determination of blood grouping is routinely done in hematology labs with the use of **Anti-A**, **Anti-B** and **Anti-H**. However the blood that is used is **fresh** with **intact RBCs** which can clump. In a dried stain, *RBCs break down to a powdered form* which cannot show clumping. Thus even if a dried stain is extracted with a regular blood solvent like Vibert's fluid and treated with corresponding antisera, no clumping would result. Therefore in a dried stain one has to take an "indirect route". *Use is made of the fact that even in powdered RBCs, antigens remain intact for a long period*. Antibodies in stains too are stable but for shorter periods than antigens. Currently most methods aim to detect antigens. Earlier methods [eg Lattes method] aimed to detect antibodies. (1) **Material necessary** – (i) About 75 mg of dried blood, or (ii) 150 mg of blood-stained material (iii) Control - free from stain.

a. Methods

i. Detection of antigen in stain

(a) Absorption inhibition

Best understood by an example. Assume that the stain is A. A small amount of Anti-A is added to an A stain→A antigen present in the stain would "absorb" Anti-A→No Anti-A available for subsequent clumping of known group A blood[Fig 29.10]. Called so because antibodies are "absorbed" and the subsequent clumping is "inhibited".

(b) Absorption elution

In the above case, it is possible to detach (or elute) the A antibodies absorbed on to the stain, by heat (56°C), ether or acid. This "eluted" antibody can then be used to clump known A cells [Fig 29.11]. Also known as "**acid elution**" test. By repeating the above two tests by

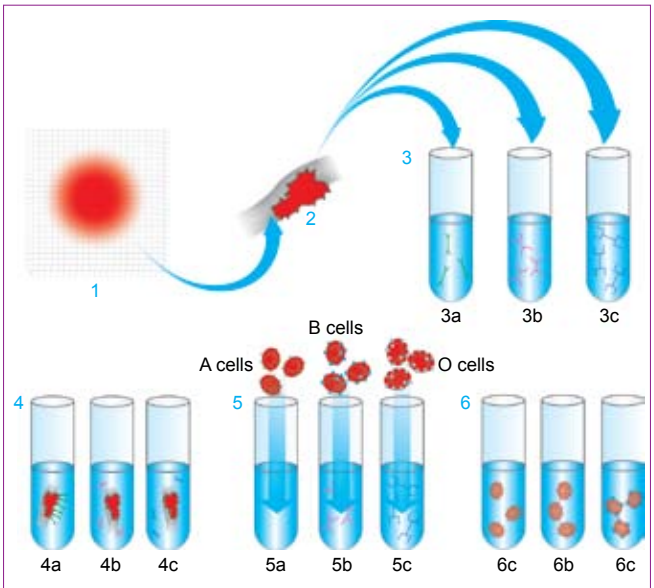

Fig. 29.10: Principle of **absorption inhibition test**. (1) a cloth with an old **A** stain. Several fibres can be pulled from here for testing (2) Fibre with an old A group stain. (3) Three such fibres are taken. Each treated separately with Anti A (3a), Anti B (3b) and Anti H (3c). (4) Fibres within antibody solutions (5) Known A cells are introduced in 5a; known B cells in 5b and known O cells in 5c (6) Since Anti A has been consumed in tube 6c, **A cells** would not clump (**inhibition** of clumping). Cells in other tubes would clump.

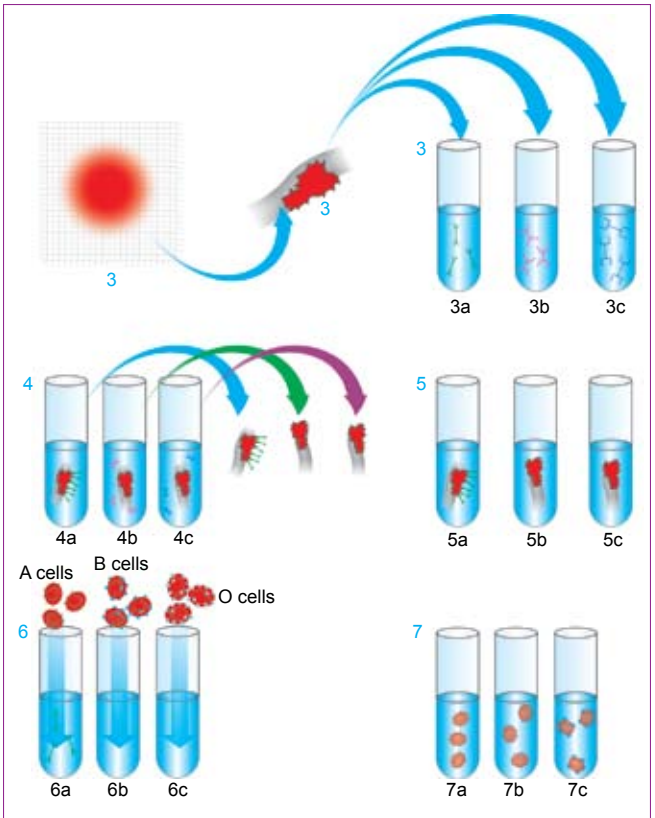

Fig. 29.11: Principle of **absorption elution test**. (1-3) as in Fig 29.10. (4) After fibres have been within antibody solutions for sometime, they are taken out. Anti A sticks to the fibre in 4a. Antibodies in other tubes do not stick (5) Fibres heated at 56°C. Individual Anti A antibodies 'elute' or detach from the fibre in 5a. No antibodies in 5b and 5c. (6) Fibres taken out and known A, B and O cells introduced in 6a, 6b and 6c respectively (7) Clumping in 7a indicates that blood stain belong to group A.

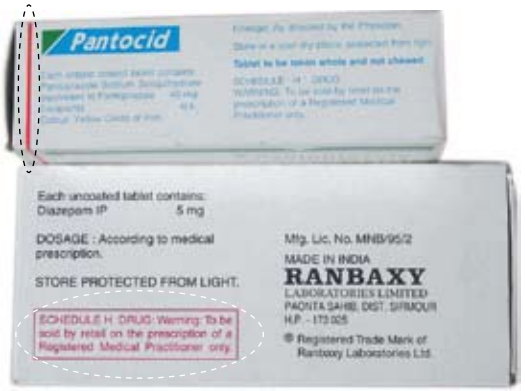

Fig. 31.2: Schedule H warning printed on containers of drugs. Pl note the conspicuous red vertical line on the left side of the upper pack. Both these are legally required in Schedule H drug packings.

(viii) The supply of any drug [other than those specified in Schedule X] on a prescription of a Registered Medical Practitioner must be recorded at the time of supply in a prescription register specially maintained for the purpose. Serial number of the entry in the register should be entered on the prescription. The register must have following particulars (a) serial number of the entry (b) the date of supply (c) the name and address of the prescriber (d) the name and address of the patient (e) the name of the drug or preparation and the quantity (f) in the case of Schedule C and Schedule H drugs - name of the manufacturer, its batch number and the date of expiry of potency, if any (g) the signature of the registered Pharmacist by or under whose supervision the medicine was made up or supplied.

G. The Pharmacy Act, 1948

Please see **Ch 2**.

H. The Drugs Control Act, 1950

It provides for the control of the sale, supply and distribution of drugs. **Salient features:** (1) "dealer" means a person who carries on the business of selling any drug. (2) The **Chief Commissioner** has the power to fix (a) maximum price of a drug (b) the maximum quantity which may be possessed by a dealer [**S.4**] (3) **Refusal to sell** — No dealer or producer can refuse to sell to any person any drug. [**S.8**] (4) **Cash memorandum to be given** — Every dealer or producer will have to issue a cash memo on selling a drug. [**S.9**] (5) **Marking of prices and exhibiting list of prices and stocks**. — All dealers and producers (i) will mark the drugs with sale prices, (ii) keep a price list of drugs held for sale, and (iii) their quantities [**S.10(1)**].

I. The Drugs and Magic Remedies (Objectionable Advertisement) Act, 1954

The objective of this Act is to ban advertisements which offend decency or morality, and to prevent self medication and treatment, which cause harmful effects. **Salient features:** (1) A **magic remedy** is a **talisman**, ***mantra***, ***kavacha*** or any other charm of any kind alleged to possess miraculous powers [**S.2(c)**] (2) **S.3** - Prohibits advertisements of drug for (a) the procurement of miscarriage or prevention of conception (b) the maintenance or improvement of sexual potency (c) the correction of menstrual disorders in women (3) **S.5** - Prohibits the advertising of remedies alleged to possess magic qualities (4) **S.7 – Penalties -** Whoever contravenes the provisions of this Act shall, on conviction, be punishable with imprisonment which may extend to **six months**, with or without fine. In case of subsequent convictions the imprisonment can be extended to **one year**. (5) **S.14** – Prescribes exceptions to above provisions, e.g.. signboards displayed by doctors are exempt, Govt advertisements are exempt [this allows Govt to advertise drugs such as Mala-D which is for prevention of conception].

J. The Medicinal and Toilet Preparations (Excise Duties) Act, 1956

This Act provides for the levy and collection of duties of excise on medicinal and toilet preparations containing alcohol and narcotic drugs.

K. The Narcotic Drugs and Psychotropic Substances (NDPS) Act, 1985

*The Narcotic Drugs and Psychotropic Substances (NDPS) Act was enacted in **1985**, by the Indian Govt (i) to **consolidate** and **amend** the law relating to narcotic drugs (ii) to make **stringent provisions** for regulation of Narcotic drugs and psychotropic substances (iii) to provide for **forfeiture of property** derived from narcotic drug trafficking, and (iv) to **implement the provisions** of the International Convention on Narcotic Drugs and Psychotropic substances*. **Salient features:** (1) **Repeals** - It repealed 3 Acts [**S.82(1)**] namely (i) The Opium Act, 1857 (ii) The Opium Act, 1878 and (iii) The Dangerous Drugs Act, 1930. (2) **Amendments** - It has been amended twice – in 1989 and 2001. (3) **Definitions:** (i) **Narcotic drug** - means coca leaf, cannabis (hemp), opium, poppy straw and includes all manufactured drugs [**S.2(xiv)**] (ii) **Psychotropic substance** - 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 [**S.2(xxiii)**]. Lists 110 substances including Benzodiazepines [Alprazolam, Diazepam, Estazolam, Nitrazepam] Amphetamines, Barbiturates [amobarbital, pentobarbital, secobarbital], Buprenorphine, Glutethimide, LSD, Mescaline, Methaqualone, PCP, [phencyclidine] and Psilocybine. (4) **Cultivation** - of psychoactive plants e.g. coca, opium or cannabis plants is prohibited except for medical or scientific purposes [**S.8**].

V. Medicolegal Aspects of Poisons

(1) **S.85, IPC - Criminal act done under involuntary intoxication** - Person is intoxicated without his knowledge or against his will→becomes incapable of judgment→commits a criminal act→Did not know what he was doing→He is not responsible for the crime. **Example** – **X** administers datura to **Y** without his knowledge→**Y** murders **Z** under the effect of Datura→**Y** is not responsible. (2) **S.86, IPC - Criminal act done under voluntary intoxication** - Person takes intoxicating drug voluntarily→looses self control→Commits a crime→taken for medical examination→Found disoriented in time, place and person [i.e. from a medical standpoint, he did not know what he was doing]→Legally speaking it would still be presumed that *he knew what he was doing*, but his *intention to do so cannot be presumed*. **Example** – **X** drinks alcohol voluntarily →loses self control and exhibits his sexual organs to a female. This is an act which insults the modesty of a woman, and is punishable **u/s 509, IPC**→He is taken to a doctor. *Medical opinion* is that he did not know what he was doing. He neither had knowledge, nor intention of exhibiting his sexual organs→*Legal position* is that he would be *presumed to have knowledge of the act* he was doing, but *he cannot be presumed* to have *intention* of doing it→Essential component of 509IPC is intention and *not knowledge*→Thus prosecution can presume knowledge. But the burden of proving *intent* still lies on it. How prosecution does it, is its own problem, but *voluntary drunkenness cannot be invoked to presume intent*. (3) **S.272, IPC** - *Adulteration of food or drink* — 6 m or ₹ 1000 fine or both. (4) **S.273, IPC** - *Sale of noxious food or drink* — 6 m or ₹ 1000 fine or both. (5) **S.274, IPC** - *Adulteration of drugs* — 6 m or ₹ 1000 fine or both. (6) **S.275, IPC** - *Sale of adulterated drugs* — 6 m or ₹ 1000 fine or both. (7) **S.276, IPC** - *Sale of drug as a different drug or preparation* — 6 m or ₹ 1000 fine or both. (8) **S.277, IPC** - *Fouling water of public spring or reservoir* — 3 m or ₹ 500 fine or both. (9) **S.278, IPC** - *Making atmosphere noxious to health* — ₹ 500 fine. (10) **S.284, IPC** - deals with negligent conduct with respect to poisonous substance. Punishment **6m** or **₹ 1000** fine or both. **Ex** –Housemaid leaves toilet cleaner (acid) negligently on a table→A young child drinks it believing it to be water→Housemaid can be sued u/s 284, IPC. (11) **S.324,** IPC*→ch (11) (12) **S.326,** IPC*→ch 11. (13) **S.328,** IPC: (i) Person gives

35
Agricultural Poisons

Agricultural chemicals *or* ***agrochemicals*** *are chemicals used in agriculture*. When ingested in toxic doses they may cause serious poisoning or death. Agricultural poisons include fertilizers, pesticides, grain preservatives etc.

I. Classification

A. Classification of Agricultural poisons

1. Chemicals/plants used as fertilizers

(i) Nitrites and nitrates (methemoglobinemia) (ii) ammonium sulfate (hyperammonemia) (iii) Anhydrous ammonia (iv) Fertilizers containing urea (v) Poisonous plants used as green manure (e.g. *Ricinus communis*)

2. Chemicals used to kill pests (pesticides)

(i) Acaricides (used to kills mites and ticks, e.g. avermectins, azobenzene, benzoximate, bromopropylate, dofenapyn, nikkomycins, tetranactin. Also known as miticides.) (ii) Algicides (used to control algae in lakes, canals, and water stored for agricultural purposes, e.g. cybutryne, hydrated lime [component of Bordeaux mixture]) (iii) Aphicides (used to kill aphids, e.g. triazamate, dimethoate, and mevinphos) (iv) Avicides (used to kill birds harmful to agriculture, e.g. 4-aminopyridine, 3-chloro-p-toluidine hydrochloride [CPTH]) (v) Bactericides (e.g. bronopol, nitrapyrin, oxolinic acid, oxytetracycline) (vi) Fumigants (Gas or vapor intended to destroy insects, fungi, bacteria, or rodents, used to disinfest interiors of buildings as well as soil before planting, e.g. Carbon disulfide, Sulfuryl fluoride, methyl bromide) (vii) **Fungicides** (Kill disease causing fungi such as rusts, mildews, blights, and molds, e.g. sodium azide, various compounds of **copper** and **mercury**, thiocarbamates, Captan, Captafol) (viii) Herbicide safeners (e.g. benoxacor, cloquintocet, cyometrinil, dichlormid, dicyclonon. These compounds basically protect crops from herbicide injury by increasing the activity of herbicide detoxification enzymes, such as Glutathione S-Transferases and cytochrome P-450s) (ix) **Herbicides**, or weed killers (e.g. Glyphosate, **Paraquat**, **Diquat**, 2-4 D or 2-4 Dichlorophenoxyacetic acid, Mecoprop) (x) **Insecticides** (e.g. **organophosphorus** compounds [OP], **Organochlorine** compounds [OC], **carbamates**) (xi) Microbial pesticides (those pesticides whose active ingredient is a bacterium, virus, fungus, or some other microorganism or product of such an organism. e.g. *Bti* which is made from the bacterium *Bacillus thuringiensis* var. *israelensis* and used to control mosquito and black fly larvae, *Bacillus sphaericus* and *Laegenidium giganteum*, a fungal parasite of mosquitoes.) (xii) Molluscicides – used to kill mollusks, such as snails and slugs (Metaldehyde) (xiii) Nematicides – used to kill nematodes (microscopic, wormlike organisms that feed on plant roots, e.g. 1,3-dichloropropene, 1,2-dibromoethane, Ethylene dibromide, diamidafos, fosthiazate, isamidofos) (xiv) Ovicides – used to kill eggs of insects and mites (xv) Pesticide Synergists (e.g. piperonyl butoxide (PBO), n-octyl bicycloheptene dicarbozimide, piprotal, propyl isome, sesamex, sesamolin) (xvi) Rodenticides (used to kill rodent pests such as e.g. Strychnine, Vacor, ANTU, Cholecalciferol, anticoagulants and Red Squill) (xvii) Virucides (e.g. ribavirin, imanin) (xviii) Miscellaneous Chemical classes including contaminants and adjuvants of some pesticides which are toxic on their own (e.g. Dioxins, present as contaminants of some herbicides produce toxicity of their own)

3. Chemicals used to disturb the feeding/growth/mating behavior etc. of pests, or used for other miscellaneous agricultural purposes

(i) Bird repellents (e.g. anthraquinone, chloralose, copper oxychloride) (ii) Chemosterilants (e.g. 1,2-dibromo-3-chloropropane (DBCP), apholate, bisazir, busulfan, dimatif, tepa) (iii) Desiccants: Chemicals which promote drying of living tissues such as unwanted plant tops or insects. (iv) Defoliants: Chemicals which cause leaves or foliage to drop from a plant, usually to facilitate harvest. (v) Feeding deterrents or antifeedants (chemicals having tastes and odors that inhibit feeding behavior, e.g. pymetrozine, azadirachtin A) (vi) Insect attractants (Attract or lure an insect to a trap, e.g. brevicomin, codlelure, cue-lure, dominicalure, siglure) (vii) Insect growth regulators or IGRs (Chemicals which disrupt the action of insect hormones controlling molting, maturity from pupal stage to adult, or other life processes, e.g. hexaflumuron, teflubenzuron and pyriproxyfen) (viii) Insect repellents (e.g. butopyronoxyl, dibutyl phthalate, diethyltoluamide) (ix) Mammal repellents (e.g. copper naphthenate, trimethacarb, zinc naphthenate, ziram) (x) Mating disrupters (disparlure, gossyplure, grandlure) (xi) Plant activators (a new class of compounds that protect plants by activating their defense mechanisms, e.g. acibenzolar, probenazole) (xii) Plant growth regulators: Substances (excluding fertilizers or other plant nutrients) that alter the expected growth, flowering, or reproduction rate of plants through hormonal rather than physical action.

4. Poisoning associated with preservation of grains

(i) **Aluminum phosphide** poisoning (ii) **Nitric oxide** poisoning (occurring in **silo Filler's disease**).

B. Pesticides

1. Classification [According to toxicity]

Pesticides (Chemicals used to kill pests, a subgroup of agricultural poisons) may further be classified according to their toxicity.

1. Least toxic [generally not fatal]

(i) Phenoxyacetic acid hormones [MCPA or 2-**M**ethyl-4-**C**hloro**P**henoxyacetic **A**cid (Agritox); DCPA or 2,6-**Di**Chloro**P**henoxyacetic **A**cid; TCPA or **Tri**Chloro**P**henoxyacetic **A**cid. They are inhibitors of growth induced by auxin. Used for weed control] (ii) Cuprous oxide (Cu_2O, fungicide) (iii) Lime sulphur washes (orchard fungicides) (iv) Tar oil emulsion (orchard ovicides) (v) Petroleum washes (orchard insecticides) (vi) Weak solutions of sulfuric acid, nitric acid, pelargonic acid or acetic acid [Acids work by lowering pH level on target weeds just enough to kill them. The residues left behind by these natural weed killers are either harmless or useful to plants as fertilizer. Sulfuric acid, for example, oxidizes to form sulfates] (vii) Natural fungicides (a) Tea tree oil (b) Cinnamaldehyde (c) Cinnamon essential oil (d) Jojoba oil (e) Neem oil (f) Rosemary oil.

2. Mildly toxic [fatal dose >10 g]

(i) Chlorinated hydrocarbon insecticides [used as agricultural insecticides] (a) DDT (b) Gammexane (c) Methoxychlor (d) Chlordane, aldrin, dieldrin (ii) Sodium chlorate [used as a mass herbicide].

3. Highly toxic [fatal dose <10 g]

(i) Arsenical compounds [used as weed-killers and orchard insecticides] (a) Sodium arsenite (b) Lead and calcium arsenate (c) Copper Acetoarsenite (Paris Green) (ii) Nicotine, sulphates, tannates [used as horticultural insecticides] (iii) HCN, KCN, NaCN [as disinfectants and raticides] (iv) Dinitro compounds [dinitrophenol (DNP), DNOC (dinitro-orthocresol). Selective weedkillers, ovicides and insecticides] (v) Organophosphorus compounds [agricultural insecticides] (a) Tetraethylpyrophosphate (TEPP) (b) Hexaethyltetraphosphate (HETP) (c) Octamethylpyrophosphoramide (OMPA) (d) Parathion.

C. Classification of insecticides

Insecticides (Chemicals used to kill insects, a subgroup of pesticides) may further be classified according to their origin.

1. Insecticides of vegetable origin

15–45 cm long, (iii) long-stalked, (iv) alternate and palmate with 5–12 deep lobes with coarsely toothed segments. (4) **Fruit** - The fruit [pod] is a spiny, greenish to reddish purple capsule [Fig 37.1] containing large, oval, shiny, bean-like, highly poisonous seeds with variable brownish mottling. Contain three seeds per capsule. (5) **Seeds** – There are two varieties of seeds (i) *Large red seed with brown blotches* [Fig 37.2] – yield 40% oil. Double the size of small seeds. Used mainly for illumination (ii) *Small grey seed with brown spots* – (a) *Size* - 1.2x0.8 cm (b) resemble croton seeds [Table 1] (c) yield 37% oil. (d) Better quality. Used for medicinal purposes. *Oil does not contain ricin*; it remains behind in bean pulp during extraction. Extraction during heated conditions inactivates ricin within the pulp too.

Fig. 37.1: Ricinus communis plant

Fig. 37.2: Ricinus communis (castor) seeds besides a ₹5 coin for size comparison. Please note that they look like ticks

(6) **Poisonous parts** - All parts of the plant are poisonous but seeds are most poisonous [ricin: 1-5%; ricinine: 0.3–0.8%]. (7) **Uses** - The seeds contain between 40% and 60% of a **yellowish oil** (**castor oil** or **ricinus oil**), which is used as an (i) *Additive* - in candles, cosmetic creams, diffusion pump oils, linoleum, lipsticks, lubricants, paints, plasticizers, printing-inks, soaps, transparent paper and varnishes. (ii) *Lubricant* - From World War I until the 1960s it was used as a lubricant for jet engines, high-speed automotives, and industrial machinery. Castor plants were cultivated in large quantities until synthetic oils became available for use. (iii) *Food industry* - Flavorings, candy (eg chocolate), as a mold inhibitor and in packaging. Castor oil is used, which is *not poisonous*. In **Nigeria**, the whole beans are eaten as food, after boiling [heat inactivates ricin] (iv) *Medicinal [purgative] and lighting purposes* (v) *Manure* – seed cake is used (vi) *Fuel, thatching material or for preparing paper pulp* – Plant stalks are used (vii) *Silkworm feed* - In the silk-producing areas, leaves are fed to the silkworms. (viii) *Ornamental* - In countries where synthetic oils have become common, the plant is now grown only for its ornamental value.

A. Active Principles

(1) **Ricin** [*toxalbumin*] (2) **Ricinine** – (i) **a** piperidine alkaloid. (ii) *A **toxalbumin** or **phytotoxin** is a toxic protein found in certain plants and bacteria, capable of stopping protein synthesis*. Common toxalbumins are **ricin, crotin** and **abrin**.

B. Mechanism of Action

Biochemically toxalbumin is a **Ribosome Inactivating Protein (RIP)**, capable of inactivating ribosomes and hence protein synthesis. **3** types of **RIP** are known – **Type I, Type II** and **Type III**. It is best to study **type II RIP** first. (1) **Type II RIP** [**syn:** type 2 RIP] is composed of an **A chain** (or effectomer) that is enzymatically **A**ctive (**Memory Aid:** A → Active) and a **B chain** (haptomer) that **B**inds (**Memory Aid.** B → Binds) the toxin to the surface of cells (Fig 37.3) [Type II *RIP is thus sometimes referred to as A–B toxin*]. Once the RIP **b**inds to the cell surface by the **B** chain, it enters cell by endocytosis. In the cytosol, the A chain inactivates ribosomes → Protein synthesis ceases. One A chain molecule can inactivate approximately 1500 ribosomes per minute, leading to rapid inhibition of protein synthesis and cell death. [**Ex** – Abrin, Crotin, Ebulin [from *Sambucus ebulus*], Modeccin [from *Adenia (Modecca) digitata*], Ricin, Shiga toxin (from *Shigella dysenteriae*) and the related Shiga-like toxin (from certain enterohemorrhagic strains of *E.coli*), Viscumin (from *Viscum album*)]. Type II RIPs evolved from type I RIPs. (2) **Type I RIP [syn:** type 1 RIP**]** consists only of A chain, and is thus active against ribosomes only *in vitro*; *it can not enter the cell on its own*. [**Ex** - Agrostin (from *Agrosternma githago*), Asparin (from *Asparagus officinalis*). (3) **Type III RIP [syn:** type 3 RIP**]** - are actually inactive precursors (**proRIPs**) that require proteolysis before they become active. They are the **least prevalent of all 3 types**. Till date, they have been isolated only from **maize** and **barley.**

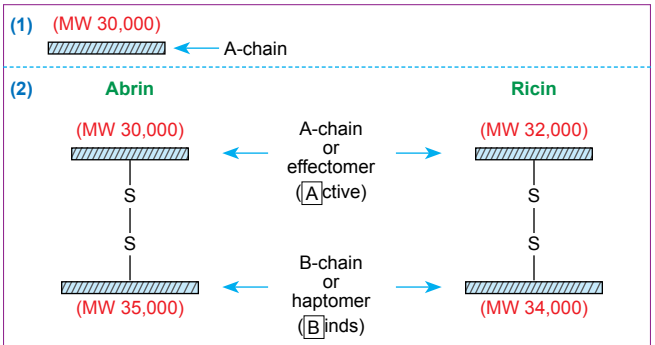

Fig. 37.3: (1) Type I and **(2)** Type II RIPs. Ricin and abrin are type II RIPs.

C. Ricin

After oil is extracted, the **bean pulp** (or fibrous residue) that is left behind contains ricin (approx 1 to 5% of seed's wt). Ricin is a water soluble toxalbumin. It is not present in the oil; castor oil is thus non-toxic (unlike croton oil which is toxic). If oil is extracted under heated conditions, ricin is inactivated. **Ricin** is a **type II ribosome-inactivating protein [RIP]** (Fig 37.3). The seeds also contain **ricinolein**, a triglyceride which is mainly responsible for their purgative action.

D. Signs and symptoms

1. Ingestion

(1) **Method of ingestion** – (i) *Swallowed* - Do not cause poisoning. They pass through the GIT with little or no toxic effects, because the hard seed coat cannot be digested by gastric juices. (ii) *Chewed* - Toxic because ricin is released. (2) **Severity of poisoning** – (i) *Mild* - results in nausea, vomiting, diarrhea, and abdominal pain. (ii) *Moderate to severe* - GIT symptoms progress to dehydration, hypotension, liver and renal dysfunction [↑BUN, ↑creatinine], and death.

2. Inhalation

Dust of seeds causes conjunctivitis, cough, sneezing, acute nasal inflammation, dyspnea, arthralgias, fever, respiratory distress and death.

3. Injection

Generalized weakness, myalgias, hypotension, multiorgan failure and death. Anaphylactic reactions may occur.

Table 1: Difference between venomous and non-venomous snakes

S.No	Trait	Venomous snakes	Non-venomous snakes
1.	General appearance	Stout, dull colored	Slender, brightly colored
2.	Head	triangular	Rounded or oval
3.	Head scales	(1) Small (in **vipers**). Large in others (2) Large (a) pit between eye and nostril → **Pit viper** (b) Third supralabial touches the eye and nasal shields (Fig 38.2) [**cobra** or **coral snake**] (c) (i) No pit ii) third supralabial does not touch the eye and nasal shields. iii) Central row of scales on back enlarged and hexagonal in shape [Fig 38.4] iv) 4th infralabial is the largest (Fig 38.5) [**Kraits**]	Large with exceptions as mentioned in adjacent column
4.	Belly scales	Large and cover entire breadth [Fig 38.3]	Small, like those on the back. May be larger, but do not cover the entire breadth of belly
5.	Anal plate and subcaudal scales	Single row	double rows [Fig 38.3]
6.	Teeth	Two long fangs and a row of smaller teeth	Several small teeth arranged in rows, but no fangs
7.	Fangs	Canalized, like hypodermic needles [vipers] or have grooves [cobra]	Short and solid. Contain no canal or groove
8.	Poison glands	Present	Absent
9.	Saliva	Contains toxic polypeptides and enzymes	No
10.	Tail	(1) may be rounded or flattened (2) tapers abruptly	(1) Always rounded (2) tapers gradually
11.	Habits	Mainly nocturnal	diurnal

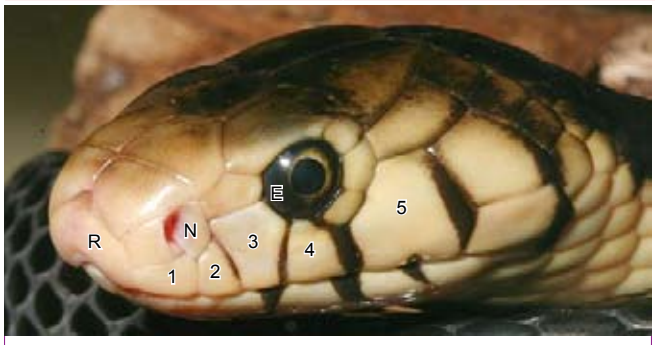

Fig. 38.2: Third supralabial (3) touches both eye (E) and nasal shields (N) in cobra and coral snake. Supralabials are counted numerically from the front, the foremost being called the **rostral scale** (R) or shield. It corresponds to the **mental scale** in the lower jaw [please correlate with Fig 38.5]. The term pertains to the rostrum, or nose. Counting of supralabials (1-5) is done from before backwards as shown.

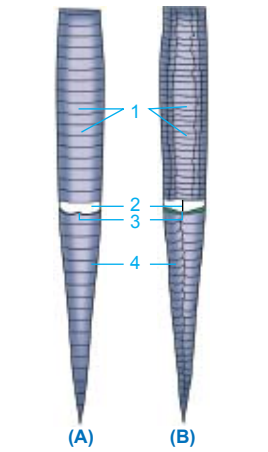

Fig. 38.3: Belly scales in **venomous (A)** and **nonvenomous (B)** snakes. (1) Belly scales (2) Anal scale (3) Cloaca (4) Subcaudal scales.

Fig. 38.4: Large hexagonal scales in the midline on the dorsum of **krait**. Blue colour has been artificially added to highlight the scales.

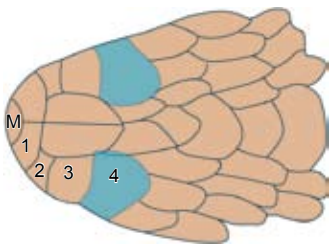

Fig. 38.5: Undersurface of the lower jaw of krait. **Infralabials** (1-4) are counted numerically from the front, the foremost being called the **mental scale** (M) or shield. It corresponds to **rostral scale** of the upper jaw [please correlate with Fig 38.2]. The term pertains to mental nerve which supplies chin. 4th infralabial (in blue) is the largest.

eg, bushmasters, copperheads, cottonmouths, green pit vipers, habus, jararacusus, Malayan pit vipers, mamushi, rattlesnakes, water moccasins]. In addition, there are 2 monotypic [containing only one biological type] subfamilies (c) **Azemiopinae** [Fea's viper] and (d) **Causinae** [night adders]. (ii) Most US venomous snakes belong to *Crotalinae*. Both major Indian vipers [saw scaled viper and Russell's viper] belong to *Viperinae*. (iii) In the past, vipers were assigned 2 families – **Viperidae** and **Crotalidae**; but now they are in subfamilies [with an "n" instead of "d"]. (iv) True vipers are evolutionarily older [called "true" because they came first]; pit vipers are more recent and highly-evolved. **Pit vipers** have a deep pit located between the eye and the nostril. The pit is infrared (IR) sensitive and works like a "thermal camera"; its functions include prey and predator detection in darkness, strike orientation, den site selection and behavioral thermoregulation. It has a maximum effective range of 14 inches. True vipers *do not possess* IR sensitive pit organs. Pit vipers have a keen sense of smell but are deaf to airborne sounds and have poor vision.

e. Hydrophidae

[Gk. *hydros*, water; *ophis*, serpent] (1) Fixed and front-fanged [like elapids], but habitat is water. (2) Sea snakes. evolved from terrestrial ancestors [elapidae]; now adapted to a fully aquatic life; unable to move on land, except for the genus *Laticauda* [sea kraits], which retain ancestral characteristics, allowing limited land movement. (3) Some taxonomists in the past included sea snakes as members of family *Elapidae*. In this scheme family Elapidae consisted of two **subfamilies** – *Elapinae* and *Hydrophinae*. This scheme is no more recognized. (4) Hydrophids are adapted to an aquatic environment morphologically and physiologically - (i) They have specialized flattened tails used for swimming (ii) Due to their need to breathe air, they have valves over their nostrils, which are closed underwater where they swim near the bottom in shallow water to feed. (5) 17 genera are known, comprising 62 species. (6) Divided into two subfamilies - (i) *Laticaudinae*, or **sea kraits** (1 genus, Laticauda, with 5 species) [more primitive] (ii) *Hydrophiinae*, or **true sea snakes** [16 genera with 57 species]. (7) Only a small

8. Cause of death
Renal failure.

9. PM Appearances
(1) Brain – Cerebral edema, chemical meningo-encephalitis, oxalate crystals are seen (2) Liver and kidneys – toxic damage, oxalate crystals are seen (3) Spinal cord – oxalate crystals are seen.

D. Isopropanol
Isopropanol *[2-propanol, isopropyl alcohol] is a clear, colorless, volatile liquid, with a bitter taste and a faint odor of acetone.* **Salient features** -(1) It is used as an antifreeze, disinfectant, industrial solvent, paint remover, preservative, sterilizing agent and for massage [rubbing alcohol is 70% isopropanol] (2) **Absorption** - through all routes (3) **Metabolism** – (i) Kidneys excrete 25%-50% unchanged (ii) rest rapidly converted to **acetone**→excreted in urine and breath. (4) **CNS depressant** - *2-3 times more potent* than ethanol (5) **Symptoms** - (i) *GIT* - (a) Abdominal pain (b) gastritis (ii) *CNS* - (a) Headache (b) Lethargy (c) Ataxia (d) Vertigo (iii) *Respiratory* - (a) apnea (b) Hemorrhagic tracheobronchitis (6) **Treatment** - (i) Gastric lavage (ii) Hemodialysis (7) **Fatal dose** – 250 ml (8) **ML Imp** - (i) In persons dying of diabetes, starvation, chronic alcoholism and severe dehydration, there are ↑conc of acetone. Isopropanol is formed endogenously from reduction of acetone by liver ADH.

V. Opium and its derivatives

A. Opium
Opium *(Afim, Poppy tears, Lachryma papaveris) is the coagulated juice of the opium poppy (Papaver somniferum) or any mixture containing it [**S2**(xv) NDPSA 1985].* **Powdered Opium** is opium dried at a temperature not exceeding 70°C, and reduced to a very fine powder. Powdered Opium yields between 10.0-10.5% of anhydrous morphine. Findings of fossilized opium poppy seeds dating as far back as 30,000 y ago suggest the use of opium by Neanderthal man. The first known cultivation of opium poppies was in Mesopotamia, approximately **3400 BC**, by Sumerians who called the plant *Hul Gil*, the "joy plant." The first reference to opium is found in the **Ebers papyrus** [1500 BC]the writings of **Theophrastus** in the **3rd Cent BC**. Arabian traders who were well-versed with the uses of opium introduced the drug to the East, where it was employed mainly for the control of dysenteries. Opium contains more than 20 distinct alkaloids. Many famous people including English poet **Samuel Taylor Coleridge** [1772 – 1834] and English essayist **Thomas de Quincey** [1785 – 1859] were confirmed opium addicts. The latter even wrote "*Confessions of an English Opium-Eater*" [1821], a classic in English literature. In **1806**, **Sertürner** isolated *morphine* [after *Morpheus*, the Gk god of dreams]. **Robiquet** isolated *noscapine* in **1817** and *codeine* in **1832. Merck** isolated *papaverine* in **1848**. In **1973**, opiate binding sites in the brain were demonstrated. In **1975**, **Hughes** discovered *enkephalin*, an opiate like factor in the brain. **Endogenous opioid peptides** [dynorphins, Enkephalins, endorphins, endomorphins and nociceptin] are the naturally occurring ligands for opioid receptors. The term **endorphin** is used synonymously with endogenous opioid peptides but also refers to a specific endogenous opioid, **β-endorphin**.

1. Plant
(a) Cultivation
Because the lab synthesis of morphine is difficult, morphine is still obtained from opium. It is cultivated in India and other Eastern countries. In India, it is mainly grown in MP, UP, and Rajasthan.

(b) Licensing
In most countries including India, it can be grown only by license from the govt. In India, legal cultivation is carried out only in MP, Rajasthan and UP. Licenses are issued by the **Central Bureau of Narcotics** [CBN]. All opium produced must be sold to the govt. If any licensed cultivator embezzles or illegally disposes of opium, he would be awarded **rigorous imprisonment** of **10-20 y** and a **fine** of between **₹ 1-2 lakhs** [*S.19 NDPSA, 1985*].

(c) Extraction of raw opium
(1) **Opium** - comes from the unripe capsules of *Papaver somniferum* (Gk *papaver*, poppy; *Somniferum*, sleep), which is an annual herb belonging to family *Papaveraceae* [Another poisonous plant of this family is *A. mexicana*. Please see ch 39 – Food poisoning] (2) **Time of sowing** - Poppy seeds are sown in early winter (November). The plant grows up to **1m** in height; flowering occurs in April; Capsules mature in June. (3) **Flowers** - are white, red or purplish, depending on variety (4) **Capsules** - One plant bears 5-8 capsules. Capsules ripen to about 4 cm in diameter; on ripening, their color changes from bluish-green to yellow. (5) **Extraction of opium** – The color change from bluish-green to yellow signals the optimum time for latex collection. while the capsules are still attached to the plant, very shallow incisions are made into their wall (lancing) with a special *nushtar* having three or four small blades, separated by spaces of about 3 mm. (6) **Poppy tears** - The latex which collects on the capsule walls is known as 'poppy tears' (Fig 40.3). On drying up, it becomes *raw opium* [Gk *opos*, juice]. (7) **Poppy seeds** - (i) The capsule has several chambers (loculi) which contain thousands of white, tiny, kidney-shaped seeds known as *khus khus*. They are less than **1mm** in length, and very light. One capsule contains more than 1000 seeds. 3500 poppy seeds weigh about 1 gram. (ii) Poppy seeds are non poisonous, demulcent and nutritive and used for flavoring food. Poppy seed bagels are common in the West. (iii) They yield 45–50% oil (poppyseed oil), which is used for cooking purposes. Also contain traces of morphine [7-60μg/g] and codeine. (iv) *Poppy straw* – Empty poppy capsule without the seeds (also opium straw, poppy chaff, poppy head, poppy pod or *post ka doda*).

Fig. 40.3: (1) Poppy capsules with incisions over surface. Drops of latex (poppy tears) can be seen oozing out. On drying they would constitute raw opium **(2)** Poppy seeds (slate gray variety).

(d) Raw opium
(1) **Physical characteristics** - (i) *Appearance* - Opium appears as a more or less rounded, oval, brick-shaped or elongated, somewhat flattened mass, usually about 8-15 cm in diameter and weighing about 0.3-2 kg each. (ii) *Odor* - Strong characteristic (iii) *Taste* - bitter [due to alkaloids present] (iv) *Consistency* - It tends to be plastic when fresh, but becomes more dense and

41
Deliriant Poisons

I. Datura

Datura *[from Sanskrit Dhurta,* cheat*] is a genus of vespertine [flowering in the evening] flowering plants belonging to the family* ***Solanaceae.*** The Solanaceae family [commonly known as nightshades family] holds **85 genera** and **2,800 species**, and contains many poisonous plants including *Atropa belladonna*, *Hyoscyamus niger*, mandrake, tobacco and *Solanum tuberosum* [**ch 39**]. *Datura* may have originated in Central America but botanists remain unsure of its exact origin. Its hypnotic and hallucinogenic properties were well known since early times. In Columbia, when a rich slave owner died, it was customary to bury his widow and slaves "live" along with his dead body. Datura extract was often administered to prepare them for live burial. Its trance like state [often called an "enlightened state"] is used by tribals in several sacred and religious ceremonies. **Salient features**: (1) *Hyoscyamus* and *Atropa* are related genera. (2) **Habitat** – Grows on waste places all over India (3) **Species** - Nine species are recognized (i) *D. ceratocaula* (ii) *D. discolor* – Desert Thorn-apple (iii) *D. ferox* – Long Spined Thorn-apple (iv) *D. inoxia* – Thorn-apple, Downy Thorn-apple, Indian-apple, Moonflower, Sacred Datura, Toloatzin, Toloache (v) *D. leichhardtii* [syn. D. pruinosa] – Leichhardt's Datura (vi) ***D. metel*** [syn. ***D. fastuosa***] (vii) *D. quercifolia* – Oak-leaf Thorn-apple (viii) ***D. stramonium*** (syn. *D. inermis*) – Jimson weed [The name "jimson weed" is derived from the mass poisoning of British soldiers in Jamestown, Virginia in 167(6) Earlier called "Jamestown weed", which degenerated to Jimson weed], Thorn-apple. Grows at high altitudes in the Himalayas (ix) *D. wrightii* – Sacred Datura, Sacred Thorn-apple. ***D. Metel*** and ***D. Stramonium*** are important. *D.Metel* has two varieties (a) White flowered plant [sometimes loosely referred to as *D. alba*] (b) Deep purple flowered plant [called *D. niger*]. Table 1 gives botanical and common names of some common deliriant plants. (4) **Plant** – characteristics of *D. fastuosa* (i) *Plant* -Leafy herb with a woody stalk that grows up to five feet tall. (ii) *Flowers* – bell shaped [Fig 41.1]. Have five petals, which open after dusk and close by mid-morning the next day [*Datura* is a nocturnal plant]. They exude a pleasant, narcotic scent, especially at night. (iii) *Fruits* – spherical, have sharp spines [Fig 41.2]. Give rise to the plant's English name 'Thornapple'. Contain up to 500 yellowish brown seeds [Fig 41.2]. They look very much like chili seeds and must be differentiated from them [Table 2, Fig 41.3]. (iv) *Poisonous parts* – All parts are poisonous [seeds, fruit, nectar (honey)].

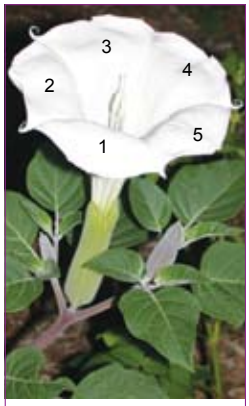

Fig. 41.1: Bell shaped white flowers of Datura. It has 5 petals as shown.

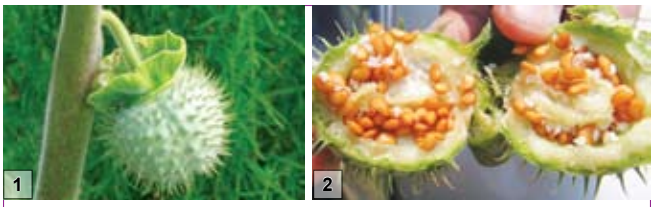

Fig. 41.2: **(1)** Fruit of Datura, showing spines **(2)** Fruit opened to show yellowish brown seeds inside. They look very much like chili seeds

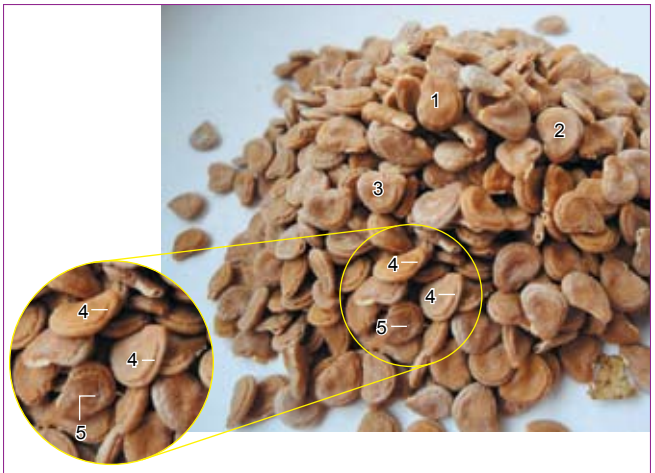

Fig. 41.3: Datura seeds showing typical characteristic. Please correlate nos. with Table 2.

Table 1: Botanical and common names of some common deliriant plants

S.No.	Botanical name	Common name
1.	*Atropa belladonna*	Deadly nightshade
2.	*Datura alba*	Safed Datura
3.	*Datura fastuosa*	Thorn apple
4.	*Datura niger*	Kala Datura
5.	*Datura stramonium*	Jimson weed, Thorn apple
6.	*Hyoscyamus niger*	Henbane, Khorasani ajwayan

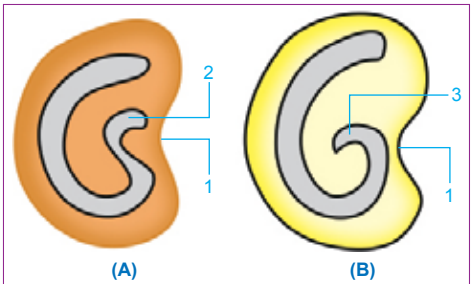

Fig. 41.4: Longitudinal section of **datura seeds (A)** and chili seeds **(B)**. (1) Hilum (2) Embryo curved outwards (3) Embryo curved inwards. (2 & 3) are visible on microscopic examination.

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Spinal and Peripheral Nerve Poisons

I. Spinal Poisons

A. Strychnos Nux vomica

[A] *Strychnos* is a genus of tropical woody plants, many of them trees, in the family **Loganiaceae** (order Gentianales). There are about **190 species**. Several are important sources of drugs or poisons, eg *S. Nux vomica* (source of strychnine) and *S. toxifera*, a native of South America (source of curare). A few species are valued locally for their sweet fruits, including *S. unguacha* and *S. spinosa* (Natal orange). *S. potatorum*, found in India is used as a coagulant to purify water. *S. Nux vomica* **(poison nut)** was first used in medicine by the Arabians, who described it in 1540. In the 16th century in Germany, it was used as a rat poison. In the 18th century, it was used in Europe, as a tonic, with poor results. Strychnine's use was extended considerably in the 19th century, following the introduction of the homeopathic remedy, nux vomica. *S. Nux vomica* is known as ***Kuchila*** in India. Other names are **Dog button** (because sometimes used to kill stray dogs) and **Quaker Buttons**. [B] **Tree** - *Strychnos nux-vomica* (Common Name: Nux-Vomica Tree) is a small evergreen tree native to Asia. (i) **Flowers** - It has yellowish-white tubular flowers which grow in terminal clusters. (ii) The **leaves** - are ovate growing to about 2 by 3.5″. (iii) The **fruit** is round, 1.5″ across and hard shelled; it varies in color from yellow to orange and resembles a small grapefruit (Fig 42.1).

Fig. 42.1: Fruit of Nux vomica

(iv) **Seeds** - On breaking open, the fruit reveals white or pale yellow jelly like pulp. Each fruit contains about **3-5 coin sized gray velvety seeds** that look like coat buttons (Fig 42.2, Fig 42.3). Size is 2.5 cm in diameter and 0.6 cm in thickness. They are flat, circular discs, or sometimes slightly convex on one side and concave on the other. Color is ash-grey or light brown. They have a shining surface and are covered with shiny silky fibres. They are very hard, tough and difficult to pulverize. (v) **Active principles:** (a) The **whole tree**, including the seeds, is poisonous. The active principles are alkaloids **strychnine** and **brucine**. Strychnine is about 10-20 times more poisonous than brucine. The alkaloidal content of the seeds ranges from 1.8 to 5.3%. They also contain a glucoside **loganin**. (b) **Leaves** - contain **vomicine** (major constituent), brucine (1.6%) and strychnine (0.025%). (c) **Bark** – The bark contains 9.9% total alkaloids (brucine 8%, strychnine 1.58%); pseudostrychnine, pseudobrucine and beta-colubrine in small amounts. (d) **Roots** – contain 0.99% alkaloids (brucine 0.28%, strychnine 0.71%). (e) **Fruit pulp** – has very low strychnine content (vi) **Use** - (a) used as **rodenticides**, and for **killing stray dogs**. The commercial baits available contain < 0.5% strychnine and are dyed red or green to make them distinctive. (b) Once used as a **tonic** and a reflex stimulant of gastric secretion (because of its bitter taste) (c) **Analeptic and Respiratory stimulant** – But only in toxic doses. [C] **Strychnine** – Strychnine was first isolated from the beans of *Strychnos ignatii* (**St. Ignatius bean**) by **Pelletier** and **Caventou** in **1818**. Its structure was determined by Sir Robert Robinson and Herman Leuchs. It occurs as colorless, odorless rhombic prisms, and has an intensely bitter taste. Strychnine is one of the **most bitter substance known**; its taste is detectable in a dilution of **1:70,000**. Despite its intensely bitter taste, surprisingly strychnine has been used for homicide quite commonly. It is because it was usually mixed in bitter alcoholic drinks (already bitter in taste), and if the subject is already under the influence of drink, he is further less likely to detect it. Strychnine is also found in **Upas tree** (*S. tieuté*), from which the Malaysian natives used to obtain poison for their arrows and darts.

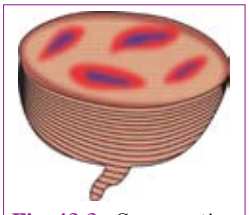

Fig. 42.2: Cross section of the fruit of Nux vomica showing position of seeds inside

Fig. 42.3: Seeds of Nux vomica

1. Absorption and Excretion

(1) Strychnine is rapidly absorbed from the GIT, nasal mucosa, and all parenteral sites. (2) It is rapidly metabolized in the liver (up to 80%) by microsomal enzymes. (3) The highest concentrations of strychnine are found in the liver, kidneys and blood. (4) Strychnine taken up by liver and muscles may be released into

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Cardiac Poisons

I. General

Cardiac glycosides are found in several plants, namely (1) Aconite (2) Balloon cotton [*Aesclepias fruiticosa*] (3) Bushman's poison [*Carissa acokanthera*] (4) dogbane [*Apocynum cannabinum*] (5) foxglove [*Digitalis purpurea*] (6) Frangipani [*Plumeria rubra*] (7) King's crown [*Calotropis procera*] (8) lily of the valley [*Convallaria majalis*] (9) oleander [*Nerium oleander*] (10) False hellebore, Pheasant's eye, spring pheasant's eye, yellow pheasant's eye [*Adonis vernalis*] (11) Redheaded cottonbush [*Aesclepias curassavica*] (12) Rubber vine [*Cryptostegia grandiflora*] (13) Sea-mango [*Cerbera manghas*] (14) squill [*Urginea maritima*/sea onion/indica bulbs] (15) *Strophanthus kombe/gratis/gratus* seeds (16) Suicide tree [*Cerbera odollam*] (17) Treacle-mustard, wormseed mustard [*Erysimum cheiranthoides*] (18) yellow oleander [*Thevetia peruviana*] (19) Wintersweet [*Carissa spectabilis*] (20) Woolly Foxglove [*Digitalis lanata*].

A. Mechanism of action

All cardiac glycosides act by following mechanisms. (1) *Inhibition of sodium-potassium-adenosine triphosphatase pump*→↑ed levels of intracellular Na^+ and ↓ed levels of intracellular K^+ [associated with hyperkalemia] (2) ↑*ed levels of intracellular* Ca^{++} →cardiac irritability and dysrhythmias. (3) ↑*cardiac vagal tone* →↓es cardiac sympathetic activity →bradycardia and heart block. *The consequences of an overdose are essentially an extension of the therapeutic effects*.

B. Laboratory Tests

Patients who have symptoms after exposure to plants containing cardiac glycosides require following tests (1) complete blood cell count, (2) electrolytes, (3) renal function tests [BUN, serum creatinine] and (4) serum digoxin level. In addition (5) continuous cardiac monitoring and (6) serial electrocardiography is essential. (7) Immunoassays indicate the presence of a cardiac glycoside, but because the actual measurement does not correlate with clinical toxicity, they are not clinically useful.

II. Aconite

Aconitum [aconite, blue rocket. Devil's helmet, helmet flower, leopard's bane, *Meetha Bikh*, *Meetha Bish*, *Meetha zeher*, monkshood, wolfsbane, women's bane] is a genus of flowering plant belonging to the buttercup family (*Ranunculaceae*). During ancient times, aconite was a common poison in Asia and Europe. It was a medicinal drug, a homicidal agent and an arrow poison, all rolled in one. The death of the Jewish High Priest **Alkimos** in **159 BC** was regarded as stroke-related (collapse and loss of speech followed shortly by death). However, the description of severe pain suggests aconite poisoning. So prevalent was the use of *Aconitum napellus* in the Roman empire that the emperor **Trajan** [98–117 AD] eventually banned the growing of this plant in all Roman domestic gardens. Roman poet **Ovid** [43 BC – AD 18] referred to aconite as the "**stepmother's poison**". The manner of death of the Roman Emperor **Claudius** in **54 AD** was consistent with some of the clinical effects of acute aconite poisoning; his spouse, Agrippina, was the suspected assassin. The Ainu inhabitants from the Japanese island of Hokkaido concocted an arrow poison called *surku* from *A. japonicum*. **Salient features**: Aconitum belongs to family *Ranunculaceae* (buttercup). There are over 250 species of *Aconitum*, but most common are ***Aconitum napellus*** and ***Aconitum ferox***. *Habitat* - Grows in the Himalayas. *Physical Description* - It is a perennial herb. Grows 2–6 ft in height with palmate leaves [leaflets radiating from the base of the leaf, like palm with outstretched fingers] and a tuberous root [Fig 43.1].

Fig. 43.1: (1) Roots of Aconite and **(2)** Roots of horseradish

Poisonous parts - All parts of the plant are poisonous. Young plants are least poisonous; more so when seeds ripen; and most when plant blooms; Roots are the most poisonous parts of the plant.

Dry root – *Size* - 5-10 cm long; 1-2 cm thick *Color* – dark brown; when freshly cut color is white on the inside, which becomes pink on exposure to air when dried and soaked in oil→ black, heavy, hard, brittle with a strong offensive odor *Smell* – Odorless *Taste* – Sweet initially; later acrid *Physical characteristics* - Arched, shriveled, shows longitudinal wrinkles, scars and bases of broken rootlets, conical and tapering *Mistaken for* – horseradish root [Table 1]. **Horseradish** is important medicolegally in another way - it can give false tests for blood [**ch 29**].

Table 1: Differences between root of aconite and root of horseradish [Fig 43.1]

S.No	Feature	Root of aconite	Root of horseradish
1.	Size	Short and conical	Long and cylindrical
2.	Color [external]	Dark brown	Yellowish white
3.	Color [on cutting]	White; becomes pink on exposure to air	White; does not change on exposure to air
4.	Taste	Sweet initially; later acrid	Pungent

Appendix 1: Medicolegally Important Sections and Acts

Note

1. **ᑫ indicates a potential question [theory, viva, MCQ]. They have also been rendered in bold type. Other sections may be remembered for extra credit.**
2. **This appendix is meant to help the student quickly revise important sections. For details, chapter number mentioned must be referred to.**

I. IPC

Chapter II: General Explanations [S 6-52A]

1. **S.44ᑫ. Definition of Injury - ch 11.**
2. **S.51.** Oath - The word "oath" includes a solemn affirmation substituted by law for an oath, and any declaration required or authorized by law to be made before a public servant or to be used for the purpose of proof, whether in a Court of Justice or not - **ch 1.**

Chapter IV: General Exceptions [S 76-106]

3. **S.80. Accident in doing a lawful act** — Nothing is an offence which is done by accident or misfortune, and without any criminal intention or knowledge in the doing of a lawful act in the lawful manner by a lawful means and with proper care and caution – **ch 1.**
4. **S.81. Act likely to cause harm, but done without criminal intent, and to prevent other harm** — Nothing is an offence merely by reason of its being done with the knowledge that it is likely to cause harm, if it be done without any criminal intention to cause harm, and good faith for the purpose of preventing or avoiding other harm to person or property – **ch 1.**
5. **S.82ᑫ. Age of criminal responsibility** in India is **7 y - ch 3.**
6. **S.83ᑫ. Act of child between 7-12 y of immature understanding** — not an offence - **ch 3.**
7. **S.84ᑫ. Criminal responsibility of mentally ill.** Indian equivalent of British McNaugthen rule - **ch 28.**
8. **S.85ᑫ. Person intoxicated against his will** - not responsible for his criminal actions - **ch 31.**
9. **S.86.** Person intoxicated voluntarily - Knowledge of act is presumed; intention to do the act is to be proven by prosecution - **ch 31.**
10. **S.87.** To participate in a risky activity, the consenting person must be >18 y. *Not meant for doctors* - **ch 2.**
11. **S.88ᑫ. Act done is in good faith for the benefit of the person** [as in case of doctor treating patient] the consenting person must be >12 y. *Meant for doctors* - **ch 2.**
12. **S.89ᑫ . Consent on behalf of minors - ch 2.**
13. **S.90ᑫ. Conditions of a valid consent - ch 2.**
14. **S.92ᑫ. Consent not required to save the life of person - ch 2.**

Chapter X - Of Contempts Of The Lawful Authority Of Public Servants [s 172-190]

15. **S.172.** Absconding to avoid service of summons or other proceeding — **ch 1.**
16. **S.174.** Non-attendance in obedience to an order from public servant - Applies also to **non-attendance in court after service of summons — ch 1.** Not to be confused with **s174 CrPC.**
17. **S.176.** Omission to give notice or information to public servant by person legally bound to give it — **ch 31.**
18. **S.178.** Refusing oath or affirmation when duly required by public servant to make it — **ch 1.**

Chapter XI: Of False Evidence And Offences Against Public Justice [s191-229A]

19. **S.191ᑫ. Definition of Perjury** [Giving false evidence] — **ch 1.**
20. **S.193ᑫ. Punishment for perjury — ch 1.**
21. **S.197.** Issuing false certificate — **ch 1.**
22. **S.198.** Using false certificate — **ch 1.**
23. **S.201ᑫ. Causing disappearance of evidence of offence — ch 31.**
24. **S.202, IPC.** Intentional omission to give information of offence by person bound to inform — **ch 31.**
25. **S.228A.** Identity of victim of rape cannot be disclosed - **ch 25.**

Chapter XIV: Of Offences Affecting The Public Health, Safety, Convenience, Decency And Morals [s268-294A]

26. **S.268.** Public nuisance - Exhibitionism, transvestism, fetishism, masturbation in public - **ch 25.**
27. **S.269.** Negligent act likely to spread infection of disease dangerous to life, eg HIV - **ch 2, 25.** [**Mnemonic - 69** is a sexual position]
28. **S.270.** Malignant act likely to spread infection of disease dangerous to life - **ch 2, 25.**

[Both 269 and 270 are applicable in ML aspects of HIV and AIDS. Also sale of adulterated food]

29. **S.272.** Adulteration of food or drink — **ch 31.**
30. **S.273.** Sale of noxious food or drink — **ch 31.**
31. **S.274.** Adulteration of drugs — **ch 31.**
32. **S.275.** Sale of adulterated drugs — **ch 31.**
33. **S.276.** Sale of drug as a different drug or preparation — **ch 31.**
34. **S.279.** Rash driving or riding on a public way so as to endanger human life →6 months or 1000 Rs fine or both.
35. **S.284ᑫ. Negligent conduct with respect to poisonous substance ch 31.**

Appendix 3: Important Values Regarding Fatal Doses etc

I. Fatal dose, fatal periods and antidotes in a nutshell

S.no.	Poison (ch no)	Fatal dose	Fatal period	Antidote
1.	Acid, hydrochloric (**ch 33**)	15-20 mL	12-24 h	-
2.	Acid, nitric (**ch 33**)	10-15 mL	12-24 h	-
3.	Acid, sulfuric (**ch 33**)	10-15 mL	12-24 h	-
4.	Aconite (**ch 43**)	Root 1-2 g Aconitine 2 mg	2-6 h	Atropine
5.	Aluminum phosphide (**ch 35**)	1-3 tab	24 h	-
6.	Arsenic trioxide (**ch 36**)	200-300 mg	1-2 d	BAL
7.	Aspirin [acetylsalicylic acid] (**ch 45**)	15-20 g	Few min-several h	-
8.	Atropine (**ch 41**)	120 mg	24 h	Physostigmine
9.	Barbiturates [long acting] (**ch 40**)	3-5 g	1-2 d	-
10.	Barbiturates [short acting] (**ch 40**)	1-2 g	1-2 d	-
11.	Cannabis (**ch 41**)	**THC** – 30mg/kg; **Charas** - 2g/kg; **Ganja** - 8g/kg; **Bhang** - 10g/kg	Several days	-
12.	Cantharides (**ch 38**)	10-80 mg	24-36 h	-
13.	Carbon dioxide (**ch 44**)	90,000 ppm	5 min	O_2
14.	Carbon monoxide (**ch 44**)	50,000 ppm	few seconds	O_2
15.	Castor seeds [crushed] (**ch 37**)	5 well-chewed seeds	2 days	-
16.	Cocaine (**ch 41**)	1 g IV	Few minutes to few hrs.	-
17	Copper sulphate (**ch 36**)	0.15-0.3 g/kg	1-3 days	d-Penicillamine, BAL
18	Croton (**ch 37**)	4 seeds; 20 drops of croton oil	6 h-3 d	-
19	Curare (**ch 42**)	60 mg	1-2 h	
20.	Cyanide (**ch 34**)	(1) HCN - 50 to 60 mg (2) NaCN, KCN – 200 to 300 mg	(1) HCN - 2-10 minutes (2) KCN or NaCN - 30 min	Amyl nitrite, sodium nitrite, sodium thiosulphate
21.	Datura (**ch 41**)	(1) Atropine or hyoscyamine – 120 mg (2) Scopolamine – 30 mg (3) Datura seeds – 50-100	24 hrs	Physostigmine
22.	DDT (**ch 35**)	15-30 g	One to several hrs	-
23.	Diamond powder (**ch 34**)	Not fatal	Not fatal	-
24.	Diazepam (**ch 46**)	100-300 mg/kg [20g for a 70 kg man].	1 day	Flumazenil
25.	Diazinon (**ch 35**)	1 g	24 hrs	Atropine
26.	Digitalis (**ch 43**)	(1) Digitalin – 15-30 mg (2) Digoxin [present in D. lanata] – 10 mg (3) Digitoxin – 4mg; (4) Leaves 1-2 (2g)	24 hrs	Digoxin-Specific Antibody Fragments or Fab
27.	Ethyl alcohol (**ch 40**)	(1) Adults - 5-8 g/kg absolute ethanol. (2) Children - 3 g/kg (3) Blood conc → >500 mg%	12-24 h	-
28.	Ethylene glycol (**ch 40**)	100-200 mL	3 days	Ethanol, fomepizole
29.	Formaldehyde (**ch 47**)	60-90 ml	1-2 days	1% ammonium carbonate; 0.1% sol^n of ammonia
30.	Heroin (**ch 40**)	50 mg	6-12 hrs	Naloxone

Textbook of Forensic Medicine and Toxicology

This book, primarily written for students, presents information in a logical, systematic, clear and concise manner for better, easier and faster memorization. Core principles of Forensic Medicine and Toxicology are explained to the overburdened undergraduate students in a simple and examination-friendly manner.

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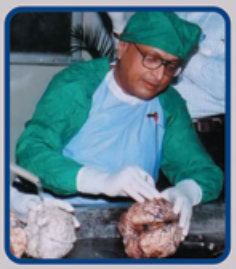

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