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FORENSIC MEDICINE
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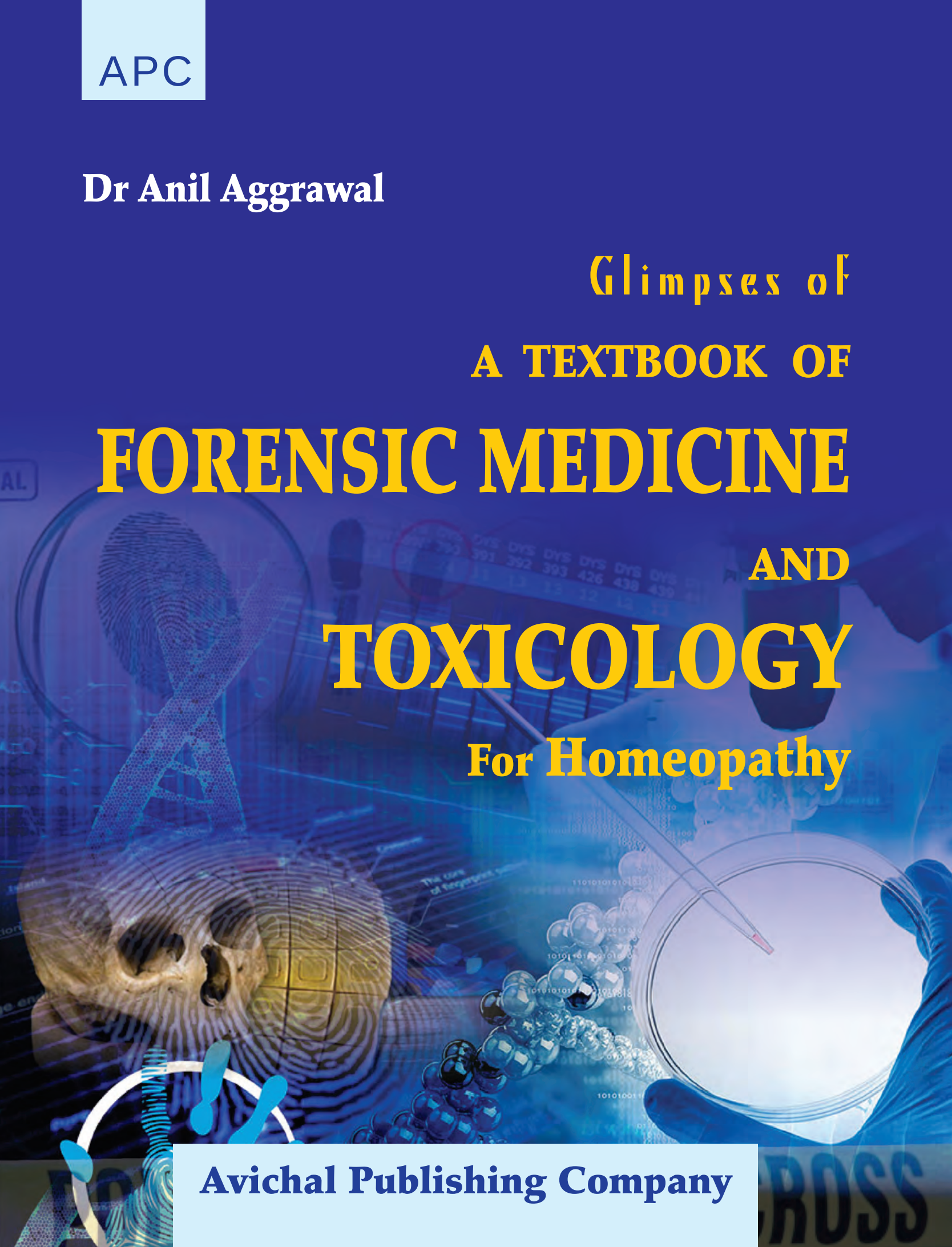
For Homeopathy



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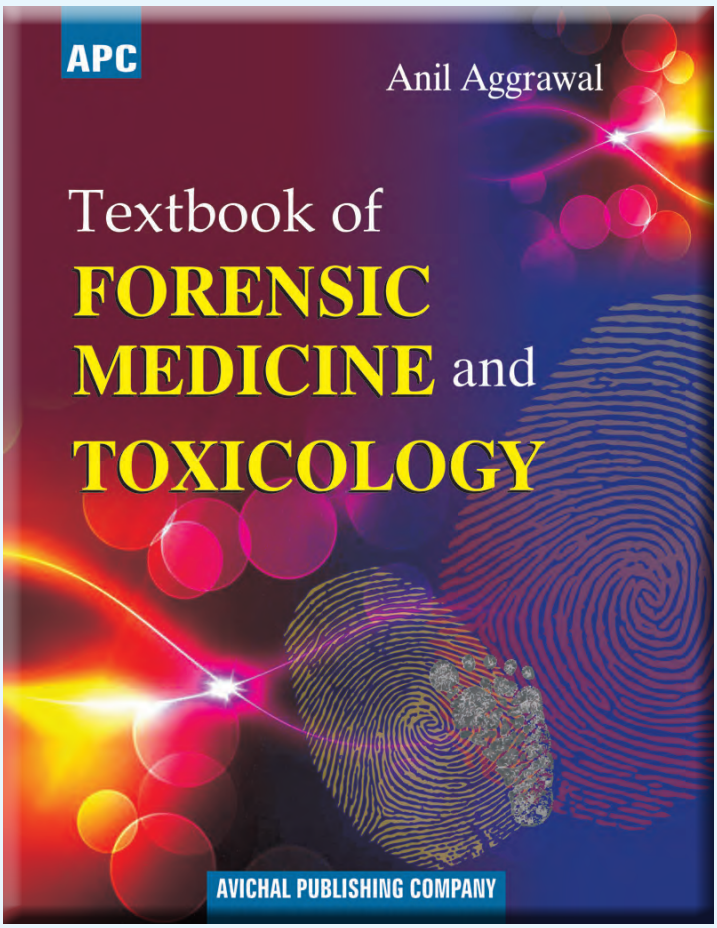
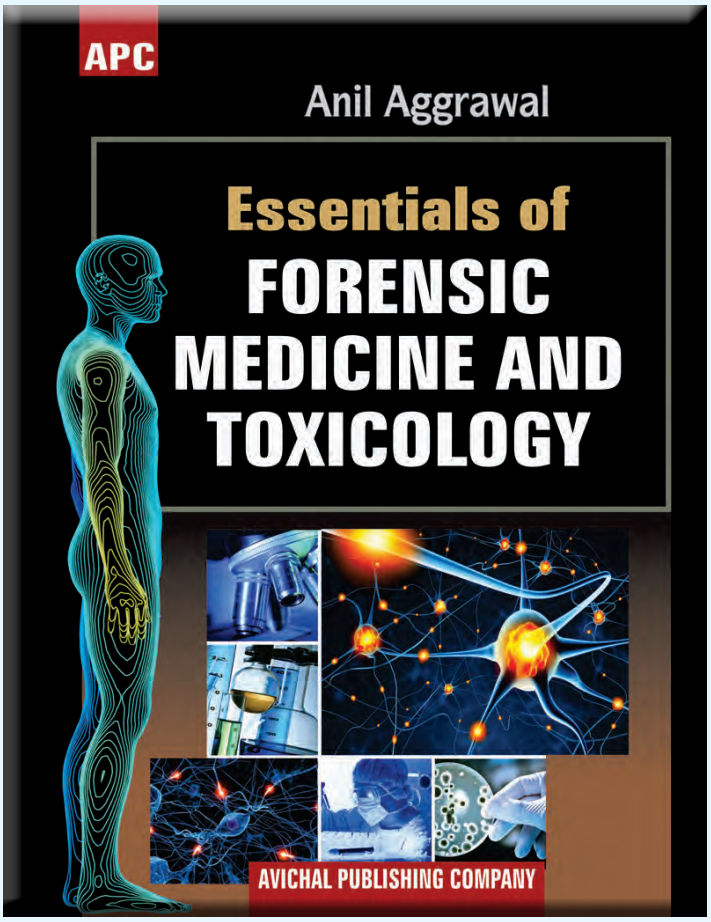




CONTENTS

Chapter 3

Identification

I. INTRODUCTION

1. ***Identification*** *is the determination of the individuality of a person – living or dead.*

Fig. 3.1: Ramendra Narayan Roy, the prince of Bhawal Estate, whose true identity was in question in early 20th century.

A. Case studies

1. Bhowal Sanyasi Case [1921]

A rich person **Kumar Ramendra Narayan Roy** [Fig 3.1] allegedly died of biliary colic in **1909** in Darjeeling. In **1921**, a Sadhu (Bhowal Sanyasi) arrived in Dacca, and **claimed that he was Kumar Ramendra**, and asked for his share in property. When denied, he filed a suit in a **Dacca court** in **1930**, for restoration of his legal rights. Most of his **identification data** was **exactly the same** as that of Ramendra including pink and white complexion, brown wavy hair, light moustache, twisted lip, sharp angle at the rim of ear, ear lobes pierced and not adherent to cheeks, broken left upper first molar, a fleshy point in the right lower eyelid, size 6 for shoes, irregular scar on left ankle, syphilitic ulcers, boil mark on the head and back, operation mark near the groin, tiger claw mark on the right arm and even a minute mole on the dorsum of the penis. He was **declared by the court** to be **Kumar Ramendra Narayan Roy** and **entitled to the property**. However the Bhowal Sanyasi died shortly after the decision of the court in **July 1946**.

II. CORPUS DELICTI

Corpus delicti *[from Latin corpus, body; dēlictum, crime] is the legal principle requiring* ***positive proof of crime*** *before someone can be convicted of committing that crime.* Also known as the ***body of crime, foundation of crime*** or ***essence of crime***. **Salient features:**

1. *Corpus delicti* includes anything which positively prove a crime.
2. In case of *homicide*, it would include the **dead body, photograph** of **dead body, blood stained clothes showing weapon marks, blood stained weapon, bullet, severed body parts (eg head)** etc.

III. IDENTIFICATION DATA

Table 1 enumerates important identification data. Please also see Fig 3.2.

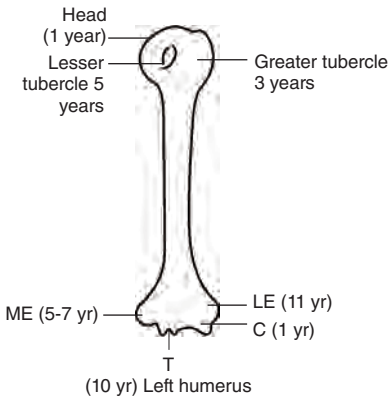
Table 1: Important Identification data

(A) Race (B) Religion (C) Sex (D) Age (E) Stature (F) Complexion (G) features (H) Hair (I) Deformities and other peculiarities such as moles, birthmarks (J) Scars (K) Tattoos (L) Occupation marks (M) Anthropometry (N) Dactylography (O) Footprints (P) Other prints, such as palatoprints, and lip prints (Q) Superimposition (R) Reconstruction of features from the skull (S) Identification from teeth and bite marks [forensic odontology] (T) Clothes and personal articles (U) Molecular methods (V) Miscellaneous methods -

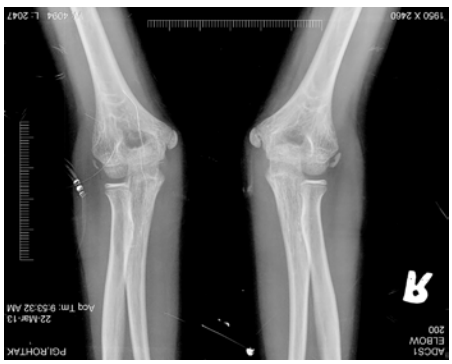
1. Biometric methods - (a) Physical characteristics (i) Bone comparisons (ii) DNA profiling (iii) Frontal sinus pattern (iv) Iris scans (v) Nails (vi) Retina scans (vii) Skull suture pattern (viii) vascular grooves (ix) Stains (x) Stomach pictures (xi) Veins on the back of hands (xii) X-rays (b) Behavioral characteristics - (i) Gait (ii) Handwriting (iii) Mental power, memory and education (iv) Speech and voice (v) Tricks of manner and habit.
2. Non Biometric methods - (a) Facial composites. Some of these data can only be used in living [eg behavioral ch], some only in dead [eg superimposition], and some in both [eg dactylography]. Except dactylography, no single feature is completely reliable, so a set of 4-5 criteria [or more] may be taken for identification. More the number of criteria, better is the identification.

A. Race

Three races are recognized: (i) **Caucasoids** (ii) **Mongoloids,** and (iii) **Negroids**. Race – as mentioned above - can be determined by:



(1)



(2)

Fig. 3.9: (1) Ages of appearance of ossification centers of humerus (left); (2) X-ray 1 – X-ray of 13 y old girl. Medial epicondyle appeared (>7 y); Lateral epicondyle appeared (>11 y). Conjoint epiphysis has formed (>12 y), but has not united with shaft (<14 y). Credit for this pic to Yogesh Vashisht.

at 1; Greater tuberosity at 3; Lesser tuberosity at 5. Remember that in English alphabet g comes before l, and in arithmetic 3 comes earlier than 5:

G → L

3 → 5

All three centers unite by 6 y, i.e. 1 y after the last centre comes.

[II] **Lower end of humerus** [Fig 3.9]→There are four major centers to remember: Medical epicondyle, trochlea, capitulum and lateral epicondyle.

Memory Aid 12 – Ossification centers of lower end of humerus

1. **C**apitulum - appears at one [remember "**c**" for child. Children are younger so it comes very early, i.e. at 1]
2. **T**rochlea - appears at **T**en (remember "**T**")
3. **L**atera**l** **Epicondyle** - appears at 11 (Remember "lateral". Both beginning and ending letters are "l". If we keep them together, they look like 11)
4. **M**edial **E**picondyle - appears at **5-7** [**F**ive-**S**even]. [**M**y **E**xcellent **F**orensic **S**cience]
5. **LTC** (**L**ateral epicondyle, **T**rochlea and **C**apitulum) join together to form a **conjoint epiphysis**, which then joins with the shaft.
6. **Medial epicondyle** joins the **shaft of humerus** separately [Table 24].

Table 24: Ossification of lower end of humerus

Criteria	Females	Males
Conjoint epiphysis forms	12	13
Conjoint epiphysis unites with shaft	14	15
Medial epicondyle unites with shaft	15	16

[III] **Radius and ulna**: Make a representation of the two bones [Fig 3.10]. Make a capital "N" beginning from the lower end of radius as shown, and write numerals 2→5→6→9

1. Lower end of radius appears at 2 years
2. Upper end of radius appears at 5 years
3. Lower end of ulna appears at 6 years
4. Upper end of ulna appears at 9 years [IV] **Femur**: Remember **1** -> **4** -> **14** [formed by the first two digits]. Head of femur appears by **1** year, greater trochanter by **4** years and lesser trochanter by **14** years [Fig 3.11].

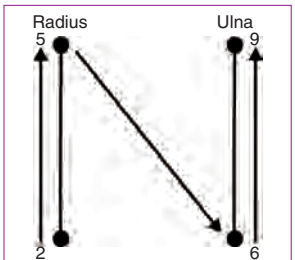


Fig. 3.10: A simple way to remember ossification centers of radius and ulna Pl correlate with X-rays in Fig 3.9 [**visual Memory Aid – 13**]

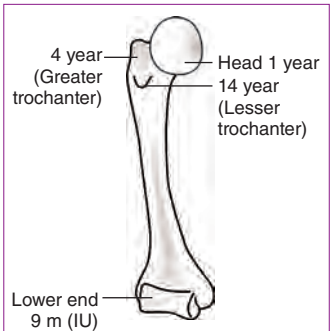


Fig. 3.11: Ossification centers in femur

Angle of neck with shaft 150° in newborn. Angle in adults is different depending on sex [Table 18].

[V] **Tibia and fibula**: Make the bones (as in case of radius and ulna) and write a capital "U". Now write the numerals 0→1→1→4 [Fig 3.12]

1. Upper end of tibia appears at 0 years, i.e. just at birth [in **80%** cases; in rest it appears soon after birth]
2. Lower end of tibia appears at 1 year

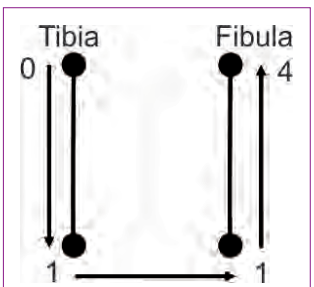


Fig. 3.12: A simple way to remember ossification centers of tibia and fibula [**visual Memory Aid – 14**]

Table 25: Major ossification centers appearing during IU life with **Memory Aid – 26** The figures represent IU month [IUM].

CSI CAST TAKES SAFE CUSHION **C**→Clavicle→1½ IUM [followed by maxilla a few days later] **S**→Shafts of long bones→2 IUM **I**→Ischium→3 IUM [If one reads backwards from "I" it reads as "**Isc**", first 3 letters of "**ischium**". It also reads as "**SC**", **S**econd **C**ervical, which appears at **4th month**]
Second **C**ervical Vertebra→4 IUM
Ca→Calcaneus→5 IUM **St**→Sternum (manubrium)→**6** IUM [by a previous mnemonic, manubrium sterni is taken to come at 5. It is okay to consider it as **5-6** months now]
Ta→Talus→7 IUM [Age of viability] Kes→It stands for nothing
Sa→Sacrum→**8** IUM **Fe**→Femur (lower end)→**9** IUM
Cu→Cuboid→**10** IUM [or just before birth]

- (I) **Sectioning** - Place sternum flat on a wooden board and cut with a cartilage knife in its long axis in midline. **1-5 ossification centers** are seen depending upon the age of fetus. (II) **Visualizing against bright light** – Alternatively hold sternum against bright light. Centers of ossification will appear as dark spots within the bone [**Fig …**]. [**Table 25**]. (b) **Lower end of femur and upper end of tibia** – (I) Leg is flexed against the thigh (II) A transverse incision is made in the knee joint (III) Patella removed (IV) End of femur pushed forward through the wound (V) Make a number of parallel cross-sectional incisions through the epiphysis starting from the articular surface and continuing till diaphysis is reached. Look for brownish red centers (VI) *Diameters of ossification center* - (1) At 36 wks, when the center appears, it is pinpoint in size (2) At 37-38 wks - diameter is 4-5 mm (3) At full term – diameter is 6-8 mm (7) Examine upper end of tibia in same way (c) **Tarsals** – (I) Grasp foot behind the heel in left hand, with toes pointing towards the dissector (II) Make incision between the interspace of 3rd and 4th toes backwards through the sole of the foot and heel (III) Centers for talus and calcaneum should be seen as brownish red spots; if not seen, make thin incisions on either side till they are visible. The upper one is talus; lower is calcaneum. If not visible, it is interpreted as not appeared. (ii) **By staining - Alizarin Red** is used as a marker to stain centers of ossification. (d) Skull (1) **Number of bones:** (i) *Calvaria* [8]– 1 frontal, 2 Parietal, 2 temporal, 1 occipital, 1 ethmoid and 1 sphenoid. (ii) *Face and jaw* [14] – 2 inferior nasal concha, 2 lacrimal, 2 maxillae, 2 nasal, 2 palatine, 2 zygomatic, 1 mandible and 1 vomer. (2) *Fontanelles:* (i) Posterior fontanelle closes by 2 months (ii) anterior by 2 years. (iii) Two postero-lateral fontanelles [mastoid fontanelles] close shortly after birth (iv) Two antero-lateral fontanelles [sphenoidal fontanelles] at 6 months.

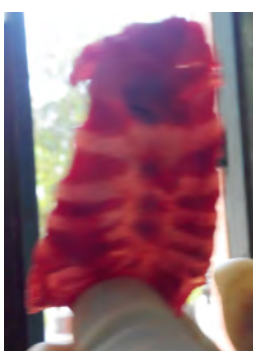


Fig. 3.…: Sternal centers visualized against bright light. Centers for MS, S1 appeared [>5 m]; S2 and S3 appeared [>7 m]; S4 not appeared [<10 m]. Age 7-10 IU months. Pl also see (**Fig. 3…**), where sternum is sectioned in the middle to see centers – another technique to see sternal centers. Credits to Pawan Mittal

Memory Aid 27 – Closure of fontanelles.

Fontanelles close from back to front.

3. **Ossification:** (i) *basiocciput unites with basisphenoid* – 18-21 y (ii) *Condylar portions of occipital bone* unite - (a)with squama – 3 y (b)with basiocciput – 5 y. (iii) *Inferior nasal concha* - ossifies from a single center, which appears at 5th month of IUL in the lateral wall of the cartilaginous nasal capsule.
4. **Suture closure:** [**Fig 3.16**] (i) Skull sutures obliterate earlier in males than in females. (ii) Suture closure begins at the inner table [5 y earlier] and progresses outwards. (iii) Metopic suture – closes at 3 y. Persists in 5-10% cases (iv) Other sutures - **Table 26**.

Table 26: Ages of skull suture closure.

Suture	Commencement	Halfway closed	Completion
Spheno-occipital synchondrosis	-	-	20
Coronal suture	25	30	40
Sagittal suture	25	30	40
Lambdoid suture	25	30	45
Pterion	40	-	65
Masto-occipital suture	45	-	80
Asterion	-	-	50

(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....: (1) Common tattoo used in India, with names of deities. Pl note that it can often reveal religion. (2) Tattoo on forehead, a rather uncommon site.

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** - 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. **UV (Blacklight) Reactive Tattoo Ink** - Blacklight tattoos will not glow in the dark but glow under UV light. 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. (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 LN and note color→If an abnormal color [eg black, red] is noted, tattoo presence confirmed. Helpful also if the person had *intentionally removed tattoos* during life. The color should be mentioned in the pm report [please correlate with **ch 5**→description of an organ].

Fig. 3....: (1) Heat applied by placing a spirit soaked burning cotton over the area can make the tattoo visible (2) Close up of tattoo made visible by this technique.

5. Complications

Abscess, AIDS, allergic reactions to tattoo inks, bruise (if bv is punctured), erysipelas, fungal infections, hepatitis, gangrene, leprosy, septic inflammation, syphilis, tetanus, tuberculosis may occur. A few cases of burns on black tattoos caused by MRI scans have been documented. Black ink commonly contains iron oxide; the MRI scanner causes the iron to heat up causing burns.

7. **Before recovery of body:** (i) *Photography* - With the body *in situ* photograph it→Clear the body surfaces with the use of a soft brush or whisk broom→Photograph the body *in situ* again (ii) *Drawing* – of the grave as well as body or skeleton should be made now. This enables various measurements to be recorded on the drawing, eg height of body, extent of flexion or extension of limbs etc, grave's distance from notable landmarks etc.
8. **Recovery of body:** (i) *If body is with a coffin* – Note condition of coffin; whether eaten up by worms, sodden, soggy, damaged, worn out, peeling on the outside etc. Whether dry or filled up with water. *If filled up with water, remove as much as possible, preserve, label and send for toxicology.* Now lower a plank or strong PVC sheet to the level of earth on which coffin rests→Gently lift coffin on to the plank or PVC sheet→Remove from grave→Transport entire coffin to mortuary (ii) *If body is without a coffin* - (a) *If decomposition is not advanced* – Use same procedure to remove body as above. Body is gently lifted and placed on to the plank or PVC sheet (b) *If decomposition is advanced* – There is possibility of body disintegrating and breaking away as soon as it is attempted to be lifted. So an alternative procedure is followed. Dig down beside and beneath the body→Insert a wooden plank under the body→Lift up the wooden plank. (c) *If body is skeletonized* – Use same procedure as above [advanced decomposition]. In addition, after removal of skeleton, remove earth from below and sides of the body and sift it in a finely-meshed screen. This can often recover **smaller bones** [*carpals and tarsals , epicondyles, epiphyses, phalanges, sesamoid bones, teeth, tuberosities*] as well as **objects** [*bullets, gall stones, kidney stones, medical prosthesis eg nails, plates, tips of knives and arrows etc*].
9. **Identification** – Body should now be identified by close relatives and friends either from features [if intact] or from clothes, objects kept within the grave, appearance of coffin etc.
10. **Note** – condition of body, burial clothes
11. **In suspected poisoning:** (i) Collect all personal effects, clothings, nails, hair, bones etc and send for toxicological analysis (ii) Collect samples of earth [about 1kg] that are in actual contact with body and also from above, below and each side of the body. Also from a site well away from the grave [**Fig 7.1**]. (iii) Also preserve samples of burial clothes and coffin to preclude the possibility of contamination from these sources.
12. First autopsy or second autopsy – please see **ch 5**.

Fig. 7.1: (1) Digging the grave is done early in the morning in the presence of doctors, executive magistrate and police. (2) Measuring the depth of grave (3) Collection of soil from above the coffin for toxicology. Later it would be collected from below and from the sides of the coffin too, as well as from some distance away from coffin as mentioned in the text (4) After the grave has been dug up to the level of coffin, it is safely lifted up with the help of ropes.

These are yellowish in color in the beginning, but as dust settles in, they become dark red (Fig 9.1) or later even black. Sometimes they may be bright red, giving rise to an artifactual appearance of subconjunctival hemorrhages.

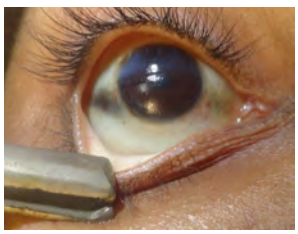


Fig. 9.1: *Taché noire de la sclérotique*. Please note black triangular area on sclera. The base sits on the outer rim of cornea and the apex touches the outer canthus. A similar triangular area on the medial side is less pronounced.

The eyes look sunken and are softer after death due to fall of Intraocular Pressure (IOP). *Normal IOP during life is between 10 to 20 mm Hg [Average – 15 mm]*. One hour after death, it is **12 mm** Hg; after 2 h, **10 mm Hg**; after 3 h, **8.5 mm Hg;** after 4 h, **7.5 mm Hg** and after 8 hours, **5 mm Hg**.[1] Measurement of IOP can thus give a rough estimate of the **pm interval** [PMI].

Memory aid 1 - IOP after death [in mm Hg]

IOP figures after death are **same as figures for circumferences of heart valves**. Pl see mnemonic for heart valves in Appendix [*weights and measurements of organs*]

Value	IOP [in mm Hg]	Circumference of heart valve [in cm]
15	At death	-
12	After 1 hour	Tricuspid
10	After 2 hours	Mitral
8.5	After 3 hours	Pulmonary
7.5	After 4 hours	Aortic
5	After 8 hours	-

4. Pupils

Normal pupil size during life is between **1-8 mm**. *No appreciable change in pupil size occurs after death.* Pupils react to atropine and pilocarpine, for about 2 hours after death (till molecular death occurs). If pressure is applied by fingers on two or more sides of the eyeball, the pupil may become oval, triangular or polygonal.

1 Balci Y, Basmak H, Kocaturk BK, Sahin A, Ozdamar K. The importance of measuring intraocular pressure using a tonometer in order to estimate the postmortem interval. *Am J Forensic Med Pathol*. 2010 Jun;**31(2)**:151-5.

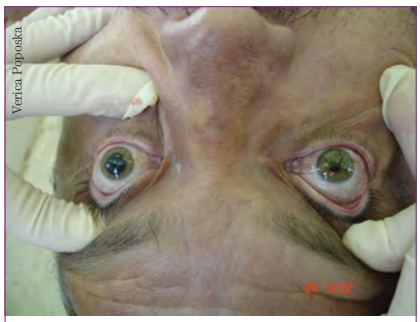


Fig. 9.…: One hour after death, atropine is instilled in the left eye and pilocarpine in the right. Please note mydriasis in the left and miosis in the right eye.

5. Retinal vessels

1. **Segmentation of the retinal blood columns** – Occurs immediately after death. On ophthalmoscopic examination: (i) the continuous blood column in the retinal blood vessels breaks up into small segments, which then collide with each other.
2. **Color of retina** – Retina becomes pale after death, and becomes more and more pale as time of death ↑s. Disk outline becomes hazy after a few hours.

6. Biochemical changes

During life, K^+ concentration is low in the vitreous humor but much higher in peripheral tissues such as retina. After death K^+ from peripheral tissues starts diffusing in the vitreous raising its concentration.

C. Cooling of the body (Algor mortis)

Cooling of the body *[Algor mortis* (L. *Algor*, cold; *mortis*, death*), chill of death] occurs after death since all metabolism comes to a stop*. **Salient features:**

1. **transfer** - During life heat is constantly transferred from one body part to other *by conduction as well*

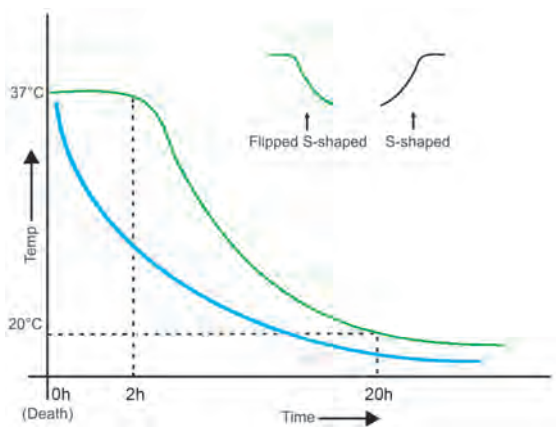


Fig. 9.2: Sigmoid or "Flipped S" shaped, double exponential cooling curve. Pl observe the initial plateau for 2 h. This happens because heat continues to generate for first 2 h after death. This is in contrast to the true Newtonian curve of cooling, which is single exponential (shown in blue).

incapacity (iii) Severe injuries to brain →(a) victim may survive rarely although the extent of damage is expected to have caused instant death. (b) May remain unconscious for several minutes before finally succumbing to injuries (c) Death may occur with no obvious naked eye changes to the brain (iv) Single stab wound of skull and brain→May not be immediately fatal, and victim may walk, run or do some other voluntary activity before death.

XII. ANTEMORTEM V POSTMORTEM WOUNDS

1. **AM v PM wounds** – [L. *ante*, before; *post*, after; *mortis*, death]. PM wounds may be caused by animals [dogs, cats, rodents etc], deliberate attempts to confuse police, mutilomaniacs, ***lust murderers*** [**ch 25**] etc.
2. **Differences** - In many situations, it becomes vital to know if injuries are antemortem [AM] or postmortem [PM].
3. **Diff** - Table 8.
4. **Perimortem wounds** [L. *peri*, around; *mortis*, death] – *Wounds caused during supravital period* [**ch 8**]. Since molecular death has not occurred, cellular reaction and enzyme histochemistry may be +ve. However since heart has stopped, spurting etc may not be present.

Fig. 8.4: (1) Gnawing by rodents **(2)** GPM Mutilation by dogs. Typical PM injuries. Note absence of vital reaction. Projecting soft parts, eg nose, eyelids, ear lobes, penis etc are chosen. **(3)** GPostmortem removal of auricle by assailants for golden ear rings. Please note absence of bleeding and other vital signs excluding antemortem injury. Postmortem gnawing by rats is excluded because much softer eyes are unharmed, as well as nose and lips.

XIII. MEDICOLEGAL QUESTIONS ON TRAUMA

Following questions on trauma are frequently asked by lawyers in courts.

A. Can there be no signs of trauma externally, yet internal injury may be so great as to cause death?

Yes, this is possible. In blunt injuries to abdomen (pliable body surfaces), there may be no external signs of trauma, yet liver and spleen may be damaged, and cause enough bleeding to cause death.

B. How would you quantify degree of trauma?

This is mostly subjective. Trauma is roughly estimated as mild, moderate, severe and extreme.

C. What are the situations in which acceleration-deceleration forces play a vital part?

1. Vehicular accidents [**ch 18**]
2. Shaken baby syndrome [**ch 27**].

D. Can the wounds be altered from their original appearance?

Yes, it is possible both in the living and in the dead (i) **In the living**: (a) **by medical treatment**- Kennedy phenomenon (please see chapter on firearms) (b) by healing (ii) **In the dead**: (a) by resuscitative measures (b) by postmortem predators (insects, animals) (c) by decomposition.

E. Is it possible for a person to die during altercation?

Yes, if the deceased already suffered from heart disease. Sudden altercation may ↑BP and heart rate. If the coronaries are already thrombosed, it may precipitate a subintimal hemorrhage leading to death.

F. Were more than one weapon employed in this case?

(i) **Gang wars and group fights** - Generally several people attack with different weapons. Different weapons would produce different injuries, which would enable the pathologist to opine that more than one weapon were used (e.g. if an incised wound and a bruise are present on the body, it can be opined that both a sharp and a blunt weapon were used) (ii) **Suicide** – If a suicide fails by one method (e.g. cutting wrists), he may employ a firearm to end his life. Presence of incised and firearm wounds would indicate, two different weapons.

4. ***Cut laceration*** *is produced by heavy cutting weapons [axe, boat propeller, bush knife, chopper, hatchet, industrial and farm machinery, lawn mower blade, machete (a large heavy knife used for cutting sugarcane), meat cleaver] wielded with a tremendous amount of force.* (i) It may best be conceived as a "cross" between a true laceration and a true incised wound. The edges of the weapon do cut the skin, but since the edges are heavy, they crush and bruise the margins of the wounds too. (ii) The wounds show an ugly, wide gape (iii) The underlying bones are frequently fractured, or show deep grooves or cuts. (iv) Also called **chop wounds.**

5. ***Avulsion*** *is produced if grinding compression [eg fast moving tire of a heavy motor vehicle (eg run over inj;* ***ch 18****), or a rotating heavy industrial machine], is applied, causing avulsion (separation) of large areas of skin from its underlying attachments and is lost* (Fig 12.15). (i) Wound is devoid of any overlying skin. (ii) also known as **flaying**. (iii) Underlying muscles are heavily bruised. Table 8 summarizes various types of laceration and how they are typically caused.

Fig. 12.….: A cut laceration showing a deep cut on the underlying mandible.

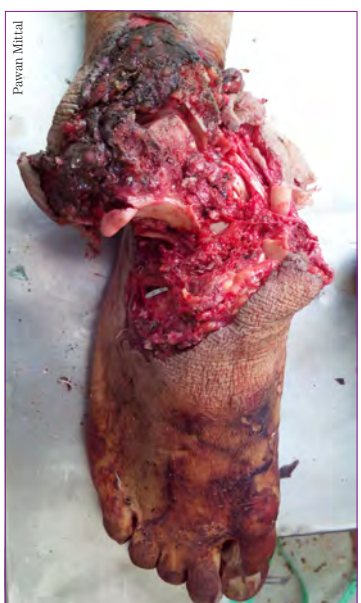


Fig. 12.….: An avulsed laceration of foot.

Table 8: Different types of lacerations and how they are typically caused

S.No	Type of laceration	How typically caused
1.	Tear	Any heavy blunt instrument (e.g. a hockey stick or a cricket bat)
2.	Split laceration	Any heavy blunt instrument, on an area of body which has some bone directly underneath with a scanty layer of intervening tissue (e.g. scalp)
3.	Stretch laceration	A heavy force acting on the body in such a manner that a portion of skin is overstretched
4.	Cut laceration	Heavy cutting weapon with an edge which may not be too sharp
5.	Avulsion	Heavy weights (e.g. a truck) passing over the body, causing large areas of skin to be avulsed and lost. Also called "flaying"

C. Age of lacerations

1. Age determination is difficult unless clear signs of healing are present [eg Fibroblasts, Granulation tissue, Organizing infiltrate].

D. AM and PM lacerations

1. Antemortem lacerations would show (i) Bleeding (ii) Blood-staining of margins (iii) Bruising (iv) Eversion (v) Gaping (vi) Vital reactions. PM lacerations would show none of these.

E. MLI

1. **Age of lacerations** – (i) *Fight* - indicates time of struggle (ii) *Disputed pregnancy* - age of perineal lacerations indicates the possible date of delivery [please see **ch 24** for more details]
2. **Combinations of lacerations with abrasions and bruises** – Abrasions, bruises and lacerations may be seen together. Each injury may be produced by different blows by the same weapon, or they may be produced at the same time (as in explosions). Punching (with a fist), kicking and stomping (with a shoe wearing foot) can produce all three injuries with different blows.
3. **Extraneous material** – may be present in lacerations (eg dirt, dust, grass, grease, sand, stone particles). This may connect the injuries with the place of occurrence
4. **Firearms [Lacerations produced by]**– By *pistol whipping* [*buffaloing*]. (i) It is the act of holding a handgun by its barrel, and clubbing the victim with the butt, in effect using it as a blunt weapon. (ii) *location* - Predominantly on forehead.

off, and sticks to the inside of barrel (**lead fouling** of the barrel). To prevent lead fouling, these bullets are lubricated with grease or some other lubricating compound. This is contained in **knurls** (Fig 13.12). Copper gilding is used extensively in .22 high-velocity rimfire ammunition.

2. **Jacketed:** (i) Two types (a) **F**ull **M**etal **J**acket (FMJ); **T**otal **M**etal **J**acket (TMJ) bullets - Bullet nose is fully encased by a jacket; base is base (b) Full Metal Case - Both the base and the nose of a bullet are fully encased by the jacket; typical of military bullet where expansion is not desired (ii) used in semi-automatic pistols. (iii) Have a lead or steel core covered by an outside jacket. It is made up of gilding metal, gilding metal-clad steel, cupro-nickel (copper and nickel), or aluminum. (iv) Jackets generally range from **0.0165 to 0.030 in.** in thickness. (v) *Advantages* – (1) Imparts additional strength to the bullet. Help to prevent the bullet from breaking apart (2) higher muzzle velocities are achieved (3) Prevent lead fouling as well as jamming of the gun that occurs if a large number of lead bullets are fired [due to lead deposition on barrel and other areas of the gun] (4) prevents damage to bores from steel or armor-piercing core materials. (vi) *Deformation* – Full metal jacketed bullets exit undeformed; unjacketed bullets show deformations (vii) *Armor-piercing bullet* [APB] - (a) APB is a special jacketed bullet fired from small arms [Armor-piercing rifle and pistol]. They are meant to penetrate bullet-resistant vests [commonly known as bulletproof jackets] and other tough targets like automobile doors.
3. **Partially jacketed** [syn - **S**emi-**J**acketed, SJ bullet, soft-nosed bullet, Soft-point bullet – Jacket is left open at the tip, exposing some of the lead inside. Exposed lead expands upon impact.

(c) According to special purpose they serve

1. **Dum-dum bullet** (Fig 13.19): (i) A special type of expanding bullet [please see below] (ii) A British military bullet developed for use in India during the late 1890s. (iii) Jacketed .303 bullet with the jacket nose open to expose its lead core. (iv) Immediately balloons out on impact with the target thus increasing the damage. (v) The phrase 'Dum-Dum' is now taken to include any soft-nosed or hollow pointed bullet.

Fig. 13.19: Dum-dum bullet after firing and recovery from target. Note expansion (mushrooming).

2. **Expanding bullet:** (i) *Expands on impact, increasing in diameter to produce a more serious wound*. (ii) Also known as **mushroom bullet**, because of the mushrooming that occurs on impact. (iii) The two typical designs are the (a) **hollow point bullet** [please see above] and the (b) **soft point bullet**. (c) Dum-dum bullet is a special type of expanding bullet
3. **Frangible bullet**: (i) Made of bonded powdered iron [or lead], which fragments on impact. [Fig 13.23]. (iii) *Caliber* – mostly .22 (iv) *Normal use* – target practice by police, shooting galleries, to stun cattle (v) *Recovery and matching* - of fragments is difficult because of fragmentation (vi) *They do not ricochet* [please see below] (vii) If bone is penetrated, they are usually recovered in an eroded state. (viii) *D/d* - An x-ray would reveal several fragments which look like small pellets. One may wrongly conclude that the victim was hit by a shot gun (iv) **Tracer bullet** - Special kind of bullets that contain a powder in their base that burns very brightly during their flight. This enables the shooter to follow the bullets' trajectories, so he can adjust his aim accordingly. It is used both in ground as well as aircraft guns.

Fig. 13.....: Tracer bullets from .50 caliber machine gun. Pl note the bright trajectories of bullets.

(d) According to Forensic scientist's point of view

1. **Crime bullet** - *Bullet with which crime is committed*. This bullet is recovered from the body of the victim, and is produced in court as evidence; hence also known as **evidence bullet**. *Crime bullet is fired by the criminal*
2. **Test bullet** - *Bullet that is fired from a suspect weapon by the forensic scientist, so he can compare the markings on the bullet with that of the crime bullet*. If the markings match perfectly, the suspect weapon is the actual weapon used for crime. Both crime and test bullet when presented in the court as evidence are known as **exhibit bullet**.

wound, and is much more common in high velocity missiles [muzzle velocity **[600–800 m/s]**. (c) In high velocity missiles, temporary cavitation produces an enormous force within the brain, which may also cause expulsion of brain [please see "**cavitation of brain**" above]. (d) May be seen in exit wounds of other firearms also eg pistols, revolvers and rifles. (e) Named after German surgeon **Rudolf Ulrich Krönlein** (1847-1910) who first described it in **1899**. (ii) **Eyeball** – may be blown out of socket.

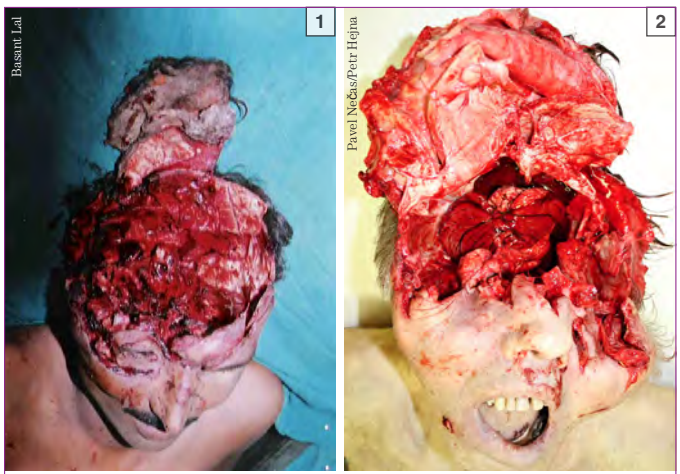


Fig. 13.…: Krönlein shots. Note virtually complete expulsion of brain from both skulls, which have exploded outwards. In 2nd pic, cerebellum may be seen lying within the skull cavity.

4. Special effects

a. Billiard ball ricochet effect

Billiard ball ricochet effect *is excessive spreading of shot pellets due to passage through an intermediate target such as a glass window.*

b. Balling or welding of shot

Balling *[syn, welding]* ***of the shot*** *is exactly the reverse of billiard ball ricochet effect.* **Salient features**

1. Here the shots stick together and travel for large distances as a single mass. Spread is thus less than what it should be and causes the forensic pathologist to estimate less distance than actual.

C. Wounds from Automatic pistols and revolvers

1. Entrance wounds

a. General

1. **Components of an entrance wound** - from center to the periphery are as follows [Fig 13.33].

Memory Aid – 5 [Zones of rifle firearm entry wound]

Mnemonic 1. **W**ild **G**oat **A**te **C**hara - **W**ound defect, **G**rease collar, **A**brasion collar, **C**ontusion collar [from within outwards]
Mnemonic 2. **CAGE** [from without inwards]. **E** represents **E**ntry wound. Either may be remembered according to comfort level.

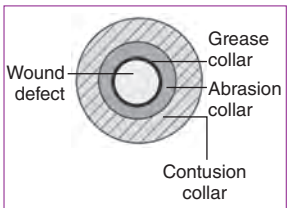


Fig. 13.33: A typical entrance wound from a rifled firearm. Please co-relate with Memory Aid 1.

2. **Revolvers and automatic pistols** - cause similar wounds [both entry and exit]. However penetrating power of pistol bullets is much greater because of greater velocity. Pistol bullets are also more often coated with hard metal.

i. Wound defect

This is the actual entrance wound of the bullet

ii. Grease collar

Grease collar *[**syn** – bullet wipe, bullet wipe soiling, burnishing, dirt collar, grease ring, lead ring, leaded edge, metal fouling, metal ring] is a ring of grease seen around the entry wound in the form of a gray coloration. It is present at all distances.* **Salient features:**

1. **Cause** – As bullet moves down the barrel, it collects debris and metal from barrel, dirt, gun oil, powder and primer residue, soot etc over it. This is deposited on to the skin, as the bullet penetrates the body. [Fig 13.12].
2. **Less common** – in jacketed bullets
3. If wounded area is covered by clothing, seen on clothing only [importance of examining clothings]
4. **Detection:** (i) Naked eye (ii) IR photography
5. **Wiping** – collar produced by grease can be wiped, but not that produced by metal, which enters inside the skin. The term "fouling" is used in two contexts: (i) fouling of the barrel [please see above], and (ii)fouling of the entrance wound [as mentioned here].
6. **MLI** - may link the entrance wound to a weapon because metallic elements from the grease collar may be linked to those from the weapon.

iii. Abrasion collar

Abrasion collar *[**syn** - Abrasion ring, marginal abrasion] is a ring of abrasion around the entry wound which is present at all distances.* **Salient features:**

4. **Site** – May be anywhere, but usually over extensor surfaces and joints
5. **Size** – Several cm in length
6. **D/d** – May resemble lacerations [rarely even incised wounds] [Table 3].

Table 3: Differences between heat rupture and lacerated wound

S.No	Feature	Heat rupture	Lacerated wound
1.	Cause	Exposure to intense heat	Blunt force
2.	Site	Fatty tissue	Anywhere
3.	Bleeding	Absent in wound and surrounding tissues [because heat coagulates blood in the vessels]	Present
4.	Margins	Absence of bruising and vital reaction	Bruising and vital reaction present
5.	Floor	Shows intact nerves and vessels	Shows lacerated nerves and vessels
6.	Associated findings	Of burns	Of trauma

8. Hair

1. **Singed** and **burnt**, but may be spared in armpits.
2. In lesser degree of burns, ends may be *bulbous* or *clubbed*.
3. **Color** – Color of hair may sometimes reveal the temp reached. (i) *Gray hair* become brassy blonde or even golden at 250°F (ii) *Brown hair* become slightly reddish when exposed to 400°F [for 10-15 min] (iii) *Black hair* do not change color on exposure to heat.

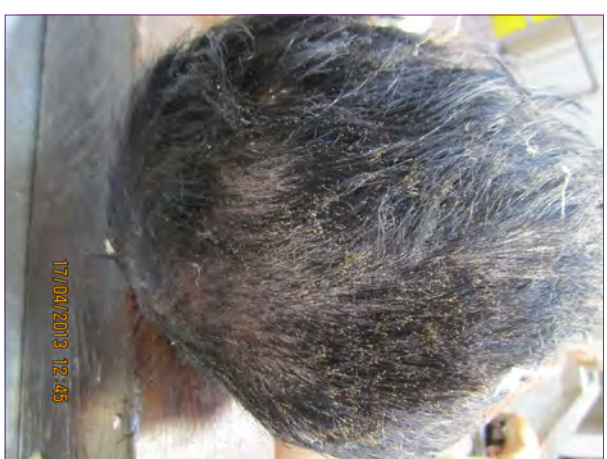


Fig. 14.…: Brassy blonde hair in burns

9. Miscellaneous findings

1. **Identification marks** – eg moles, scars and tattoos may be destroyed in 5th and 6th degree burns.
2. **Abdominal wall** – may show rupture. Caused by (i) burning of the abdominal wall (ii) expansion of gases within the intestines exerting pressure on weakened abdominal wall. Intestines may be seen protruding through this gap, which may themselves be ruptured rarely.
3. **Anus** – Gaping may be seen because of shrinkage of the perianal tissue. This may be misinterpreted for sodomy or homosexuality
4. **Pre-existing lesions** – may become smaller and change in shape due to shrinkage of the skin. For example, originally slit like stabs may assume a circular shape. Lesions may sometimes migrate toward the center of the thermal damage.
5. **Complete destruction** - If the flame is unchecked, the body is reduced to a shapeless, coal like mass and finally to a heap of grey and yellow ashes. These are known as **cremains.**

B. Internal

1. Heat hematoma

Heat hematoma *is the collection of blood in the extradural space due to excessive heat*. **Salient features:**

1. **Degree of heat required** – occurs mostly when the heat is enough to cause *charring of the skull*.
2. **Appearance:** (i) **Gross** - ***Honeycomb appearance*** due to presence of air bubbles inside (ii) **Color** - light chocolate color, or pink if blood contains CO. (iii) **Consistency** - **Soft and friable** [bubbles] (iv) **Thickness** – 1.5 mm to 1.5 cm. Volume up to 120 ml. (v) **Differences from antemortem EDH** – please see Table 4.
3. **Adjacent area of brain** – is hardened and discolored (due to heat)
4. **Distribution** – of heat hematoma follows closely the distribution of the charring of the outer table of skull. Found also along the superior sagittal sinus. *Most common site is* ***parietotemporal region***.
5. **Mechanism of development** – Not clear. Could be expansion of blood in the diploe (due to heat) and rupture of dural venous sinuses.

2. Heat fractures (Thermal fractures)

Heat fractures *are postmortem fractures produced by excessive heat*. **Salient features:**

1. **Appearance** - Bones are burnt and assume a gray-white color. Usually shows a fine superficial

(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 19.4). **Granny** [syn, **lubber's**] and **reef** [syn, **flat, square**] knots are rather common. But some other special types of knots are exceedingly rare and may sometimes give away the profession of the person. Even simple knots can reveal predilection or hobby of the person, eg a **sailor** or a **scout** may use a **reef knot** rather than a **granny knot**. (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 19.5) [for more on ***Forensic knot analysis***, please see **chapter 30** – Forensic Science Laboratory].

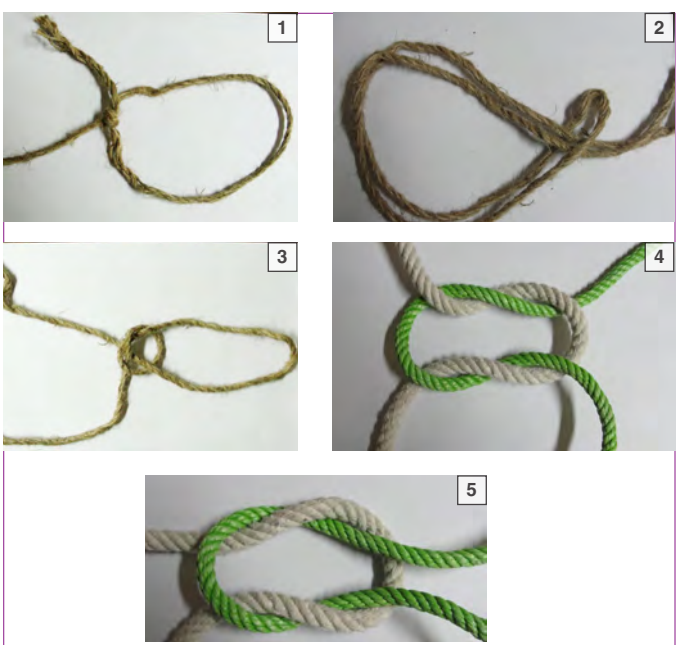


Fig. 19.4...: (1) Fixed knot **(2)** & **(3)** slip [syn, quick release, running, slipped loop] knot – two different types. Less common are **(4)** Granny knot and **(5)** Reef knot. In granny knot, the crossings are opposite, while in reef knot they are same. Pl see dotted circles.

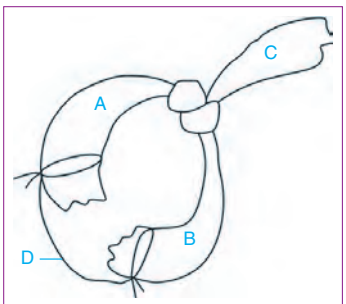


Fig. 19.5: Method of securing the knot. Limbs A and B form the actual noose around the neck. C is the free end of the ligature. If A and B are not secured with a string D, it would be difficult to demonstrate in court that A and B indeed formed the noose. Any two of the three limbs would appear to consist of original noose.

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 19.6]
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. If the head has been hanging on one side, it may be one-sided [Fig ????].

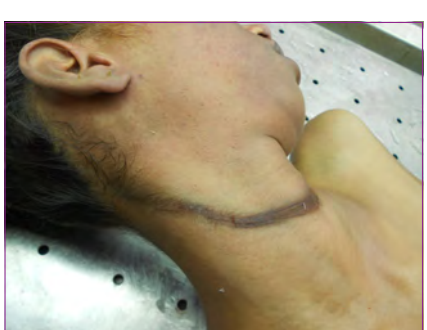


Fig. 19....: Classical oblique ligature mark in hanging.

3. **Manner** - (i) Suicidal (ii) Homicidal – (a) *Unwanted infants* - may be killed by this method [**ch 27**] (b) *In adults* - Robbers may stuff a cloth inside mouth of a *chowkidar* to keep him silent. There is simultaneous tying of hands (to prevent removal of gag) and legs (to prevent running away). The cloth may gradually swell up and cause gagging. Strictly speaking it is not homicidal, as the *intention of robbers* was only to keep him silent. Generally more than one person is required to gag a healthy struggling adult individual [one needed to restrain, and another to gag]. (iii) Accidental – (a) *False dentures* – may impact throat during anesthesia (b) *Dental procedures* - Gagging [mostly nonfatal] commonly occurs during dental procedures, such as making a maxillary impression. Causes (I) Tactile stimuli - dentist's fingers or instruments contacting the oral mucosa or even by (II) Non tactile stimuli - Patient seeing the dentist or remembering a previous dental experience. (c) *Injuries to nose and mouth* – Blood may seep to the back of the throat and may clot, producing an "artificial gag" of clotted blood. [if it trickles down trachea, it would be choking] (d) Breast pushed too far in the mouth of a suckling baby (e) *During sexual activities* [Fellatio]
4. **Cause of death:** (i) Asphyxia [most common] (ii) Reflex vagal inhibition [rarely]
5. **PM appearances:** (i) classical signs of asphyxia (ii) Cloth must be removed and sent for forensic examination [fibre, manufacturer or laundry's mark, saliva etc].

E. Choking

Choking *is a form of asphyxia caused by an obstruction within the air-passages* [Fig 19.27]. **Salient features:**

1. **Manner of death** - Choking is almost always accidental.
2. **Level of blockage** – foreign body gets arrested at or just below the vocal cords. May produce an inflammatory reaction with edema.
3. **Seen most commonly in** – People whose power to swallow or masticate is severely impaired, eg (i) very young (ii) very old (iii) psychiatric patients (iv) sick and infirm
4. **Common circumstances** – (i) laughing or crying simultaneously with food intake (ii) ingestion of alcohol
5. **Objects causing choking** – Any small object which can enter trachea can cause choking. Most common objects are (i) Balloon [when trying to inflate it] (ii) Battery (iii) Button (iv) Cloth [esp after ENT operations, eg tonsillectomy] (v) Coin (vi) Corn (vii) Cotton (viii) Dentures (artificial) (ix) Fish (live) [as during some unorthodox treatments] (x) Fish bone (xi) Food [eg loaf, meat, milk] (xii) Fruit stone (xiii) hairpin (xiv) Leaves (xv) Marbles (xvi) Mud (xvii) nails [during biting](xviii) Onion (xix) Peanut butter (xx) pen cap (xxi) Potato (xxii) Rag (xxiii) Rice [Fig. .....] (xxiv) ring (xxv) Rubber balloons (xxvi) Rubber teat (xxvii) Seed (xxviii) Tooth (broken, extracted by a dentist).
6. **Choking by vomit** or **regurgitated food** - (i) during *anesthesia* (ii) *insensibility* from any cause eg poisoning (iii) during *rape* or violent sexual intercourse(iv) *Head injury* – irritation of brain causes vomiting, which may be inhaled due to unconsciousness (v) during fit of *epilepsy* (vi) *Infants* – usually regurgitate clotted milk after a meal and choke on it (vii) *Tranquilizing drugs* – may suppress gag reflex and may cause choking especially psychiatric patients
7. **Choking by Blood** – (i) Facial injuries during blunt trauma as in a fall, fight, vehicular accident etc [broken nose, dislodged teeth, laceration of lips and gums] (ii) ENT operations [in *Dr. Suresh Gupta v Government of NCT of Delhi 2004*, the patient choked during an ENT operation – **ch 2**].
8. **Choking by Gauze piece** - (i) during operations
9. **Natural diseases** - (i) Diptheria (ii) growths in epiglottis larynx and bronchus (iii) *H. influenzae* infection in children (iv) hemoptysis in pulmonary tuberculosis (v) hemorrhage into trachea (vi) infectious mononucleosis (vii) pharyngeal abscess (viii) rupture of aortic aneurysm in air passages and (ix) a tuberculous gland eroding in a bronchus and prolapsing in its lumen.

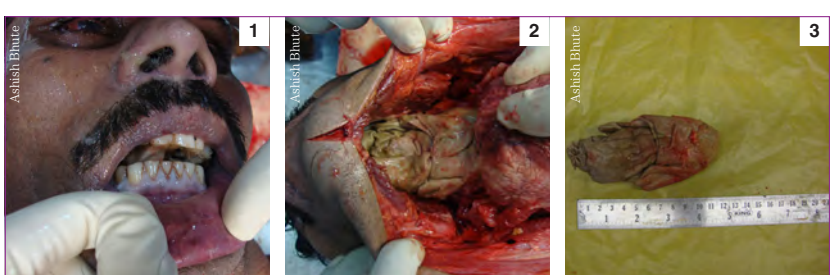


Fig. 19....: (1) Case of accidental gagging. Gauze piece can be seen in the mouth behind teeth **(2)** On reflecting the tongue, the piece can be seen extending back upto epiglottis, completely blocking airways **(3)** Gauze piece taken out. It is a large piece measuring 12x6 cm.

Chapter 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.

Fig. 20.1: Death due to starvation. Note the general emaciation, especially sunken eyes and cheeks

Chapter 30

Forensic Science Laboratory

I. LOCARD'S EXCHANGE PRINCIPLE

Locard's exchange principle *states that when any two objects come into contact, there is* ***always*** *a transfer of material from each object on to the other*. This principle is often used as a sure proof of crime. The principle was first enunciated by **Edmond Locard** (1877–1966), often referred to as the Sherlock Holmes of France.

Fig. 30.1: Edmond Locard

II. INSTITUTE OF FORENSIC SCIENCE - ORGANIZATION

A comprehensive **Institute of Forensic Science** should comprise of **3** categories of service (i)**Clinical** (ii) **Pathological** and (iii) **Laboratory services**. In addition it should have several other facilities such as (iv)**exhibit room** (where exhibits brought by police, e.g. blood stained weapons, viscera etc) are stored (v)**stores** (for equipment, chemicals etc) (vi)**workshop** (for training) and (vii)**library**.

A. Central and State FSLs

1. Central FSLs are funded by central government.
2. **Central CFSLs** (Central Forensic Science Laboratories) – **7** in India [**Bhopal, Chandigarh, Guwahati, Hyderabad (Telangana), Kolkata, New Delhi, Pune**]. Of these, **6 CFSLs** [except the one at ND] are under the **Directorate of Forensic Science Services** (DFSS). The one at New Delhi is under CBI [Address: Block No.4, CGO Complex, Lodhi Road, New Delhi-110003]. In addition there is one **institute of serology**, located at **Kolkata**. It was established in 1912 starting with Forensic Serology as its core activity, but now has branched out into different field of Serology. CFSLs meet the demand of police from all over India.

B. Interpol

Interpol (**International Criminal Police Organization**), with headquarters in Lyon, France, was established in **1923**. It facilitates international police cooperation. **190 countries**, including India are its members. It has a comprehensive forensic science laboratory.

III. FORENSIC SCIENCE LABORATORY – INDIVIDUAL SECTIONS

The main functions of forensic science laboratory are: (i) to reconstruct a crime and (ii) to analyze trace evidence (blood, hair, fiber etc from victim, accused and the scene of crime and to link the three together. This work is carried on by different sections. The student is advised to co-relate each section with appropriate chapters in the book.

A. Lie Detection

Following techniques are mostly being used for lie detection.

1. Tests for Lie Detection

a. Brain mapping (Brain fingerprinting)

Brain mapping *for* ***brain fingerprinting*** *is a forensic science technique that determines whether specific information (regarding a crime) is stored in a subject's brain*. Scientific name of the technique is **Memory and Encoding Related Multifaceted Electroencephalographic Response** (**MERMER**). Invented and developed by **Lawrence Farwell** of US in 1990s.

i. Principle

1. **Storage of information in brain** - If the subject has committed a crime, the information would be **stored in his brain**.
2. **EEG responses to crime related information:** (i) If crime related information (eg picture of the crime scene, photograph of victim, motive of crime etc) are presented to the subject on a computer screen, he may **consciously deny the information**, but his brain would recognize the information. (ii) This would elicit **relevant EEG waves** which could be detected. (iii) EEG response to an image, sound etc is known as **event related potential** (**ERP**) (iv) *Memory* is an integral part of ERP. (a) If a subject is shown a familiar image (e.g. inside of an aircraft engine to an aircraft mechanic), his EEG would show **memory related waves** in ERP. Similarly if the

surfaces (iv) Photography and (v) Straw hats [manufacture]

2. **Domestic** - (i) bleach (ii) ink remover (iii) paint, rust, stain and varnish remover
3. **Illegal** - (i) removing writing and signature from papers [eg wills, contracts, bonds] (ii) removing election stain marks on fingers [to enable voting again].

4. Mechanism of action

1. **Locally** – acts as a corrosive
2. **Systemically** –Reacts with Calcium in plasma →Forms calcium oxalates→(i) *Hypocalcemia* (ii) *Precipitation* of calcium oxalate crystals in liver, kidneys, heart and lungs (iii) Excretion of *envelop shaped crystals* [calcium oxalate] in urine.

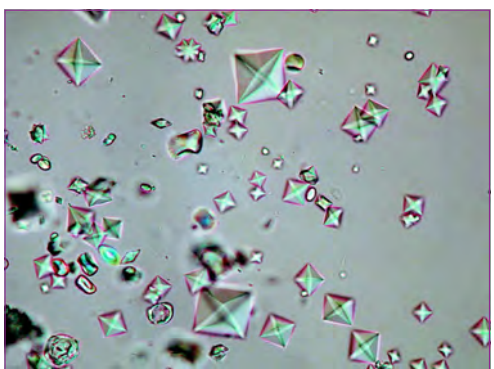


Fig. 33.….: Typical envelop shaped calcium oxalate crystals in urine of a patient with oxalic acid poisoning

5. Signs and symptoms

a. Contact

1. Skin - is rarely damaged. May just be discolored
2. **Mucosa** – of eye, mouth etc may be greatly damaged and may give rise to a "scalded" appearance. Sometimes production of acid hematin may give rise to black color.

b. Ingestion

i. Immediate

1. Burning, sour or bitter taste in the mouth which goes up to the stomach
2. Sense of constriction around the throat
3. Intense thirst
4. Mouth – may appear "scalded" or sometimes black
5. Severe pain – Begins in the epigastrium, but soon radiates all over the abdomen
6. Abdomen is tender
7. Persistent vomiting, eructations and diarrhea. Vomitus contains altered blood ["coffee-ground" appearance] and mucus.
8. Signs and symptoms due to hypocalcemia - (i) Tetany (ii) Numbness and tingling of fingertips and legs (iii) *Chvostek sign* +ve.

ii. Delayed

If patient survives initial poisoning episode, delayed symptoms may be due to **renal failure** [calcium oxalate crystals in kidneys]

1. **Urine** – Scanty or suppressed. Contains traces of blood, albumin and calcium oxalate crystals.

6. Management

1. **Gastric lavage** – If patient seen early, perform gastric lavage carefully with calcium salts [chloride, gluconate, lactate, chalk powder (1.5g of chalk neutralizes 1g of acid), lime water, milk]. Converts acid into insoluble calcium oxalate.
2. **Antidote** – Calcium preparations orally [same as those used for gastric lavage].
3. **Calcium gluconate IV** – 10 ml of 10% at frequent intervals
4. **Parathyroid extract** – 100 units IM in severe cases. Mobilizes Ca^{+} from bones.
5. **Dialysis or exchange transfusion** – for renal failure
6. **Miscellaneous** - (i) Demulcents (ii) Evacuation of bowels – by castor oil or enema (iii) Symptomatic.

7. Fatal dose

600 mg/kg. For a 60 kg human 36 g.

8. Fatal period

1-2 hours.

9. PM appearances

1. **Mucus membrane of tongue, mouth, pharynx, esophagus** - (i) *In case of concentrated solution* - (a) **Whitened** as if "bleached". Similar to a "scalded" appearance (b) sometimes **brown** or **black** due to formation of acid hematin (ii) *In case of weaker solution* - **Reddened** because of irritation
2. **Esophagus** – Mucosa corrugated. Shows longitudinal erosions
3. **Stomach** – (a) *Mucosa* - (i) soft and reddened (ii) Shows punctate erosions (iii) May be black [acid hematin] (iv) numerous dark brown or black streaks are seen running longitudinally along the length of the stomach, often with intercommunicating branches (v) Often entire mucosa is corroded (b)

submucous hemorrhages – in curved lines (iv) *Red velvety appearance* – stomach wall is soft and red like a valvet [Fig 36.2].

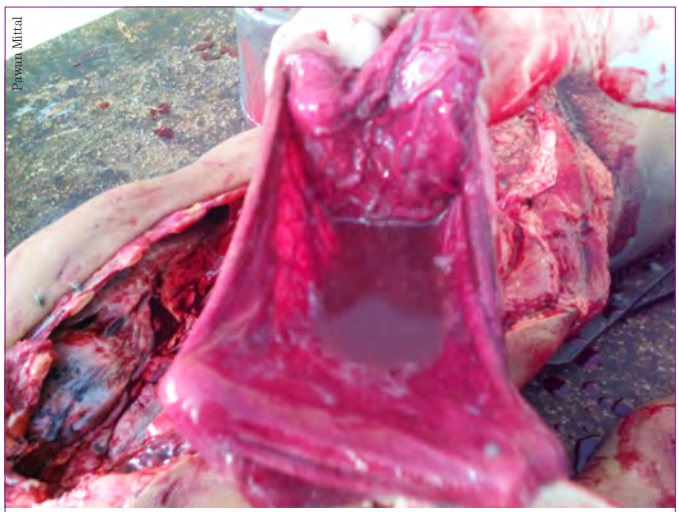


Fig. 36.2: Velvety red stomach mucosa in acute arsenic poisoning.

Groups of petechiae scattered over mucosa. Sometimes large submucosal and subperitoneal hemorrhages. (v) *Pyloric region* - is especially affected. (vi) *If putrefaction has occurred* – yellow streaks are found in the subperitoneal layer of the stomach and to a lesser extent in intestines [As converts to yellow sulphide due to H_2S gas]

3. **Small intestine:** (i) Appears flaccid (ii) *Mucosa* – Inflamed, pale violet. Shows submucous hemorrhages along the entire length (iii) Contains large flakes of mucus with very little fecal matter
4. **Cecum and rectum** – slightly inflamed
5. **Liver, spleen, kidneys:** (i) Congested, enlarged, show cloudy swelling and if survival is for few days, fatty change. (ii) Glomerular nephritis
6. **Hemorrhages** – in all abdominal organs, mesentery and occasionally in larynx, trachea and lungs
7. **Lungs** – congested with subpleural ecchymoses
8. **Heart:** (i) subendocardial petechial hemorrhages [SEPH] of the ventricles. May be seen even in absence of gastric inflammation. (ii) *Conditions in which SEPH are found* - (a) Acute infectious diseases [eg influenza], (b) Heat stroke, (c) Poisonings [Arsenic, Barium, Mercury, Phosphorus] (d) Traumatic asphyxia (iii) if survival is in days – fatty change
9. **Brain:** (i) *Edema* - with patchy necrosis or hemorrhagic encephalitis (ii) *Meninges* – congested.

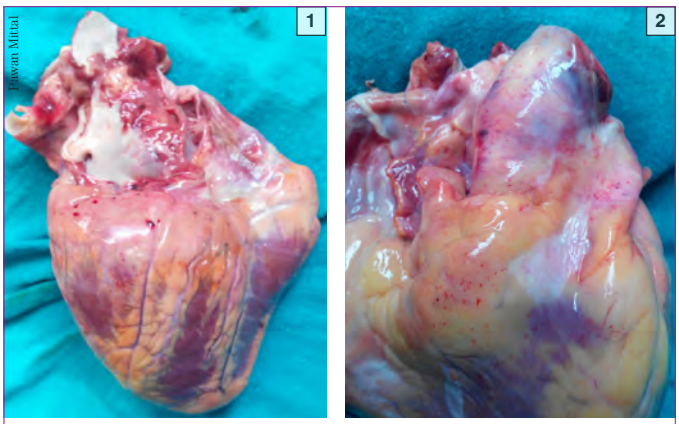


Fig. 36.3: Subpericardial hemorrhages in acute arsenic poisoning **(1)**Anterior surface **(2)** Posterior surface

E. Chronic Poisoning

*WHO defines **chronic arsenic poisoning** [syn, **arsenicosis**] as a chronic health condition arising from prolonged ingestion [not less than six months] of arsenic above a safe dose, usually manifested by characteristics skin lesions, with or without involvement of internal organs.* Some authorities consider the period of onset as within 4 wks of continuous ingestion. **Salient features:**

1. Occurs in following circumstances (i) **Occupational** - due to repeated accidental ingestion of small doses of arsenic by those working with the metal (ii) **Through contaminated water** - People ingesting arsenic through **water contaminated with arsenic** (as in several parts of West Bengal and Bangla Desh) (iii) **Arsenophagists** [please see below] (iv) **Homicidal** – when someone gives repeated doses to his enemy (v) **Recovery from single heavy dose.**

1. Signs and symptoms

Occur in 4 stages.

a. 1st stage [Gastrointestinal]

1. **General:** (i) Circumscribed edema of lower eyelids and ankles [due to localized transudation of intravascular fluid] (ii) Loss of weight (iii) Malaise (iv) Pulse rate↑ (v) Temp↑
2. **GIT:** (i) Loss of appetite (ii) *Gums* – (a) red and soft (b) Black line on gums due to arsenic sulphide (iii) *Tongue* – coated with a thin, white, silver fur (iv) Nausea, vomiting [vomitus contains mucus tinged with bile] (v) salivation (vi) Abdominal cramps (vii) colicky pain (viii) Constipation. Sometimes diarrhea.

b. 2nd stage [Dermatological]

1. **Dermatological:** (i) *Hyperkeratosis* – (a) Discrete, multiple, hard wart like growths appear on (I) palms

it became a popular homicidal poison too. **Thallium acetate** was once used to check the "night sweats" in tuberculosis. When it was noticed that it caused loss of hair, Sabouraud instituted its therapeutic use for tinea in 1898. It was supplied as a cream (Koremlou cream), containing 7.18% thallium acetate. This practice was abandoned half a century later, when it was realized that it was a potential poison. Thallium is used in electronics industry [App 60–70% of Tl production], pharmaceuticals, in glass manufacturing and in infrared detectors.

A. Acute poisoning

Can occur from

1. oral ingestion [most common]
2. inhalation from contaminated dust from pyrite burners, cadmium manufacturing, lead and zinc smelting, and
3. as a contaminant of heroin or cocaine. Symptoms occur after a latent period of 12 hours to 12 days.

1. Signs and symptoms

1. **GIT:** (i) *Abdominal pain* [most common]. Accompanied by (ii) vomiting and either (iii) diarrhea [if irritant effect of Tl is predominant] or (iv) constipation [if involvement of vagus nerve is predominant →↓d intestinal motility, ↓d peristalsis] (v) severe symptoms, eg hematemesis and bloody diarrhea are more rare.
2. **Chest** – tightness and pain [vagus involvement]
3. **CVS:** (i) Tachycardia and (ii) hypertension [both develop after 1-2 wks. These are due to (a) vagus involvement (b) Tl stimulates ATPase in the chromaffin cells→catecholamines ↑→sinus tachycardia. *Persistent and pronounced tachycardia points to poor prognosis* (iii) **ECG changes** [due to direct myocardial damage] – (a) prolongation of the QTc interval, (b) T-wave flattening or inversion, and (c) nonspecific ST-segment abnormalities
4. **Skin** – Maculo-papular skin eruption having butterfly distribution on face [very characteristic]
5. **Coma** – occurs with large exposures. If recovery occurs, patient may suffer from permanent Guillain-Barré like polyneuritis.

2. Fatal dose

6-40 mg/kg of Tl salts. Death is due to coma with loss of airway-protective reflexes, respiratory paralysis, and cardiac arrest.

3. Fatal period

24-36 h.

B. Chronic poisoning

Due to repeated administration [homicidally or taken as medicine]. **Most characteristic lesions** are *abdominal* pain [seen also in acute poisoning], *alopecia* and *neurologic symptoms [Thallium triad]*.

1. **Alopecia:** [Fig 36.7] (i) *Most common and classic manifestation of Tl toxicity*. (ii) Typically it is the *presenting symptom*. (iii) Begins in 10 days. Total hair loss in 30 days.
2. **Neurologic symptoms:** (i) painful ascending peripheral neuropathy [appears after 2-5 days]
3. **Skin:** (i) acne, (ii) palmar erythema, (iii) anhidrosis and (iv) dry scaly skin [results from damage to the sebaceous glands].
4. **Nails** - Mees lines appear within 2-4 weeks after exposure [also seen in arsenic].

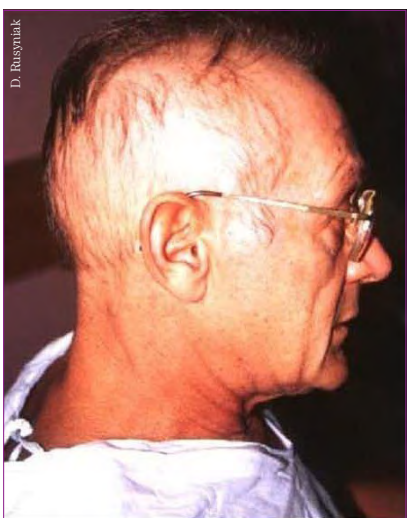


Fig. 36.7: Thallium-induced alopecia.

C. D/d

Must be differentiated from neuropathy seen in

1. poisoning by (i) arsenic, (ii) colchicine, and (iii) vinca alkaloids;
2. botulism;
3. thiamine deficiency; and
4. Guillain-Barré syndrome.

D. Management

1. **Gastric lavage** - with **1% KI** or **Prussian blue.** After the lavage Prussian blue is instilled through a duodenal tube directly into the duodenum [since Tl diminishes gastric and intestinal motility]. The dose is 125 mg/kg bd, with 50 ml of 15% mannitol [laxative to counter constipation].

4.	Microscopy of the prismatic cells in the outer coats of seeds	1. **Size -** (i) length of cells - 250µ (ii) size of Lumen - 3µ 2. **cell-walls -** show fine transverse striae giving the cells a ribbed appearance.	1. **Size -** (i) length of cells -300µ (ii) size of Lumen - 1µ 2. **cell-walls -** striae not noticeable. Cells do not show a ribbed appearance.

B. Signs and symptoms

1. Ingestion

1. **GIT** - (i) Vomiting and diarrhea (ii) Salivation (iii) Hot burning pain from mouth to stomach
2. **Systemic** - (i) Vertigo (ii) Prostration (iii) Collapse (iv) Death.

2. Application on skin

Application of oil on skin produces

1. burning,
2. redness,
3. vesication.

C. Fatal dose

1. **Seeds** – 4
2. **Croton oil** – 20 drops.

D. Fatal period

6 hours -3 days.

E. Management

1. Gastric lavage
2. Demulcents
3. symptomatic.

F. PM appearances

1. **Mucosa of GIT**- congested, inflamed, excoriated
2. **All internal organs** - congested.

G. ML importance

1. **Accidental poisoning** – results from ingesting croton seeds or oil by mistake, or when taken in large doses as medicine (purgative), or by inhalation of dust
2. **Abortifacient** – Root and oil **[ch 26]**
3. **Arrow poison** – paste made by crushing the soft parts of plant [not the seeds]
4. **Ayurveda** - In Ayurvedic system of medicine croton seed oil is used in minute doses in cerebral affections [apoplexy, convulsions, insanity], dropsy, high blood pressure, intestinal obstruction, lead poisoning and obstinate constipation. May cause poisoning if incorrectly used. Formerly also used as a counterirritant in all systems of medicine.
5. **Suicide and homicide** – Rare. Fruit is boiled in water and added to food
6. **Poisoning animals** – croton oil has been used to poison fish in tanks. Mixed with beef it has been used mischievously to kill pet tiger belonging to a circus party.
7. **ordeal poison [ch 31]**
8. **As a counterirritant** - Diluted with a suitable inactive vehicle, croton oil was once used as a counterirritant.
9. **Chemical peels** – Due to its caustic exfoliating effects, croton oil [in combination with phenol] has been used in chemical peels. Leads to initial skin sloughing and then eventual regeneration.
10. As an **additive to fuel** to render it unsafe for drinking – Can be added to fuel (comprising of alcohol) to render it unsafe for drinking (much as methyl alcohol is added to ethyl alcohol to denature it). US did so in **World War II** to prevent sailors from drinking fuel (comprising of grain spirits). Reportedly a number of sailors distilled alcohol away from the mixture and still consumed it!

III. ABRUS PRECATORIUS (*RATTI*)

Abrus precatorius *(Ratti, Gunchi, Jequirity, Crab's Eye, Rosary Pea) is a slender, perennial climber found all over India that twines around trees, shrubs, and hedges*. In **1891, Hellin** discovered the agglutinating properties of abrin.

Salient features:

1. **Leaves** – compound, feather like; 1-2.5 cm long; 10-15 pairs of narrow leaflets.
2. **Flowers** – small and pale violet, arranged in clusters, with a short stalk.
3. **Seed pods** – 2.5-5 cm long, borne in clusters. Green when immature; on maturing, become dry and brown; split open while remaining on vine with 4-6 seeds exposed. All parts of the plant are poisonous.
4. **Seeds** – (i) *Size* - Pea sized. (ii) *Color* - Bright scarlet color with a large black spot at one end [Fig 37.5]. Rarely

Fig. 37.5: *Abrus precatorius* seeds.

fabricated bruises, preserve for chemical analysis. (ii) Apply antiseptics.

2. **If ingested** - (i) Gastric lavage, (ii) demulcents. (iii) Preserve gastric lavage for chemical analysis in all cases.

F. PM appearances

1. Mouth, palate, throat, other oral structures – inflamed, show blisters
2. Stomach – congested. May show fragments of seed.

G. MLI

1. **Artificial bruises** – are made on arms, thighs, breasts etc to bring about a false charge of assault.
2. **Criminal abortion** – juice is applied to the cervical os.
3. **Infidelity** – juice is introduced in the vagina as a punishment.
4. **Injury** - Juice is sometimes thrown on the body of other to cause injury
5. **Malingerers** – put the juice inside their eye to produce ophthalmitis
6. **Manner of poisoning** - (i) *Accidental poisoning* – Common. Poisoning may occur because the nut is used in Indian medicine for a variety of disorders eg allergic dermatitis, ascites, asthma, colitis, cough, diarrhea, dyspepsia, leprosy, leukoderma, piles, poisonous bites, scaly skin, tumors and worms. The oil is topically applied on swollen joints and traumatic wounds. May result in false bruises. (ii) *Suicidal and homicidal poisoning* – Rare
7. **Occupational poisoning** - Dhobie mark itch (**Dhobie mark dermatitis**) is *urushiol dermatitis* seen in Indian washermen who use **marking nut** in their work. Has been reported even in persons, who wear clothes laundered and marked in this way. The latter mode was the cause of dermatitis affecting several English soldiers stationed in pre-independent India.

VI. CALOTROPIS GIGANTEA (AKDO, MADAR)

A. Active principles

Calactin, calatoxin, calotropin, trypsin, uscharidin and uscharin. They have CNS depressant and anticonvulsant activity.

B. Signs and symptoms

1. Dermal application

1. Redness
2. vesication and bullous eruptions.

Fig. 37.8: (1) *Calotropis gigantea*. A stem has been broken to show the irritant juice coming out of it. **(2)** Plant showing fruit, with two punctures showing long silky hairs inside. Please see text for its uses.

2. Instillation in eye

Instillation of juice in the eye is often done for local eye ailments. Produces 1.keratitis, 2.conjunctivitis, 3.corneal edema, 4.dimness of vision.

3. Ingestion

1. Acrid bitter taste
2. Burning pain in mouth, throat and stomach
3. Salivation
4. Stomatitis
5. Vomiting and diarrhea
6. Pupils – dilated
7. **In animal models** – *cardiotoxic* and *hepatotoxic*. In rats and sheep produces tracheal exudates, pulmonary edema, tachycardia, transitory cardiac arrhythmias, ascites, liver hemorrhages, hydropericardium, flaccid heart, and pale juxtamedullary renal cortex.
8. Tetanic convulsions
9. Collapse and death.

C. Fatal dose

Uncertain.

D. Fatal period

6-12 hours.

E. Management

1. Gastric lavage
2. demulcents
3. symptomatic.

F. PM appearances

1. Pupils – dilated
2. Mouth – stomatitis, froth
3. nostrils – froth

venom from wound may not be poisoned if he swallows poison. However if he has minor abrasions in mouth, the poison may be absorbed from there and *secondary poisoning* may occur in the rescuer.

III. SCORPION

***Scorpions** are predatory arthropods of class Arachnida.*

A. Mode of action

[S]corpion venom opens neuronal **[s]**odium channels **[Memory Aid ……]. This causes (i) [r]epetitive and [s]pontaneous depolarization of** both sympathetic and **[p]**arasympathetic nervous system, causing an autonomic storm **[pl co-relate with signs and symptoms].**

Memory Aid … - MOA of scorpion venom

[S]co[r][p]ion venom causes – [r]epetitive and [s]pontaneous [s]ympathetic and [p]arasympathetic depolarization

***Typically the parasympathetic effects are** transient, **but sympathetic are more** prolonged.* **(ii) →catecholamine release from adrenal gland (iii) catecholamine induced cardiac hypoxia (iv)→ renin secretion at the juxtaglomerular apparatus. (iv) Hyperkalemia, hyperglycemia, →insulin secretion, →aldosterone.**

B. Signs and symptoms

Most stings occur on extremities. There are significant differences in signs and symptoms produced by different species. Stings from *M. tamulus* [Indian red scorpion] produce following symptoms.

1. **Local** - (i) edema (ii) erythema (iii) pain [severe, excruciating] (iv) regional lymphadenopathy. Last 1-2 h.
2. **Systemic** - (i) Nausea, vomiting (ii) extreme restlessness (iii) Allergic reactions, including anaphylaxis (iv) Cyanosis (v) Pupils - dilated (vi) Fever (vii) Paralysis (viii) Respiratory depression
3. **Sympathetic** - (i) cardiogenic shock (ii) BP↑ [BP↓ if parasympathetic predominates] (iii) MI (iv) myocarditis (v) pulmonary edema (vi) arrhythmias, tachycardia
4. **Parasympathetic** - (i) priapism (ii) sweating (iii) vomiting
5. **Other** - (i) coagulopathy (ii) pancreatitis (iii) stroke
6. **Rare** - (i) Intracerebral hemorrhage [*Mechanism* - venom induced autonomic storm→hypertension, vasculitis].
7. **Death** – if occurs is preceded by convulsions and coma. Mortality except in children is negligible.

Memory Aid … - Signs and symptoms of scorpion stings

[All] [S] [C] [O] [R] [P] [I] [O] [N] [S] cause [MI], but [fe]w [dilate pupils]

[All]ergy

[S]alivation, sweating

[C]yanosis, cardiogenic shock, coagulopathy,

[O]edema

[R]egional lymphadenopathy, restlessness, respiratory depression

[P]ain, paralysis, pulmonary edema, priapism, pancreatitis

[I]nspirations slow [respiratory depression]

St[O]rm [autonomic]

[N]ausea & Vomiting

[S]troke

[C]onvulsions and coma precede death

[MI], Myocarditis

[fe]ver

[dilated pupils]

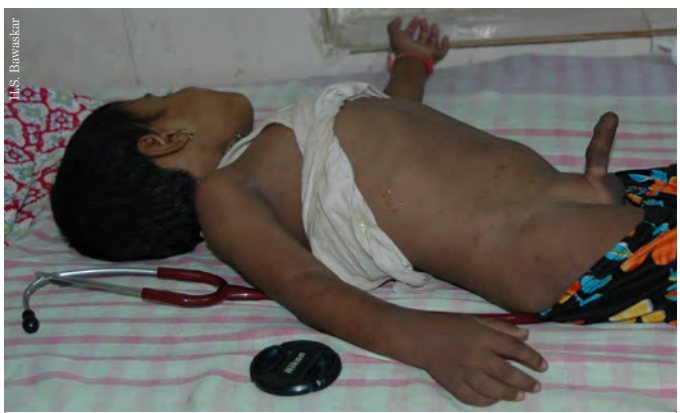


Fig. 38.…: Priapism in a child in scorpion bite.

C. Fatal period

Few hours.

D. Diagnosis

1. **ECG** – (i) Peaked T waves in leads V_2-V_6. (ii) Q wave, ST segment elevation in leads I and aVL (iii) Left anterior hemiblock.

E. Differential Diagnosis

1. **Snake bite** – Snake bite shows two punctures; scorpion stings shows one. Confirmation by ELISA.

F. Management

1. **First aid** - (i) Immobilize limb (ii) Tourniquet – Technique and pressure as in snakebite (iii) Local washing – with weak soln of ammonia, borax or

constricted (vii) sweating, (viii) Twitching of limbs (ix) coma, convulsions, collapse, death

4. **Hepatic and renal toxicity** – develops in 3-6 days, in cases of survival.

2. Fatal dose

One mushroom.

3. Fatal period

24 hours.

4. Test

Meixner test -

1. Sample (stool, gastric lavage) + methanol→ centrifuge→filter
2. Add 2 drops of filtrate to a newspaper
3. Encircle the spot with a pencil and dry it
4. Add a drop of conc. HCl to the dry spot→**blue color**→Amatoxins present. Phallotoxins do not give blue color.

Memory Aid 1 – Meixner test

Meixner test is done in Mushroom poisoning

5. Management

1. Stomach wash – with $KMnO_4$.
2. Activated charcoal
3. Forced diuresis, Hemodialysis
4. Benzyl penicillin 3-10 lakh units daily
5. Atropine sulphate
6. **Antidotes:** (i) Anti-phalloidin antibodies (ii) *Cimetidine* - Potent cytochrome P450 system inhibitor. Protects liver against alpha-amanitin. Dose 4-6 g/day. (iii) *Iridoid glycosides* - These are a class of secondary metabolites found in a wide variety of plants and in some animals. Named after Iridomyrmex genus which contains a defensive chemical iridomyrmecin. (a) *Aucubin* - derived from the leaves of *Aucuba japonica*. Found to be protective against Amanita phalloides. (b) *Kutkin* - Kutkin is a mixture of iridoid glycosides picroside I and kutkoside isolated from the roots of *Picrorhiza kurroa*. Found to be protective against Amanita phalloides. Efficacy is superior even to silibinin. (iv) *N-acetylcysteine* - Same dose as in paracetamol poisoning (v) *Silibinin* - A semi-purified fraction of silymarin derived from milk thistle [*Silybum marianum*]. (vi) *Thioctic acid* - Once thought to be useful in hepatic damage. But now obsolete.
7. Management of acute liver failure -
8. Symptomatic

6. PM findings

1. **GIT** - inflammation of mucosa
2. **Heart, kidneys** - Fatty degeneration. Kidneys may be pale and swollen.
3. **Liver** - Shows most characteristic findings as mushrooms are hepatotoxic. Central zonal necrosis, centrilobular hemorrhage, fatty degeneration and steatosis. Findings of hepatic failure, eg anasarca, pulmonary edema, scleral icterus and serous effusions are seen.
4. **Brain** - Congestion, petechial hemorrhages in serous membranes and in substance of brain, especially if neurotic symptoms were present during life.

7. ML Importance

1. Poisoning is usually accidental. Rarely homicidal.

C. Argemone mexicana

Argemone mexicana (*Kutila*, Mexican poppy, *Phirangi Datura*, *Pila-Dhatura*, prickly poppy, *Satyanasi*, *Sial Kanta*, *Ujar kanta*) grows wild all over India in the cold season. **Salient features:**

Fig. 39.3: *Argemone mexicana.*

1. It is an annual herb, belonging to the family *Papaveraceae* [Another poisonous plant of this family is *P. somniferum*. Please see **ch 40**], which grows wild all over India during winters [Fig 39.3]
2. **Plant:** (i) It grows 1-4 ft high, with spreading branches. (ii) *Leaves* – sessile, spiny, thistle like (iii) *Flowers* - 1-3 inch across and are colored yellow. (iv) *Capsules* – Prickly oblong or elliptic 2-4 cm long. Contain seeds (v) *Seeds* - (a) blackish brown, round and netted and resemble those of mustard [*Brassica nigra]* (b) Differences - (I) Argemone seeds have spiny edges. Mustard seeds do not have such spiny edges. (II) When seeds of *A. mexicana* are pressed on a slide, they burst with a report; mustard seeds collapse quietly.
3. **Alkaloids** - All parts of plant are poisonous. The extract of the whole plant (latex) contains two alkaloids *Berberine* and *Protopine*.
4. **Katkar oil:** (i) *A. mexicana* seeds yield 22-36% of a pale yellow, nauseous, bitter, non-edible oil [Katkar oil or Argemone oil]. (ii) The oil is rich in two

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*).

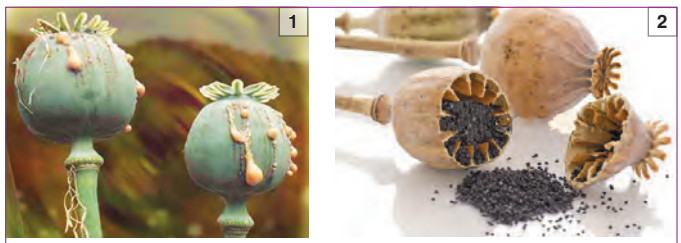


Fig. 40.4: **(1)** Poppy capsules with incisions over surface. Drops of latex (poppy tears) can be seen oozing out. On drying they would constitute raw opium **(2)** Poppy seeds (slate gray variety).

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 tough on storage.
2. **Alkaloids** – contains about **25 alkaloids**, combined with meconic, lactic and sulfuric acids. Chemically they form two groups (a) **Phenanthrenes** [*morphine* [Indian Opium yields approx 9.5-14%. The morphine content is standardized in the Govt opium factory at Ghazipur, to contain 10% morphine. This is known as **Standard** or **Standardized Opium**. The total alkaloids of this opium are 40%], *codeine* (0.5%) and *thebaine* (0.2%)] and (b) **Benzylisoquinolines** [*noscapine* (formerly called *narcotine)* (6%), *papaverine* (1%), *narceine*(0.2%)].
3. **Terminology – Differences between opiates and opioids** - **Opiates** are drugs *derived from opium*, and include *natural* products morphine, codeine, thebaine and *semisynthetic* congeners derived from them eg heroin. **Opioid** (Gk *eidos*, similar to) on the other hand – is a much broader term which refers to *any* agent that binds to opioid receptors. It is *a more inclusive term* applying to all agonists and antagonists with morphine-like activity. The term *opioid* would thus include completely synthetic products such as diphenoxylate, fentanyl, loperamide, methadone, pethidine (meperidine), propoxyphene, *which are not included under opiates*.

Memory Aid 8

Opioi**d** is a much broa**d**er term.

2. Classification of opioids

1. **Natural** – morphine, codeine
2. **Semi-synthetic** – Heroin, Pholcodeine. Many others like -Hydrocodone, Hydromorphone, Oxycodone and Oxymorphone are not used in India.
3. **Synthetic** – Dextropropoxyphene, Diphenoxylate, Fentanyl, Methadone, Paregoric, pethidine [Meperidine], propoxyphene, Tramadol.

3. Absorption, distribution, fate and excretion

1. **Opioids** - (i) Absorbed from the GI tract and through rectal mucosa. (ii) *Placental barrier* - Most opiates cross placenta, and appear in fetal circulation within 5 min following maternal IV injection.
2. **Morphine** - (i) There is significant *first-pass metabolism* in the liver, because of which oral routes are *less effective* than parenteral. (ii) *Half life of morphine* - in plasma is **2 hours**. (iii) *Elimination of morphine* - (a) is by glomerular filtration. (b) **Enterohepatic circulation** of morphine and its glucuronides occurs, which accounts for the presence of small amounts of morphine in feces and urine for **several days** after the last dose.
3. **Codeine** - and its analogs [levorphanol, oxycodone, methadone] have a lower first-pass metabolism in the liver.
4. **Heroin** (diacetylmorphine) - is rapidly hydrolyzed to 6-monoacetylmorphine (6-MAM), which in turn is hydrolyzed to morphine.

4. Mechanism of action

Opium acts through opioid receptors, which are a group of **G protein-coupled receptors** (**GPCRs**). These are mainly located in the CNS.

5. Acute Poisoning

a. Signs and symptoms

i. On contact

1. If a person is sensitive to opium, he may experience
2. Erythema
3. Itching dermatitis
4. Urticaria.

alkaloids. This may give rise to unintentional poisoning [*toxic honey*. Please see more in **ch 39** – "Food poisoning"]. Some hummingbirds, eg sword-billed hummingbird (*Ensifera ensifera*), can feed on the nectar from *Datura* flowers without ill-effects.

II. ATROPA BELLADONNA

Atropa belladonna *[syn. Belladonna, Devil's Berries, Death Cherries, Deadly Nightshade], is a perennial herbaceous plant of the family Solanaceae*. **Salient features**:

1. Active principles are hyoscyamine [82-97%], atropine [3-15%] and scopolamine [2.5%].
2. Signs, symptoms, treatment and pm appearances are same as that of Datura.
3. Because it produced senselessness, it was used in the Middle Ages as an anesthetic for surgery.
4. The plant has reddish-purple flowers.

A. ML importance

1. Arrow poison.
2. **Manner of poisoning** - (i) *Homicide* - The ancient Arabs used it as a poison. The wife of Emperor Augustus and the wife of Claudius both used it to murder their enemies.

III. CANNABIS

Cannabis (Indian hemp, pot, dope, grass) is a genus of flowering plants that includes **three** species, *Cannabis sativa*, *Cannabis indica*, and *Cannabis ruderalis*. All three are found in India, Central Asia, and South Asia (Fig 41.3). **Salient features**:

Fig. 41.3: Cannabis plant

1. **Plant** - (i) Cannabis is an annual, dioecious (male and female plants occurring separately), flowering herb. (ii) The leaves are digitate with serrate leaflets
2. **Frequency of use** - Currently it is the most commonly used *illicit* drug of abuse. If *socially acceptable drugs* are also taken into account, then it is 4th most common drug of abuse - (i) Caffeine (ii) Nicotine (iii) Alcohol (iv) Cannabis.

A. Active principles

1. Active principles are **cannabinoids** [*syn*, phyto-cannabinoids], secreted by trichomes. *At least 90 plant Cannabinoids have been isolated from cannabis.*
2. Cannabinoids of importance are (i) **Tetrahydro-cannabinols** or **THC**. Most important THCs are Δ^8**- tetrahydrocannabinol** and Δ^9**- tetrahydro-cannabinol**(pronounced as delta-8 and delta-9 tetrahydrocannabinol) (ii) **cannabinol** [CBN] (iii) **cannabidiol** [CBD].

B. Mechanism of action

1. **Endogenous cannabinoid receptors** - (i) Cannabinoids exert their effects by interaction with specific endogenous cannabinoid receptors [**CB_1, CB_2, GPR18**, **GPR55** and **GPR119**]. (ii) Discovered in 1990s. (iii) All are **G protein-coupled cannabinoid receptors** [GPRs]. (iv) *Location* - CB_1 is located in the brain, and CB_2 in peripheral tissues of the immune system.
2. **Endogenous cannabinoids** - **Anandamide** [from Sanskrit word "Anand", happiness], an endogenous cannabinoid, attaches to these receptors. Produces natural pain relief and controls immunity.
3. **THC** – Additionally acts on CB_1 and enteric nervous system to suppress emetogenic stimuli communicating between them. Acts as an effective antiemetic.

C. Forms of Cannabis

1. Bhang

Bhang *[Patti, Siddhi, Sabji] consists of dried mature leaves and stems of cannabis*. **Salient features:**

1. **Potency** - It is the *mildest form* of Cannabis and contains **15%** of active principle. Fresh *Bhang* is more potent; it loses its potency on storage. *Bhang* stored for 2-3 years has very mild euphoriant properties.
2. **Forms in which consumed** - (i) *As an infusion* – Seeped in water, strained through a muslin cloth and used as a beverage (ii) *Cannabis tea* – leaves boiled in water just like tea leaves [please see ch 40 for "poppy tea"]. The resulting infusion is supposed to have more Δ^9**-THC** [main bioactive component of cannabis], because heat converts tetrahydrocannabinolic acid (THCA) to Δ^9**-THC**. (iii) *As a pill or bolus* – Leaved rubbed with black pepper on a stone. Sugar is added to make a pill or bolus [*Bhang golis* or balls] (iv) *Mixed with milk* – Almonds and spices (mainly black pepper) are also added and served on festive occassions such as *Holi* in March and *Vaisakhi* in April, especially in places like Mathura and Varanasi [*Bhang Ki Thandai*, *Shardai*] (v) *Mixed with fruit juice*

C. Fatal dose

Digitalin – 15-30 mg **Digoxin** [present in *D. lanata*] – 10 mg **Digitoxin** – 4mg; **Leaves** 1-2 (2g) Therapeutic blood levels for digoxin are 0.5-1.0 ng/mL. Toxic levels for digoxin are >2.0 ng/mL.

D. Fatal period

24 h.

E. Management

1. **Gastric decontamination** – by emesis or lavage is *rarely needed* because digitalis is emetic It is rapidly absorbed from GIT. If required, tannic acid is used.
2. Whole bowel irrigation.
3. **Activated charcoal** – Even late administration is useful as both digoxin and digitoxin have enterohepatic and enteroenteric circulation. Multiple Dose Activated Charcoal (MDAC) is useful for the same reason **[ch 32].**
4. **Steroid-binding resins** - eg *cholestyramine* and *colestipol* prevent reabsorption from the GI tract. Also interrupt enteroenteric and enterohepatic circulation.
5. **Digoxin-Specific Antibody Fragments** or **Fab** [Digibind, DigiFab]. (i) **Availability** – *Digibind* [38 mg/vial] or *DigiFab*[40 mg/vial] in lyophilized form. (ii) **Administration** – (a) Vial is reconstituted with 4 mL of sterile water. (b) **Dose** – (I) *Acute ingestions* - 10-20 vials in both child and adult. (II) *Chronic toxicity* – Adult – 3-6 vials; children - 1-2 vials. (c) **Used in** - Toxicity with digoxin, digitoxin, and all natural cardioactive steroids, eg nerium, oleander, squill, and toad venom.
6. **If Fab fragments are not available** - (i) *Antiarrhythmics* - (a) *Lignocaine* - 1mg/kg IV bolus. Followed by continuous infusion at 1-4mg/min in adult; 20-50 µg/kg/min in child. (b) *Phenytoin* [*dilantin*]- 50 mg/min slow IV. Max 1g in adult; 15-20 mg/kg in child. Maintenance oral dose - 300-400 mg/day in adults; 6-10 mg/kg/day in child. (c) *Propranolol* and *Reserpine* - have been found useful.
7. **Management of bradycardia** - (i) *Mechanism of bradycardia and varying degrees of heart block* - digitalis induced effects of ↑ed vagal tone on SA node rhythmicity and on AV node conduction (ii) *Atropine* - 0.5 mg IV in adult; 0.02 mg/kg in child. Repeat every 5 min till normalization of heart rate. (iii) *External or transvenous pacemaker* - in severe bradycardia.
8. **Management of extrasystoles and arrhythmias** - (i) *Magnesium* - 20mL of 20% sol^{n} over 20 min by slow infusion. To be used with caution in renal failure (ii) *Potassium* salts.
9. **For hypercalcemia** - Trisodium EDTA.
10. **Hemodialysis** - Does not remove digitalis, but restores serum K^{+} levels to normal.
11. **Hemoperfusion** - using antidigoxin antibodies.
12. **Percutaneous cardiopulmonary bypass** - for therapy resistant cardiac arrest.
13. **Symptomatic** - Electrolyte disturbances must be treated, eg magnesium and potassium levels which may be abnormally low or high.

F. Postmortem appearances

Fragments of leaves in GIT – if leaves were consumed **GIT mucosa** – inflamed Pulmonary congestion **Confirmatory** - Detection and measurement of Digoxin and other cardiac glycosides is confirmatory.

G. ML Importance

1. **Bitter taste** - Although foxglove is widely grown as an ornamental plant, few people ingest it because of its bitter taste
2. **Accidental ingestion** - may occur from **consumption of contaminated water** in or near which plants have been standing or from **smoke from burning plants**
3. **Mistaken for comfrey** [*Symphytum officinale*] - Comfrey leaves resemble those of foxglove when the plant is not in bloom. Comfrey is used as a herbal medicine for treating fractures, tendon injury, GIT ulceration, lung congestion, joint inflammation, and promoting wound healing. Comfrey tea is usually taken for these ailments, and leaves must be gathered from wild. Wild-food gatherers mistake the plant for **comfrey**, and use it in their herbal concoctions. This accounts for most cases of toxicity.
4. **Iatrogenic poisoning** – Can occur during overambitious treatment with digitalis.

IV. NERIUM OLEANDER

Both ***Nerium oleander*** (white or pink Oleander, *Kaner*) and ***Thevetia peruviana*** (yellow oleander) belong to the Dogbane family, Apocynaceae.

Description [Fig 43.1]-

1. *N. oleander* grows wild in India. It is an evergreen

Fig. 43.1: Nerium oleander

Chapter 47

Miscellaneous Poisons

I. FORMALDEHYDE

Formaldehyde (HCHO) is a gas at room temperature. *Aqueous solutions of formaldehyde are referred to as* ***formalin***. Saturated solution of formaldehyde (100% formalin) contains 40% by volume or 37% by mass of formaldehyde (i.e. if 1000 g of commercial solution of formalin is taken, 370 g of it would be formaldehyde. It is customary to add a small amount of stabilizer, usually **10% to 12% methanol**; *it helps prevent oxidization and polymerization of the formaldehyde*, either of which could result in fire or explosion). (i) For histopathology work **10% formalin** is prepared by mixing 1 part formaldehyde (100% formalin) with 9 parts water. Since it contains 4 parts by volume of formaldehyde it is also known as **4% formaldehyde**. Thus **4% formaldehyde = 10% formalin**.

A. Signs and symptoms

1. Acute exposure

(a) **Dermal Contact**:

1. skin hardening
2. cracking
3. bleeding
4. Contact dermatitis

(b) **Inhalation**:

1. Burning of eyes and lacrimation
2. violent coughing
3. constriction in chest and palpitation4.asthma like symptoms

(c) **Ingestion**:

1. Similar to strong corrosive acid.
2. Since methanol is added to formalin solution, concomitant **methanol toxicity** is also seen.
3. **Extreme tachycardia** – is a prominent feature
4. ↓body temperature.

2. Chronic exposure

1. Chronic obstructive pulmonary disease
2. reduction in ventilatory capacity
3. Optic neuritis.

B. Fatal dose

60-90 ml[4]

C. Fatal period

1-2 days.

D. Management

1. **Stomach wash** – with milk, water, 1% ammonium carbonate or 0.1% solution of ammonia. The latter reacts with formalin to form harmless **Hexamine**.
2. Activated charcoal
3. Mild saline catharsis
4. Keep the patient warm
5. Watch for GIT hemorrhage.

E. PM Appearances

1. **Smell** – of formaldehyde on opening the body
2. **Mucosa of stomach** – Hard and tough like leather. Red, inflamed and eroded
3. **Internal organs** – congested
4. **Liver** – Fatty degeneration.

F. ML importance

1. **Accidental poisoning** - occurs mostly. But suicidal and homicidal poisoning are also known
2. **Occupational hazards** - Formaldehyde is used by embalmers and laboratory workers. It is a potential occupational carcinogen. Embalmers and funeral directors exhibit (i) a higher incidence of leukemia and cancers of brain and colon. (ii) "**Embalmer's eczema**", a persistent skin condition. Embalmers must use masks and wear gloves during embalming work.
3. **Toxicology** - Formalin *should never be used as a preservative* [**ch 5**].
4. **Used illegally as a food preservative** – Formaldehyde is not allowed as a food preservative, but it continues to be used so. In 2005 in Indonesia and in 2007 in Vietnam, it was found to be used for **preservation of noodles** and other food products. Causing a major scare.

Appendix 1: Medicolegally Important Sections and Acts

Note

1. **Q 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.**
3. **Abbreviations**

   SI- Simple Imprisonment [no work allotted to prisoner]

   RI - Rigorous Imprisonment [work allotted to prisoner]
4. **Chapter numbers in blue** refer to **chapters of IPC, CrPC and IEA [Also mentioned as a whole word "Chapter"]; chapter numbers in black** refer to **chapters within this book [mentioned as an abbreviated word "ch"]**. *Exact page within the chapter can be seen from the index.*

I. IPC

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

1. S.31. – Definition of a will - ch 28
2. S.34. - Acts done by several persons in furtherance of common intention — each person is liable equally - ch 28
3. S.40 - Definition of offence – ch 1.
4. **S.44Q. Definition of Injury - ch 11.**
5. S.46. Definition of Death – ch 8.
6. **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.**
7. S.52. Good faith — Nothing is said to be done or believed in "good faith" which is done or believed without due care and attention – ch 2.

Chapter IV: General Exceptions [S 76-106]

8. **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.**
9. **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.**
10. **S.82Q. Age of criminal responsibility** in India is **7 y - ch 3.**
11. **S.83Q. Act of child between 7-12 y of immature understanding** — not an offence - **ch 3.**
12. **S.84Q. Criminal responsibility of mentally ill.** Indian equivalent of British McNaugthen rule - **ch 28.**
13. **S.85Q. Person intoxicated against his will** - not responsible for his criminal actions - **ch 31.**
14. **S.86.** Person intoxicated voluntarily - Knowledge of act is presumed; intention to do the act is to be proven by prosecution - **ch 31.**
15. **S.87.** To participate in a risky activity, the consenting person must be >18 y. *Not meant for doctors* - **ch 2.**
16. **S.88Q. 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.**
17. **S.89Q . Consent on behalf of minors - ch 2.**
18. **S.90Q. Conditions of a valid consent - ch 2.**
19. **S.92Q. 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]

20. **S.172.** Absconding to avoid service of summons or other proceeding — **ch 1.**
21. **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.**
22. **S.176.** Omission to give notice or information to public servant by person legally bound to give it — **ch 31.**
23. **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 [S 191-229A]

24. **S.191Q. Definition of perjury** [Giving false evidence] — **ch 1.**
25. **S.193Q. Punishment for perjury — ch 1.**
26. **S.197.** Issuing false certificate — **ch 1.**
27. **S.198.** Using false certificate — **ch 1.**
28. **S.201Q. Causing disappearance of evidence of offence — ch 31.**

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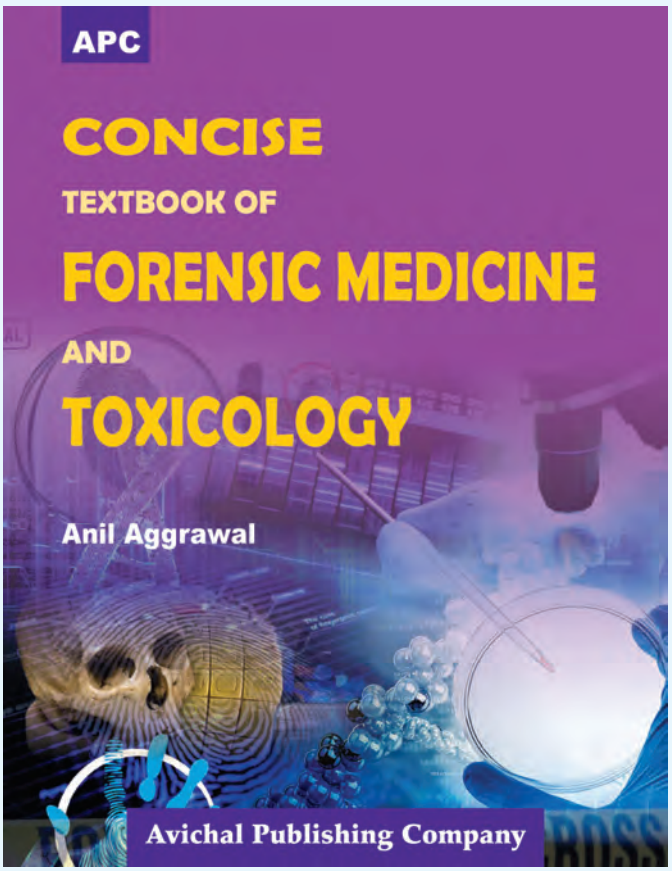
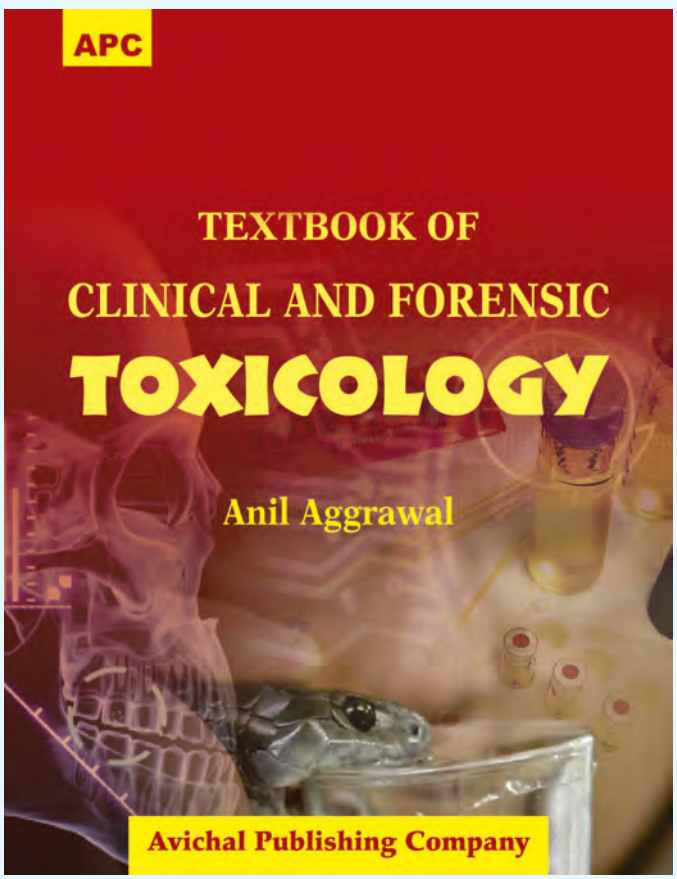
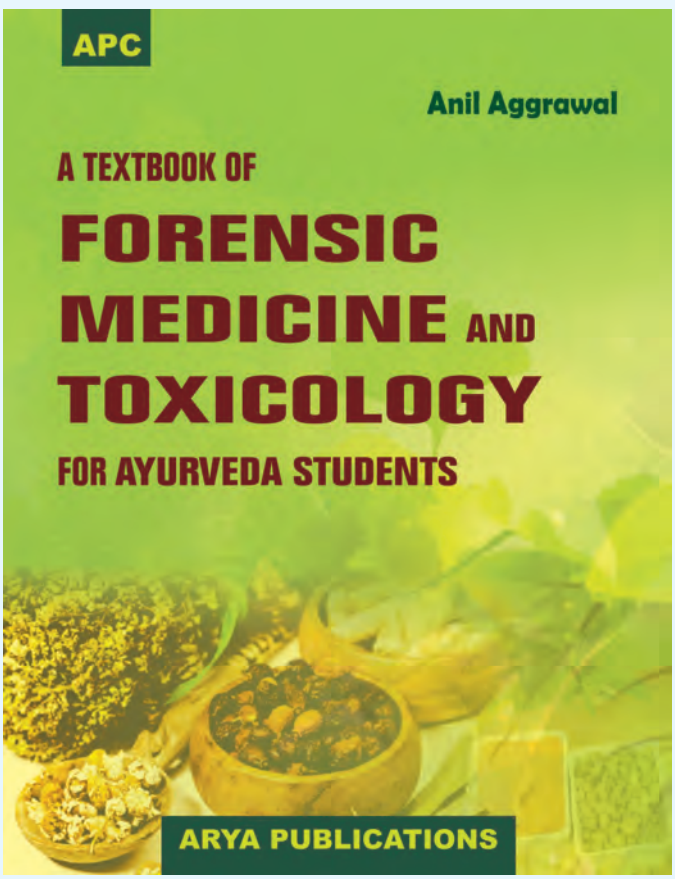






A Textbook of

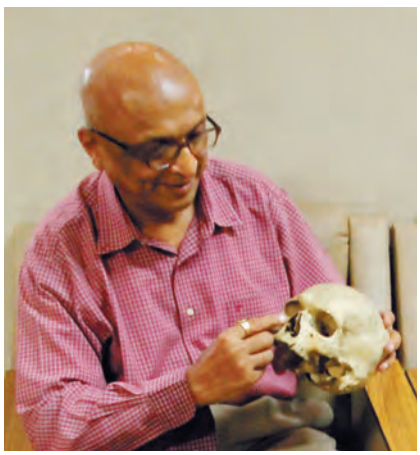
Forensic Medicine and Toxicology

for Homeopathy

There has been no genuine textbook of forensic medicine and toxicology for homeopathy students thus far. Consequently students were forced to read books primarily written for MBBS students, which neither matched their course content, nor were written with an average homeopathy student in mind.

This is a student friendly book written especially for a homeopathy student. For example, this is the first book, which gives the new MTP (Amendment) Bill, 2014, which allows even homeopathic practitioners to conduct Medical Termination of Pregnancy. Information is presented in a logical, systematic and clear manner. About 200 memory aids – flagged off in the margins with a special logo – make memorization quick and easy. Core principles of Forensic Medicine and Toxicology, are explained in an examination-friendly style, which homeopathy students will find useful. Last ten years papers of homeopathy from all major universities were consulted before writing this book.

The Author



Anil Aggrawal obtained his MD in Forensic medicine and toxicology from the All India Institute of Medical Sciences, New Delhi and then proceeded to Edinburgh – traditionally regarded as the home of Forensic Medicine – for higher training. He was trained further in Japan and the US under the prestigious WHO fellowship.

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