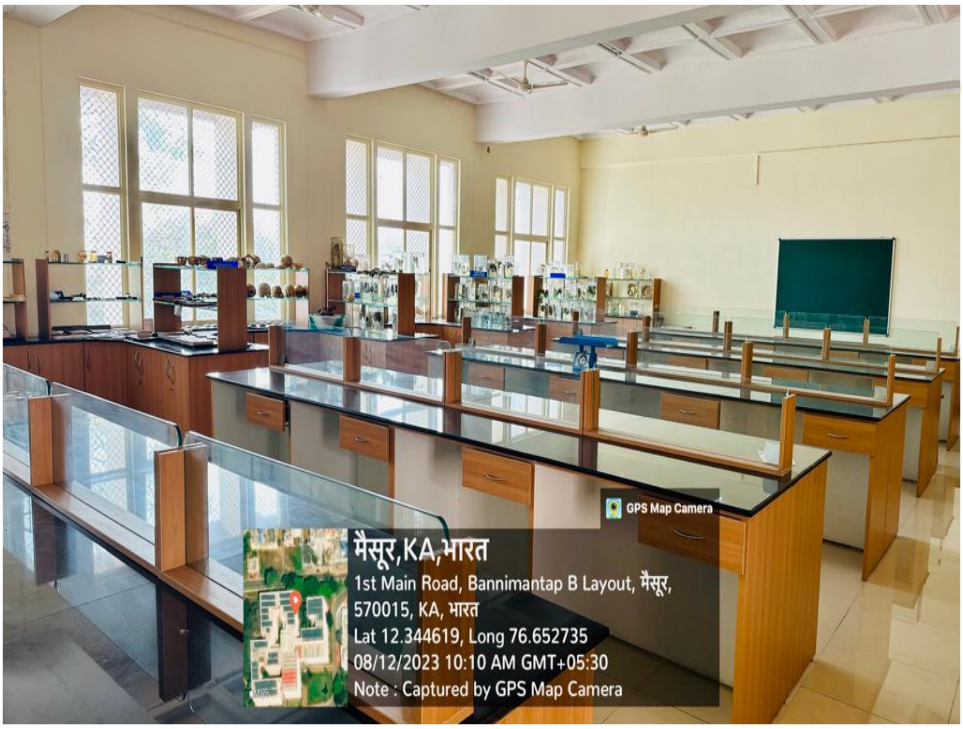
DEPARTMENT OF FORENSIC MEDICINE AND TOXICOLOGY MUSEUM

The museum of the Department of Forensic Medicine and toxicology was established in the year 1999. The museum was renovated in the year 2017. The museum comprises of mounted wet specimens, Toxicology specimens, weapons and instruments. The department also has photography museum, wherein the photos showing different postmortem features in various medicolegal deaths are on display. There is facility to view photographs of all postmortems conducted at JSS Medical College Mortuary in the digital photograph library. The department also has moot court setup to familiarize the students regard court proceeding.

Museum specification

Size	**200 sq.mt**
specimens arrangement	**Wooden show cases.**
Mounted	**170**
Unmounted	**25**
Proto-type fire arms and others	**25**
Wax Models	**36 (plaster of Paris)**
Poisons	**90**
Charts	**96**
Photographs	**800**
Models	**36**
Slides	**50**
Bones	**300**
Weapons	**76**
Catalogues of the specimens	**20 Copies**
Chair with cushion	**60**
Desktop	**01**
Digital display	**01**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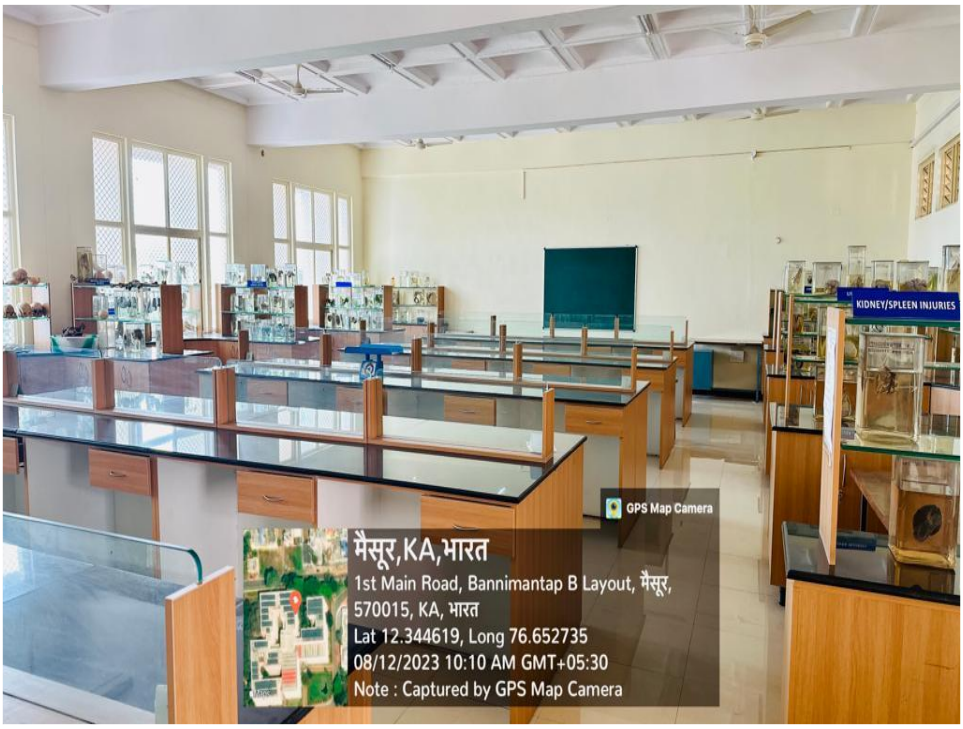
KIDNEY/SPLEEN INJURIES
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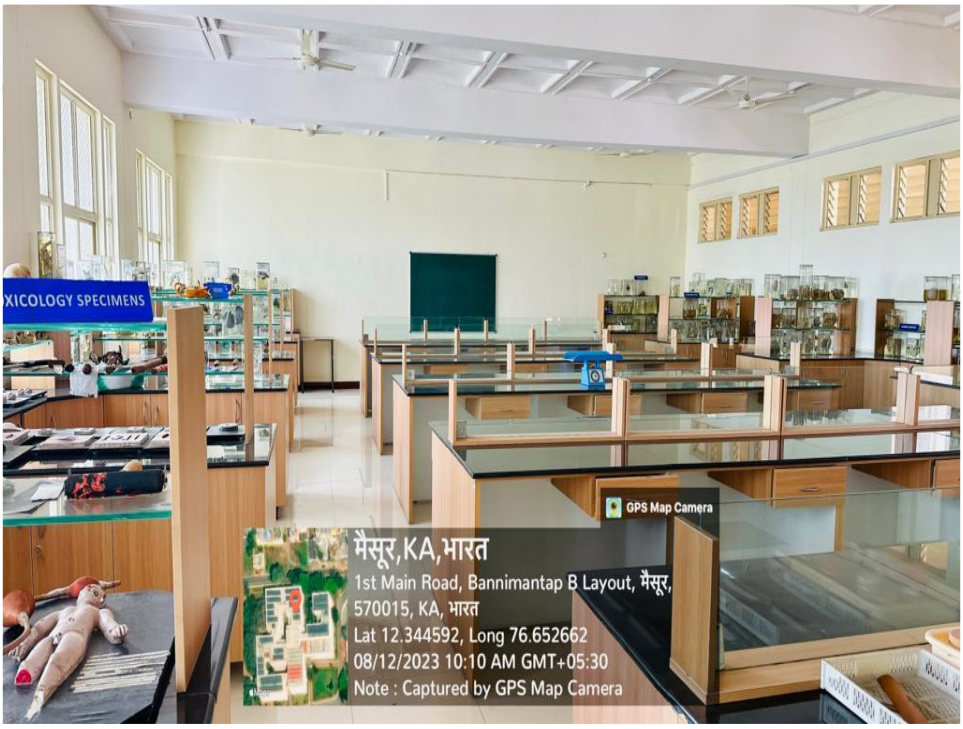
XICOLOGY SPECIMENS
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COURT ROOM SETUP

DIGITAL PHOTOGRAPH LIBRARY

SPECIMEN LIST WITH NUMBERING

A1. FOAMY LIVER (HONEY COMB APPEARANCE)

A2. FOAMY LIVER

A3. MUMMIFIED FOOT

A4. MUMMIFIED FOOT

A5. ADIPOCERE

B1. SCAR OVER SKIN

B2. TATOO MARK ON SKIN

C1. NORMAL HEART (MALE)

C2. NORMAL HEART (FEMALE)

C3. HEART

C4. HEART

C5. DISSECTED HEART

C6 .DISSECTED HEART

C7.DISSECTED HEART

C8.DISSECTED HEART WITH AORTA

C9.SPINALCORD

C10.CEREBELLUM

C11.ACCESSORY SPINAL CORD

C12.BRAIN

C13.BRAIN

C14.PONS

C15.NECK STRUCTURES

C16.NECK STRUCTURES

C17.THYROID CARTILAGE

D1. ATHEROMATOUS PLAQUE-AORTA

D2.CORONARY STENT (IN-SITU)

D3. HEART WITH STENT

(HEART WEIGHT 708 GM)

E1. FATTY HEART

E2.HYPERTROPHIED HEART (460 GM)

E3.HEPATOMEGALY WITH CIRRHOSIS AND SPLENOMEGALY

(CHRONIC MYELOID LEUKEMIA CASE)

E4. HEPATOMEGALY WITH CIRRHOSIS

E5.POLYCYSTIC KIDNEY

E6.HEPATOMEGALY

E7.ENLARGED SPLEEN

F1.LIVER IN STARVATION

F2. SHRUNKEN LIVER WITH DISTENDED GALL BLADDER

(STARVATION CASE)

F3.TISSUE PAPER APPEARANCE-SMALL INTESTINE

(STARVATION DEATH)

F4.FATTY HEART- STARVATION

G1.EXTRA DURAL CLOT

G2.EXTRA DURAL CLOT

G3.ORGANIZED EXTRA DURAL CLOT

G4.BRAIN - SUBARACHNOID HAEMORRHAGE

G5.BRAIN - SUBARACHNOID HAEMORRHAGE

G6.ORGANIZED SUBDURAL HAEMORRHAGE

G7.BRAIN DIFUSE SUBARACHNOID HAEMORRHAGE

G8.SUBARACHNOID HAEMORRHAGE

G9.SUBARACHNOID HAEMORRHAGE

G10.SUBARACHNOID HAEMORRHAGE

G11.SUBDURAL AND SUBARACHNOID HAEMORRHAGE IN RTA CASE

(PM NO 94/10)

G12.HEALED SUBDURAL HAEMORRHAGE WITH PRESSURE EFFECT

G13.PONTINE HAEMORRHAGE

G14.PONTINE HAEMORRHAGE

G15.PONTINE HAEMORRHAGE

G16. SUBARACHNOID HAEMORRHAGE WITH PONTINE HAEMORRHAGE

G17. PONTINE HAEMORRHAGE

G18.SUBARACHNOID HAEMORRHAGE AND SEVERED MIDBRIAN

G19.PONTINE HAEMORRHAGE

G20.PONTINE HAEMORRHAGE

G21.CROW FEET(SPLINTER)HAEMORRHAGE IN PONS

G22.PONTINE HAEMORRHAGE

G23. CONTRECOUP LACERATION AND CONTUSION OF FRONTAL AND TEMPORAL LOBE

G24.COUP AND CONTRECOUP LESIONS (RTA)

G25.INTRA- CEREBRAL HAEMORRHAGE(SUDDEN NATURAL DEATH CASE)

G26.CONGESTED AND EDEMATOUS BRAIN

G27.TRAUMATIC INTRA CEREBRAL HAEMORRHAGE AT PERIPHERY AND BASAL GANGLION AREA, MID BRAIN

G28. BRAIN-PRESSURE EFFECT(DEPRESSION)OVER LEFT FRONTO-PARIETAL AREA DUE TO EXTRA DURAL CLOT

G29.CONTRECOUP OVER LEFT FRONTAL AND TEMPORAL LOBES

G30.LACERATION OF CEREBELLUM

G31.CONTUSION OF LEFT PARIETO-TEMPORAL LOBES WITH SUBARACHNOID HAEMORRHAGE

G32.CEREBRAL AIR EMBOLISM

G33.CEREBRAL FAT EMBOLISM IN RTA

G34.CEREBRAL FAT EMBOLISM

H1.STAB INJURY HEART (PM NO.57/12)

H2. STAB INJURY HEART

H3.HEART STAB INJURY (SINGLE EDGED WEAPON)

H4.HEART STAB WOUND

H5.HEART STAB WOUND

H6.STAB INJURY TO ARCH OF AORTA

H7.LACERATED HEART IN RTA

H8.LACERATION OF RIGHT VENTRICLES BY SHEAR FORCES

H9.LACERATION HEART (RTA)

H10.POST INFARCTIVE RUPTURE OF HEART

H11. RUPTURE HEART

H12.RUPTURE HEART

H13.COIN CONTUSION IN ELECTROCUTION

H14.COIN CONTUSION IN LIGHTENING

I 1.CIRRHOSIS LIVER

I 2.EARLY CIRRHOSIS OF LIVER-CHRONIC ALCOHOLIC CASE (MICRO NODULAR)

I 3.CIRRHOSIS OF LIVER

I 4.CIRRHOSIS OFLIVER

I 5.FATTY LIVER

J 1.LACERATION OFLIVER

J 2.LACERATION OF LIVER

J3.TRANSCAPSULAR MULTIPLE LACERATION OF LIVER (RTA)

J4. LACERATION OF LIVER WITH CONTUSION

J5. NON-COMMUNICATING LACERATION OF LIVER

J6. TRANSCAPSULAR LACERATION OF SUPERIOR SURFACE OF LIVER

J7. CONTUSION OF LIVER

J8. LIVER-STAB INJURY (BY DOUBLE EDGED WEAPON)

J9. LIVER STAB INJURY (BY SINGLE EDGED WEAPON)

K1. CONTUSION OF LUNG

K2. LIGHTENING CASE-LUNGS WITH PARENCHYMAL CONTUSION DUE TO PASSAGE OF CURRENT

K3 CONTUSION OF LUNG

K4 CONTUSION OF LUNG

K5 CONTUSION OF LUNG

K6 CONTUSION OF LUNG

K7 CONTUSION OF LUNG

K8 CONTUSION OF RIGHT BASE OF LUNG

K9 CONTUSION OF BOTH LOBES OF LEFT LUNG

K10 CONTUSION OF LUNG

K11 BULLAE IN LUNG

K12 LUNG SPINDLE SHAPED STAB INJURY (DOUBLE EDGED WEAPON)

K13 LACERATION OF LUNG

K14 LACERATION OF RIGHT LUNG

L1 GASTRIC EROSIONS SEQUELAE OF HEAD INJURY. (PM No:30/10)

M1 AIRWAY DISSECTED-(INDICATION BURNS/DROWNING: PM NO.81/11)

N1 LOBULATED KIDNEY

N2 LOBULATED KIDNEY (CONGENITAL ANOMALY INCIDENTAL FINDING IN RTA)

N3 KIDNEY: MULTIPLE LACERATION WITH PERINEPHRIC CONTUSIONS

N4 PERINEPHRIC CONTUSION KIDNEY IN RTA (PM NO.94/10)

N5 KIDNEY (PALE,TRAUMATIC SHOCK: PM NO.10/12)

N6 POLYCYSTIC KIDNEY

N7 NEPHROSCLEROTIC KIDNEY IN HYPERTENSION

N8 HORSE SHOE KIDNEY

N9 LACERATION SPLEEN

N10 LACERATION SPLEEN

N11 SPLEEN -INCISED WOUND

O1 NON-POISONOUS SNAKE

O2 SAND-BOA (NON-POISONOUS SNAKE)

O3 SAND-BOA (NON-POISONOUS SNAKE)

O4 GREEN VINE SNAKE

O5 GREEN VINE SNAKE

O6 NON-POISONOUS SNAKE

O7 NON -POISONOUS SNAKE

O8 NON-POISONOUS SNAKE

O9 NON-POISONOUS SNAKE

O10 NON-POISONOUS SNAKE

O11 CENTIPEDE

O12 NON-POISONOUS SNAKE

O13 NON-POISONOUS SNAKE

O14 SCORPION

O15 SCORPION

O16 NON-POISONOUS SNAKE

O17 NON-POISONOUS SNAKE

O18 COMMON KRAIT

O19 NON-POISONOUS SNAKE

O20 POISONOUS SNAKE

O21 COMMON COBRA

O22 COMMON COBRA

O23 COMMON COBRA

O24 COMMON KRAIT

O25 RUSSELL'S VIPER

O26 RUSSELL'S VIPER

O27 RUSSELL'S VIPER

O28 BANDED KRAIT

O29 BANDED KRAIT

O30 RICINUS COMMUNIS

O31 CALOTROPIS GIGANTEA

O32 NERIUM ODORUM

O33 DATURA FASTUOSA

O34 ARGEMONE MEXICANA

O35 CALOTROPIS PLANT WITH FRUIT

O36 STOMACH IN FORMALIN POISONING (PM NO 78/10)

O37 TONGUE IN FORMALIN POISONING

O38 STOMACH IN SULPHURIC ACID POISONING

O39 OESOPHAGUS-STOMACH IN SULPHURIC ACID POISONING

O40 ULCER OVER STOMACH MUCOSA - CORROSIVE POISONING

P1 FOETUS LENGTH: 8.5CMS (2.5-3 MONTHS)

P2 FOETUS - 4 MONTHS

P3 FOETUS - 18 WEEK

P4 FOETUS - 5 MONTHS

P5 FOETUS- 6 MONTHS

P6 FOETUS WITH PLACENTA-7 MONTHS

P7 FEMALE FOETUS-8 MONTHS

P8 FOETUS - 9 MONTHS

P9 RESPIRED LUNGS

P10 UNRESPIRED LUNGS

P11 OSSIFICATION CENTRE FOR FOOT & STERNUM

P12 OSSIFICATION CENTRE FOR FOOT AND KNEE
P13 OSSIFICATION CENTRE FOR FOOT AND KNEE

P14 DECAPITATED HEAD

P15 UTERUS WITH MUMMIFIED FOETUS

P16 ANENCEPHALY (NEURAL TUBE DEFECT)

P17 MULTIPLE CONGENITAL ANOMALIES (A) GASTROSCHIS (B)BILATERAL CLEFT LIP (C)ANENCEPHALY

P18 DOLICOCEPHALUS

P19 RACHISCHISIS NEURAL TUBE DEFECT

Q1 NORMAL UTERUS WITH TUBES AND OVARIES

Q2 PLACENTA WITH UMBILICAL CORD

Q3 UTERUS WITH PRODUCTS OF CONCEPTION 4-6 WEEKS

Q4 UTERUS WITH PRODUCTS OF CONCEPTION

Q5 UTERUS WITH PRODUCTS OF CONCEPTION

Q6 FIBROID UTERUS

Q7 UTERUS WITH INTRAMURAL FIBROID

Q8 TESTIS WITH SPERMATIC CORD

Q9 TESTIS WITH SPERMATIC CORD (PMNO.09/12)

Q10 CONTUSION OF SCROTUM AND TESTIS

Q11 UTERUS WITH COPPER 'T'

Q12 COPPER T – UTERUS

Q13 OVARIAN CYSTS

Q14 OVARIAN CYSTS

Q15 UTERUS WITH OVARIAN CYSTS

R1 SOOT IN TRACHEA – BURN CASE

R2 SOOT IN TRACHEA

R3 SOOT IN TRACHEA

R4 SOOT IN TRACHEA

R5 NECK STRUCTURES

R6 NECK STRUCTURES WITH TRACHEOSTOMY WOUND

R7 CHOKING

R8 TRAUMATIC ASPHYXIA – SUBMUCOSAL PETECHIAE IN TRACHEA AND EPIGLOTTIS

R9 LIGATURE MARK

S1 CRUSH INJURY HAND

S2 BURR HOLE

S3 DEPRESSED FRACTURE

S4 OBLITERATED SUTURE WITH PACCHIONIAN DEPRESSION

S5 EXTRADURAL CLOT

S6 STERNUM - STAB WOUND

S7 FRACTURE OF STERNUM

S8 STERNAL END OF CLAVICLE: PENETRATING INJURY BY PELLET

S9 TRANSVERSE FRACTURE OF STERNUM

S10 POST MORTEM BURNS OVER LEG

S11 STAB INJURY THROUGH RIGHT 3rd INTERCOSTAL SPACE

S12 SPLIT LACERATION OF SCALP

S13 RETROSTERNAL CONTUSION

T1 LUNGS AND SPLEEN

T2 LUNGS

T3 BRAIN CUT SECTION

T4 HAEMORRHAGE IN INTERNAL CAPSULE

T5 HAEMORRHAGE SPOTS IN LEFT VENTRICLE: ENDOCARDIUM

T6 ENTRY WOUND OF RIFLED FIRE ARM CLOSE RANGE – POWDER TATTOOING.

SPECIMEN NOTES

THANATOLOGY

A1, A2 :A PIECE OF FOAMY LIVER

- Foamy liver is a decomposition change after death.
- Noticed usually 2-3 days in summer & 5 to 6 days in winter after death.
- It is mainly because of Clostridium welchi, which produces gas.
- It forms clumps in tissue and are first small, multiple, opaque yellowish grey, dendritic areas, when gas is evolved the organ will have honey comb, vesicular or foamy appearance.

Medicolegal importance:

1. Evidence of decomposition.
2. Helps in determining time since death.

A3,A4 : MUMMIFIED FOOT

Definition: It is a modification of putrefaction by dehydration or desiccation (drying) and shrinkage of body.This occurs because of evaporation of water content of body.

Distribution: First appears over exposed parts of body like face, hands, feet, limbs, later it can spread all over and even internal organs.

Features: Skin is dry, leathery, rusty brown or black closely adhered to bones.

Climatic condition: It develops, if body is exposed to dry, hot climate, in sandy areas.It can also occur in freezing condition because of no air and no proliferation of organisms.

Marked dehydration by cholera, gastroenteritis before death will help formation of mummification. Chronic Arsenic or Antimony poisoning will favor formation of mummification.
Time of onset: About 3 months to 1 year. A single body can show both adipocere & Mummification.

Medico Legal importance:

1. Time since death
2. Climatic condition, nature of burial
3. Identity
4. Recognition of Injuries, poisons, help in diagnosing cause of death.

A5: ADIPOCERE OF HAND

Definition: It is a modification of putrefaction where in fatty tissues are converted into higher fatty acids. It is also called Saponification.

There is gradual hydrolysis and hydrogenation of fats to higher fatty acids.Lecithinase released by Cl. welchi helps in hydrolysis.

Factors influencing onset of adipocere:

1. Intrinsic lipase & bacterial enzymes (Lecithinase).
2. Water for hydrolysis.
3. Body buried in damp &moist areas.

Properties of adipocere:

a) It is greyish white or brownish in color and has rancid or sweetish odor.
b) Fresh adipocere is soft, greasy &moist, old adipocere is dry, brittle & easily breaks.
c) It burns with a yellow flame.
d) It floats in water and it dissolves in ether & alcohol.

Distribution: It first appears in fatty tissue and can be seen all over body. Muscles are pinkish; organs are preserved for longer period.

Time of Onset: In temperate climate as early as 3 weeks in summer.

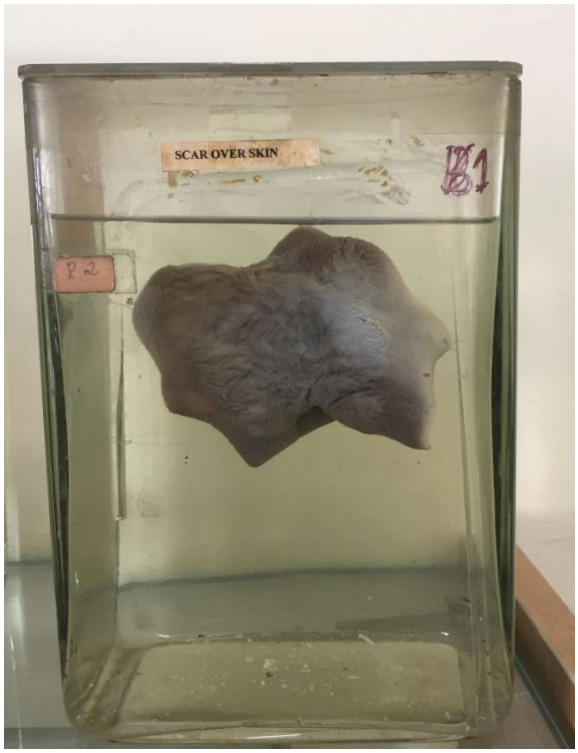
In India seen as early as 3 days &all over the body in 3-6 weeks.

Usually doesn't appear in a fetus less than 7 months as there is no fat.

Medico Legal importance:

1. Time since death
2. Climatic condition, nature of burial
3. Identity and Recognition of Injuries, poisons, help in diagnosing cause of death

IDENTIFICATION



B1: SCAR OVER SKIN

A scar is a fibrous tissue covered by epithelium, devoid of hair follicles, sweat glands, or pigment – which is produced from the healing of a wound.

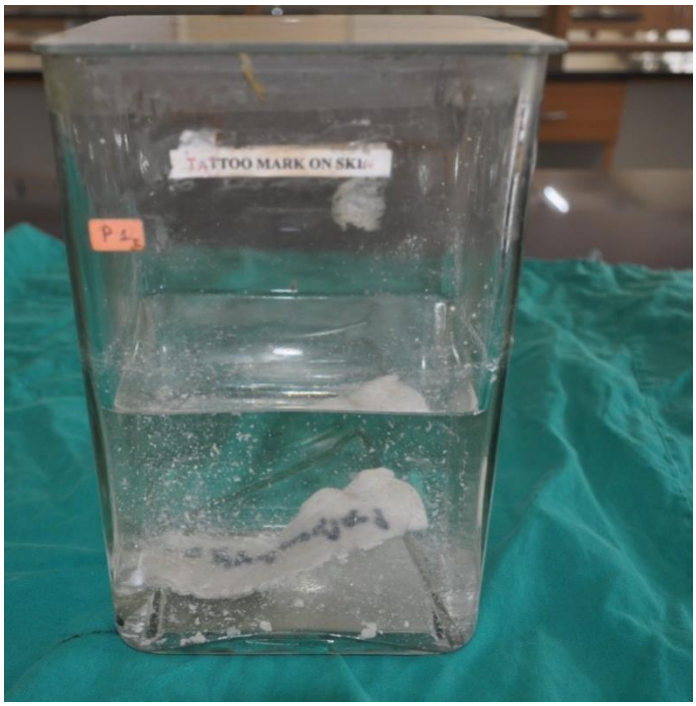
Injuries to the dermis produce a scar and are permanent.

Ageing of scars

- Vascular to avascular (2wk-2months)
- Tender to non- tender (2m-6months)
- Soft to thick (>6months)

Medicolegal importance

1. Important marks for identification.
2. The shape of the scar may indicate the type of weapon or agent that caused the injury.
3. Age of the scar can be found.
4. Multiple parallel scars over the wrist indicative of previous suicidal cuts.
5. Disfiguration of face due to scars is a grievous hurt (Sec 320 IPC).



B2 TATTOO OF A NAME OVER A PIECE OF SKIN

Tattooing (Polynesian) is the procedure of producing tattoo marks by dipping, a needle or penetrating object into dye & injecting into skin.

Dyes used: Indigo, Indian ink, Prussian blue, vermillion, cinnabar & ultramarine give good tattoo marks which can last for 10 – 15 yrs.

Practiced all over the world. In India it is common in tribals and in poor &lower socio economic status people.

The tattoo marks can be in the form of designs, names of god, goddess, dates, etc.,

Sites: Common over forehead, chin, arm, forearm.

Complication: when procedure is crude, done without aseptic precautions, there can be dermatitis, allergic reactions, abscess, gangrene, tetanus, leprosy, syphilis, AIDS, hepatitis, etc.,

Erasure: The tattoo can be removed by

1. Surgery
2. Burning the area
3. Electrolysis
4. CO_2 snow
5. Laser Beam
6. Use of Corrosives
7. Injury
8. Tattooing by white pigment titanium oxide

Medico – Legal importance:

1. Identity
2. Race, religion
3. Important events of life
4. Cobra, dragon, scorpion over criminals, gangsters.
5. Blue bird over 1st web space of hands in homosexuals
6. Tattoo over sclerosed veins in addicts.
7. Phrases like 'Born to Lose', 'Race with Devil' in addicts.
8. Speaks of history, war, travel, occupation, sex interest.
9. No. 13 over inner aspect of lower lip in drug peddlers.
10. Tattoo over knuckles &dorsum of 1st web space in women has a belief that food prepared by them is delicious and does not become stale.
11. In decomposed body, evidence of tattooing in regional area can be detected by seeing dye in regional lymph nodes.

NORMAL ORGANS

C1 TO C8: HEART

ANATOMY:

The heart is located between the lungs in the middles of the chest, behind and slightly to the left of the breastbone(sternum). A double layered membrane called the pericardium surrounds the heart like a sac. The outer layer of the pericardium surrounds the roots of the heart's major blood vessels and is attached by ligaments to the spinal column, diaphragm, and other parts of the body.

DIFFERENT WEIGHTS IN DIFFERENT GENDER:

Females: 225gm -285 gm

Males: 285gm - 340 gm

PRINCIPLE OF DISSECTION:

- The pulmonary arteries should be palpated before they are cut and looked for an embolus when they are incised.
- It is opened in the direction of flow of blood: **Inflow-outflow method.**

PROCEDURE OF DISSECTION:

The pericardium is examined and incised and any blood or fluid in pericardium is noted. The right atrium is cut between the openings of superior and inferior vena cava. The enterotome cuts through the tricuspid orifice and opens the right ventricle along the lateral margin. The enterotome is introduced into the right ventricle close to the apex, and the conus pulmonalis and pulmonary valve are cut along the interventricular septum in the anterior wall of the right ventricle. The incision should extend into the pulmonary trunks and the left pulmonary artery. The left atrium is cut between the openings of the pulmonary veins. Then, the left atrium is cut along its lateral wall. The incision extends through the mitral orifice and passes along lateral margin of left ventricle up to the apex. The next incision extends from the apex along the interventricular septum into the aorta, opening the aortic valve. Both auricular appendages should be examined for presence of thrombi. The heart should be weighed, and measurements should be taken of the circumference of the valves. Thickness of the right and left ventricle should be taken.

C9, C11: SPINAL CORD

ANATOMY:

- It is a cylindrical structure of nervous tissue composed of white and grey matter.
- It is uniformly organized and divided into five regions as:Cervical,Thoracic,Lumbar,Sacral,Coccygeal.
- Spinal cord consists of 31 pairs of spinal nerves include:
  - Cervical 8 pairs,Thoracic 12 pairs,Lumbar 5 pairs,Sacral 5 pairs and Coccygeal 1 pair.

PROCEDURE OF DISSECTION:

- An incision should be made from the occiput to sacrum in midline along the vertebral spines.
- The skin and muscles on the back should be dissected. The laminas of the vertebrae should be cut on either side and the spinal cord should be exposed and gently lifted.
- The opening of its covering should be severed and examined.
- The spinal canal need not be opened and examined unless an indication of disease or injury exists.

C10 : CEREBELLUM.C12, C13: BRAIN. C14 : PONS , T3 :BRAIN CUT SECTION

BRAIN:

ANATOMY:

- It is composed of cerebrum,cerebellum and brainstem.
- *Cerebrum:*
- Largest part of brain having right and left hemispheres.
- Cerebral hemispheres have distinct fissures which divide into lobes; they are Frontal,Temporal,Parietal and Occipital.
- *Cerebellum*:
- Located under cerebrum.
- *Brainstem:*
- Relay center connecting the cerebrum and cerebellum to the spinal cord.
- It is divided into Medulla, Pons and Midbrain.

PROCEDURE OF DISSECTION:

- After removal of skull cap exposes the duramater. It should be observed for the presence of extradural hemorrhage.
- The dura should be incised one cm on either side of the midline and remove the falxcerebri after releasing its attachment to crista galli.
- The remaining duramater should be reflected from the sides of the brain by cutting at its middle along coronal plane and observe for presence of subdural hemorrhage.
- The brain should be gently lifted up, from the base of cranium using fingers and nerves at the base are severed.
- The medulla should be cut as low as possible and later the brain is washed.
- After keeping the brain in anatomical position, large knife should be kept on corpus callosum at 45 degree angle and the cerebrum is cut.
- Then posterior part of brain should be turned upwards and multiple incisions should be given over the midbrain horizontally.
- The medulla oblongata should be incised vertically at its middle. The cerebellum should be examined after incising it horizontally or two vertical incisions oneither side. Later cerebrum should be cut horizontally at multiple places, to look for any evidence of intracerebral hemorrhage and pathological lesions.

C15 , C16, R5 : NECK STRUCTURES

- ✓ In cases of compression of neck, dissection of neck is done in bloodless field to look for contusion of muscles, fractures of hyoid bone, thyroid cartilage and tear in the carotid artery.
- ✓ In cases of drowning death, white , fine , leathery froth is noted in the airway.
- ✓ In choking deaths,Impaction of solid bodies occur in upper airway.
- ✓ Collection of saliva, mucous, oedema of nasal and pharyngeal mucosa occurs in death due to gagging.
- ✓ Bee stings, wasps, penicillin cause swelling of the laryngeal lining.

C17:HYOID BONE

Ossification: The hyoid is ossified from six centers: two for the body, and one for each cornu. Ossification commences in the greater cornua toward the end of fetal life, in the body shortly afterward, and in the lesser cornua during the first or second year after birth. In early life greater horn is connected to body by cartilage whereas after middle age the connection between the body and greater cornu is by bone.

Hyoid bone fracture:

Causes:

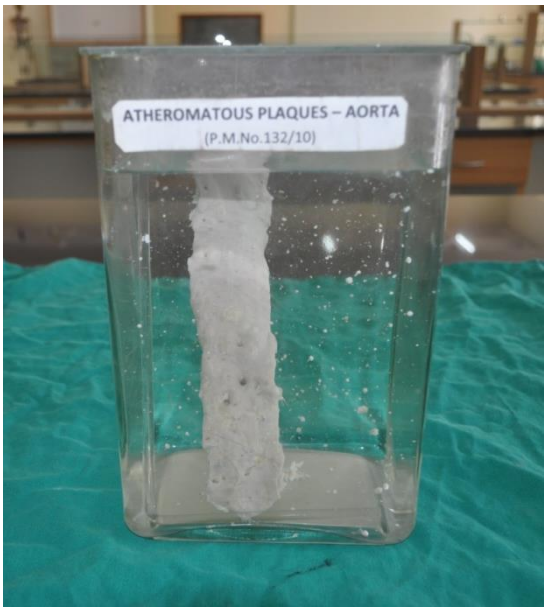
1) In case of manual strangulation, in younger persons since the horns are pliable they return to their normal position on release of pressure but, variably after third decade, they may be sufficiently calcified to fracture.
2) The ligature strangulation and hanging can also cause this lesion
3) Direct violence like punch or kick in the throat can break the cornuae
4) Blunt pressure can fracture the horns, such as arm-lock behind squeezing the larynx against the cervical spine.
5) Sudden hyper-extension of the neck

Types:

1) **Inward compression fracture**

2) **Anteroposterior compression fracture:**

3) **Avulsion fracture**



D1:ATHEROMATOUS PLAQUE-AORTA

Aortic atheromas (aortic atheromatous plaques) are defined by an irregular thickening of the intima ≥2 mm, and a complex plaque is defined as a protruding atheroma ≥4 mm with or without an attached mobile component. Stroke incidence is approximately 25% in patients with mobile plaques of the aortic arch and 2% in patients with quiescent nonmobile plaques.

D2,D3:CORONARY STENT (IN-SITU)

Angioplasty is a procedure to open narrowed or blocked blood vessels that supply blood to the heart. These blood vessels are called the coronary arteries.

A coronary artery stent is a small, metal mesh tube that expands inside a coronary artery. It isoften placed during or immediately after angioplasty. It helps prevent the artery from closing up again. A drug-eluting stent has medicine embedded in it that helps prevent the artery from closing in the long term.

ENLARGED ORGANS

E1. FATTY HEART E2.HYPERTROPHIED HEART (460 GM)

Cardiac hypertrophy is the abnormal enlargement, or thickening, of the heart muscle, resulting from increases in cardiomyocyte size and changes in other heart muscle components, such as extracellular matrix. Causes can be physiological – for example, the amount of exercise performed by an athlete – or pathological – for example, as a result of hypertension or valvular disease.

E3.HEPATOMEGALY WITH CIRRHOSIS AND SPLENOMEGALY(CHRONIC MYELOID LEUKEMIA CASE)

Liver involvement is often observed in several hematological disorders, resulting in abnormal liver function tests, abnormalities in liver imaging studies, or clinical symptoms presenting with hepatic manifestations. In hematologic malignancies, malignant cells often infiltrate the liver and may demonstrate abnormal liver function test results accompanied by hepatosplenomegaly or formation of multiple nodules in the liver and/or spleen. These cases may further evolve into fulminant hepatic failure.

E4. HEPATOMEGALY WITH CIRRHOSIS, E6.HEPATOMEGALY

Hepatomegaly is enlargement of the liver beyond its normal size and occurs mainly as a consequence of pathologic conditions

Liver diseases

- Cirrhosis
- Hepatitis caused by a virus — including hepatitis A, B and C — or caused by infectious mononucleosis
- Nonalcoholic/ Alcoholic fatty liver disease
- Amyloidosis
- Wilson's disease
- Hemachromatosis
- Gaucher's disease
- Liver cysts
- Noncancerous liver tumors, including hemangioma and adenoma
- Obstruction of the gallbladder or bile ducts
- Toxic hepatitis

Cancers

- Leukemia
- Liver cancer
- Lymphoma

Heart and blood vessel problems

- Budd-Chiari syndrome
- Heart failure
- Pericarditis

E5,N6.POLYCYSTIC KIDNEY

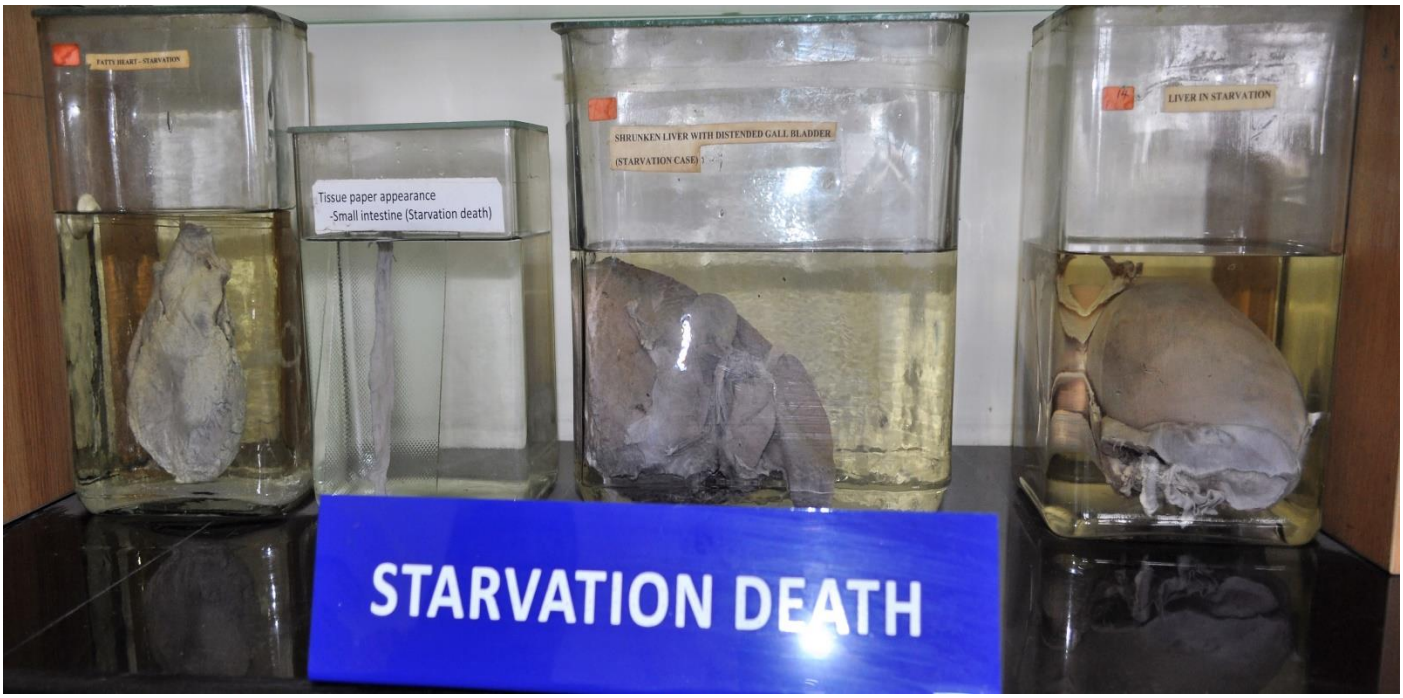
Cystic kidneys are common causes of end-stage renal disease, both in children and in adults. Autosomal dominant polycystic kidney disease (ADPKD) and autosomal recessive polycystic kidney disease (ARPKD) are cilia-related disorders and the two main forms of monogenic cystic kidney diseases. ADPKD is a common disease that mostly presents in adults, whereas ARPKD is a rarer and often more severe form of polycystic kidney disease (PKD) that usually presents perinatally or in early childhood.

E7.ENLARGED SPLEEN

Causes:

- Viral infections, such as mononucleosis
- Bacterial infections, such as syphilis or an infection of your heart's inner lining (endocarditis)
- Parasitic infections, such as malaria
- Cirrhosis and other diseases affecting the liver
- Various types of hemolyticanemia
- Blood cancers, such as leukemia and myeloproliferative neoplasms, and lymphomas, such as Hodgkin's disease
- Metabolic disorders, such as Gaucher's disease and Niemann-Pick disease
- Pressure on the veins in the spleen or liver or a blood clot in these veins

STARVATION DEATH



F1, F2:.LIVER IN STARVATION

In Starvation deaths, there is general reduction in the size and weight .The liver is atrophied and may show necrosis due to protein deficiency. Gall bladder is distended.

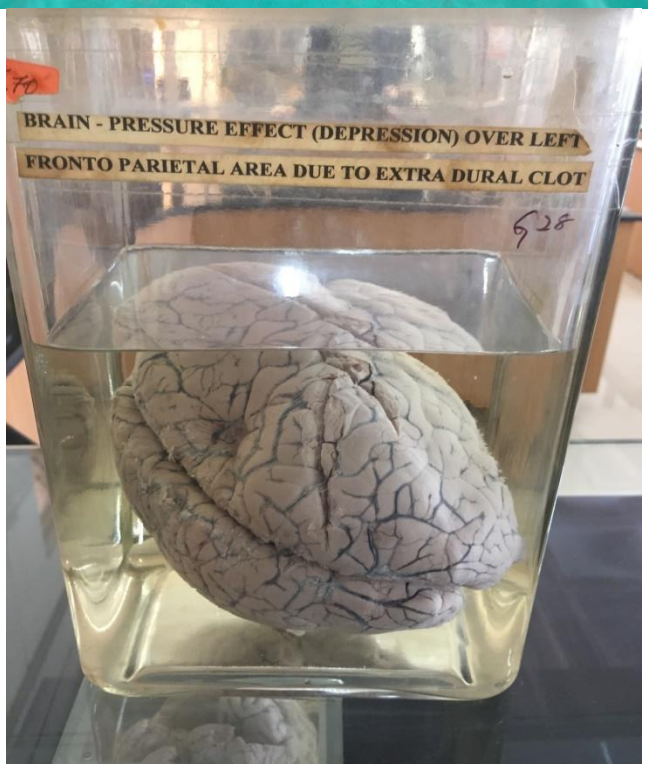
F3.TISSUE PAPER APPEARANCE-SMALL INTESTINE

The stomach and intestines show atrophy of all coats and the mucosa is stained with bile in cases of starvation. . The walls of the intestine appear like tissue paper with atrophy of mucosa. The bowel contains offensive fluid and gas. There may be superficial but extensive non- specific ulceration of the bowel like those seen in ulcerative colitis**.**

F4. HEART- STARVATION

In cases of starvation, blood volume is greatly reduced and there is marked anemia. The heart is small from brown atrophy and th chambers would be empty.

CEREBRAL INJURIES



G1 – G3, S5: EXTRADURAL CLOT,G28.BRAIN-PRESSURE EFFECT (DEPRESSION)OVER LEFT FRONTO-PARIETAL AREA DUE TO EXTRA DURAL CLOT

Extradural or epidural clot is said to be almost always traumatic. Occurs at the site of impact. It is often associated with complete fissured or depressed fracture.

Site: Commonest site is temporoparietal area with rupture of middle meningeal artery.

Other sites where it can be seen is over frontal , occipital lobes and rarely over the base.

Age: Commonly seen in age group of 25 years to 45 years, usually not seen in child below 2 years because dura is firmly adhered and there is no bony groove for vessel.

The clot is said to contain often arterial blood. But venous blood can be there if meningeal veins, diploic veins, dural sinuses are involved.

Symptomatology: Person may have temporary unconsciousness, regains and loses consciousness again which is termed as **lucid interval.**Hemiparesis, Hemiplegia can be present.

Medicolegal significance

1. Doctor may be charged with negligence→if patient discharged during lucid interval & dies later.
2. EDH on the contralateral side should be carefully excluded.
3. Condition may resemble drunkenness & patient may die in police custody.
4. Helpful in alcohol & drugs estimation.

G4, G5, G7 TO G11: SPECIMENS OF BRAIN WITH DIFFUSE SUBARCHNOID HAEMORRHAGEG31.CONTUSION OF LEFT PARIETO-TEMPORAL LOBES WITH SUBARACHNOID HAEMORRHAGE

This is the commonest type of meningeal bleeding

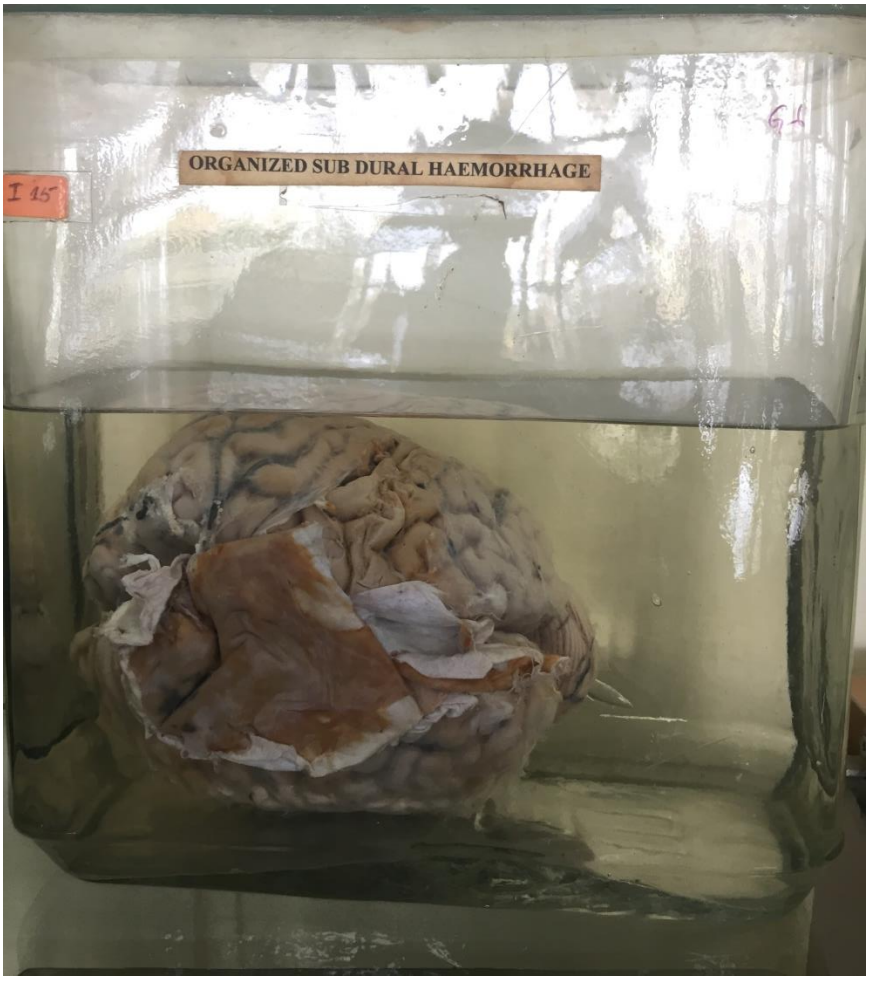
Types: - Immediate (Acute)
- Delayed (Reactionary)

Causes:
1. Rupture of brain vessels
2. Laceration of brain
3. Rupture of saccular – Berry's aneurgysm's of the circle of willis.
4. Bleeding of tumours
5. Asphyxial deaths (Hanging, drowning, strangulation etc.,)
6. Blood dyscrasias (Leukemia, Purpu a etc.,)
7. Vertebral artery rupture.

Site: Often bilateral all over cerebral hemispheres, in mild form around contused& lacerated brain.

Symptomatology: Headache, irritability, softness of neck, photophobia, Deterioration of consciousness.

At Autopsy: One can differentiate subdural and subarachnoid hemorrhage by gently washing brain by a delicate stream of water.Subdural clots will be washed and subarachnoid which is below piamater persists.



G6, G12: SUBDURAL HAEMORRHAGE

- Blood accumulates between dura & arachnoid.
- More commonly seen in falls & assaults than vehicle accidents.
- Commoner - in children and old age.
- With head trauma, the dura moves with the skull, and the arachnoid moves with the cerebrum, . As a result, the bridging veins are sheared causing hematoma in the expansile subdural space.
- It could occur anywhere however commonly seen in supratentorial areas and upper lateral surface.
- Collection of 100- 150 ml is associated with fatality.
- Source of bleed is Essentially venous or capillary.

G13 TO G22 :PONTINE HAEMORRHAGE

The pontine hemorrhage is thebleeding in pons and it can be due to trauma or disease.

Signs of Pontine hemorrhage:The three ‘P’s are **1 .Paralysis 2. Pin point pupil 3. Pyrexia**

Difference between traumatic pontine hemorrhage and pontine hemorrhage due to disease

Traumatic

1.Age : Any age `

2.H/o accident

3.Associated with other head injuries

4.Usually multiple and coalesce if patient survives for few days or scattered on one side

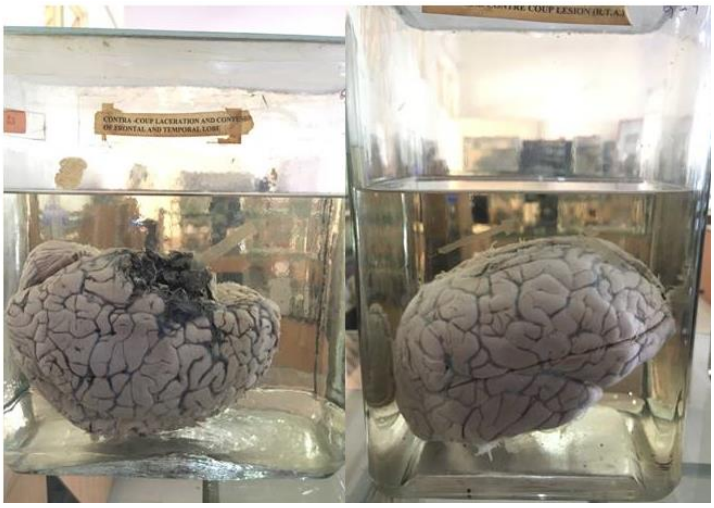
Disease

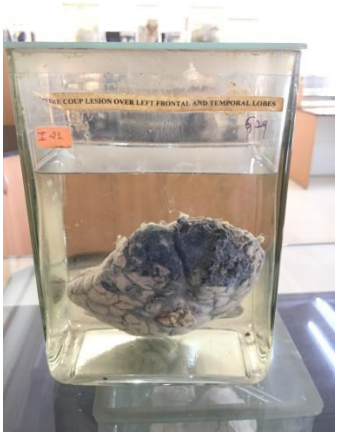
1.Old age

2. H/o Hypertension, Diabetes, Tumors, Bleeding disorder etc

3. Hemorrhage in basal ganglia and internal capsule

4.Occupy ½ or 1/3 of pons or linear in midline





G23.CONTRECOUP LACERATION AND CONTUSION OF FRONTAL AND TEMPORAL LOBEG24.COUP AND CONTRECOUP LESIONS (RTA) G29.CONTRECOUP OVER LEFT FRONTAL AND TEMPORAL LOBES

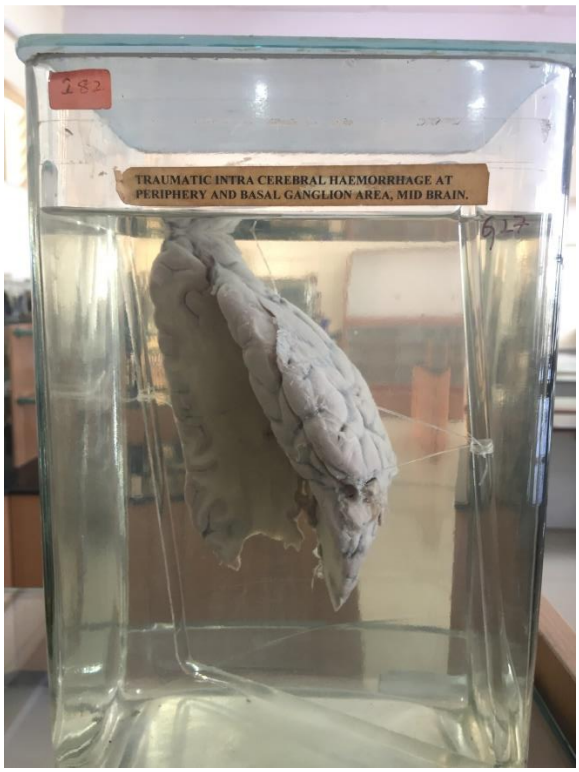
COUP INJURY

- ✓ The injury is located beneath the area of impact & results directly by the impacting force.
- ✓ Head is static.

CONTRE COUP INJURY

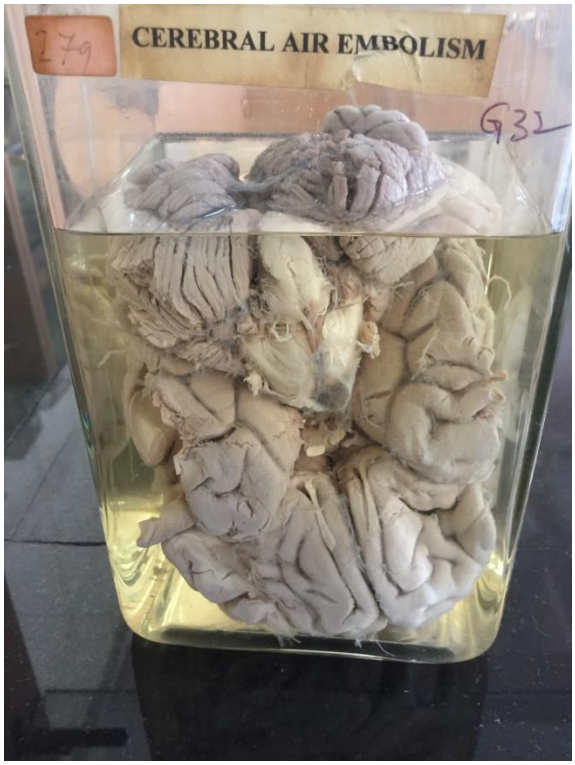
- ✓ Lesion is present in an area opposite to the side of impact.
- ✓ Head in motion.

- ✓ Contrecoup injury can occur without coup lesion.
- ✓ Tips and undersurface of frontal and temporal lobe – common location.
- ✓ Temporal impact – contrecoup in ipsilateral hemisphere.
- ✓ Extent of contrecoup – disproportionate to coup damage.



G25.INTRA- CEREBRAL HAEMORRHAGE (SUDDEN NATURAL DEATH CASE)G27.TRAUMATIC INTRA CEREBRAL HAEMORRHAGEAT PERIPHERYAND BASAL GANGLION AREA,MID BRAIN,T4 HAEMORRHAGE IN INTERNAL CAPSULE

- Intracerebral hemorrhage is characterized clinically by an abrupt onset and rapid evolution
- Intracerebral hemorrhages are uncommon in the younger age groups.
- Hypertension is virtually always present. There is usually only one episode of hemorrhage at the time of the attack. Recurrence of bleeding is not usually encountered.
- The primary sites for intracerebral hemorrhages are the putamen and adjacent internal capsule, the thalamus, the cerebellar hemispheres, the pons, and the white matter
- In intracerebral hemorrhage, the brain is asymmetrically swollen, with the swollen hemisphere containing the hemorrhage.
- On sectioning, the brain tissue adjacent to the hemorrhage is swollen and edematous.
- Death is generally due to compression and distortion of the midbrain, or hemorrhage into the ventricles.



G32.CEREBRAL AIR EMBOLISM

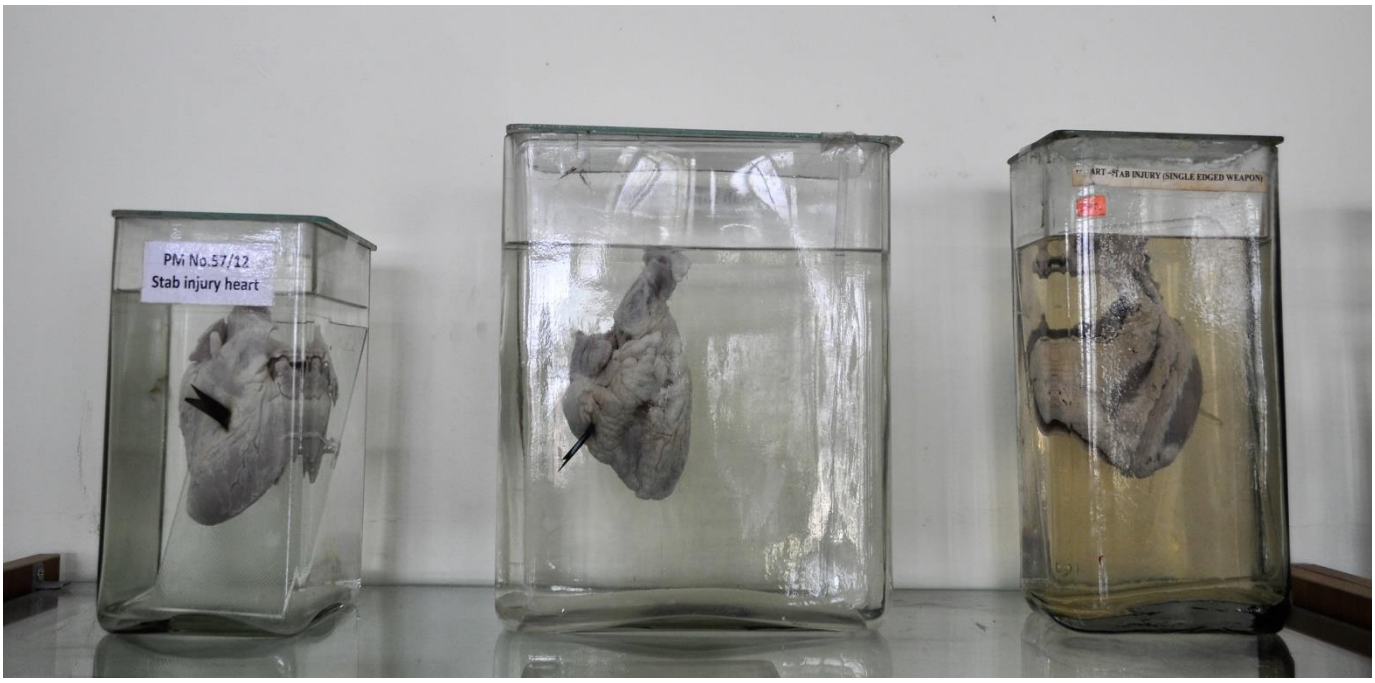
- If systemic air embolism is suspected the head should be opened first and the surface vessels of the brain examined for gas bubbles.
- In acute cases, gas bubbles will be visible within cerebral arteries but not in the cortical veins and the presence of gas bubbles in the pial veins is an artefact.
- The brain should be lifted gently and small artery forceps are used to gently clamp the intracranial part of both internal carotid and vertebral arteries. The vessels should be cut from below and then the brain is removed in usual manner.
- Then the brain is submerged in the water, ligatures released, and vessels slightly compressed to look for air bubbles.

G33, G34 CEREBRAL FAT EMBOLISM

The condition develops when fat emboli are forced from the pulmonary capillaries into systemic circulation in sufficient quantity to affect the brain.

The clinical history is fairly characteristic about 16-48 hrs after the injury, the patient exhibits a large number of petechial hemorrhages in small clusters in the skin of shoulder, neck and upper extremities and also slips into coma. At autopsy the brain is filled with small petechial hemorrhage grouped in clusters confined strictly to white matter.

CARDIAC INJURIES



H1 to H6: STAB INJURY TO HEART

- The stab injuries are due to any penetrating object or fractured ribs.
- The oblique stab injury to left ventricle may act as valve and bleed less.
- The stab injury to right ventricle and auricles bleed profusely and are dangerous giving no time for surgical intervention.
- **Cardiac tamponade:** There is a sudden accumulation of 300-400 ml of blood or fluid in the pericardial sac , which interferes with functioning of heart . The heart fails to dilate and contracts and hence fails to pump blood effectively. This leads to death by syncope.
- If pericardium is injured as in stab injuries,lacerations, person bleeds to death. Cause of death being hypovolemic shock.

H7 to H9: LACERATED HEART IN RTA

Traumatic lacerations of heart are due to blunt force violence to chest; fractured ribs and sternum.

The oblique lacerations of left ventricle act as valve and there is slow bleeding. Auricular lacerations bleed profusely,hence more dangerous.

The common sites of traumatic rupture are right auricle, right ventricle, left auricle, interventricular septum and valves.

H10 to H12: POST INFARCTIVE RUPTURE OF HEART

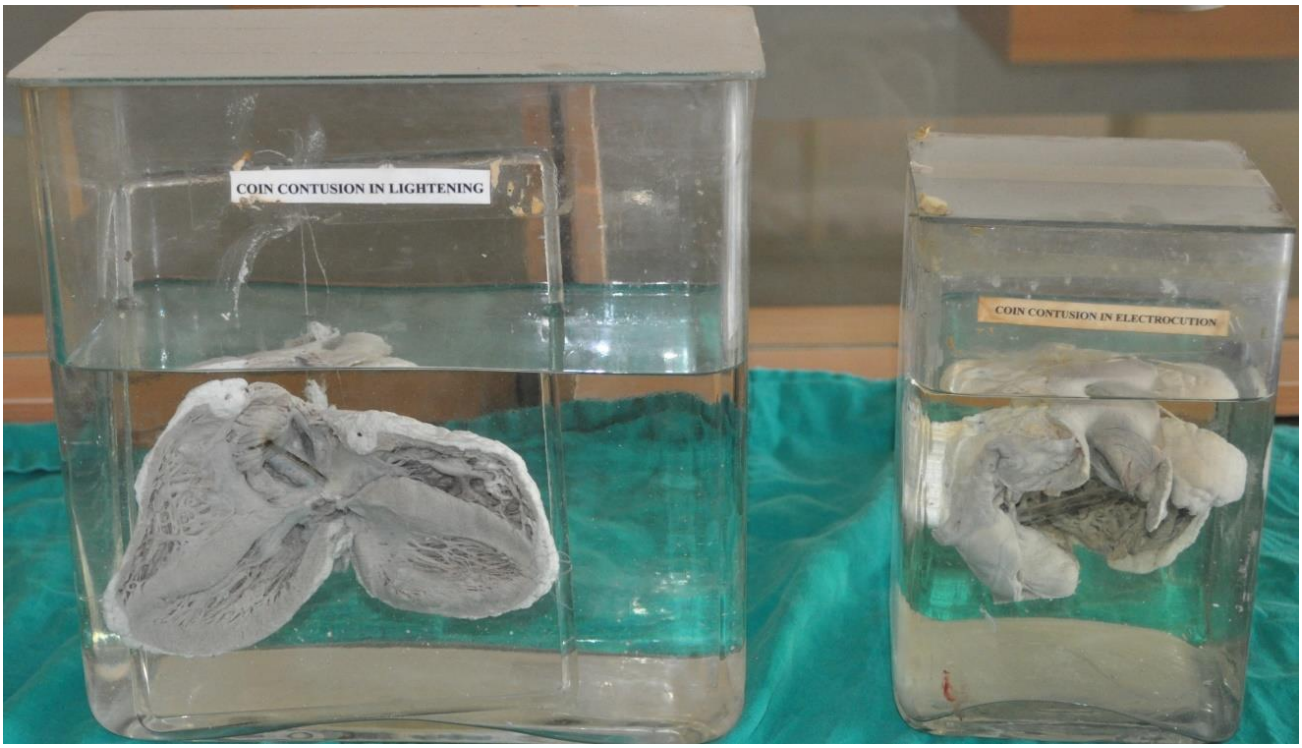
The rupture occurs at the site of infarction where in the tissues are weakened, fibrosed and thinned out.

Post infarction ruptures can occur within few days after infarction, where there is cardiomalacia.

On a later date, ruptures are also known to occur at the site of healed fibrosed infarct. Usually emotional disturbances, sudden rise in pressure precipitate ruptures.

The cause of death is due to cardiac tamponade.

The histopathological examination at the site of ruptures confirms myocardial infraction in doubtful cases.



H13, H14: COIN CONTUSION ELECTROCUTION / LIGHTENING

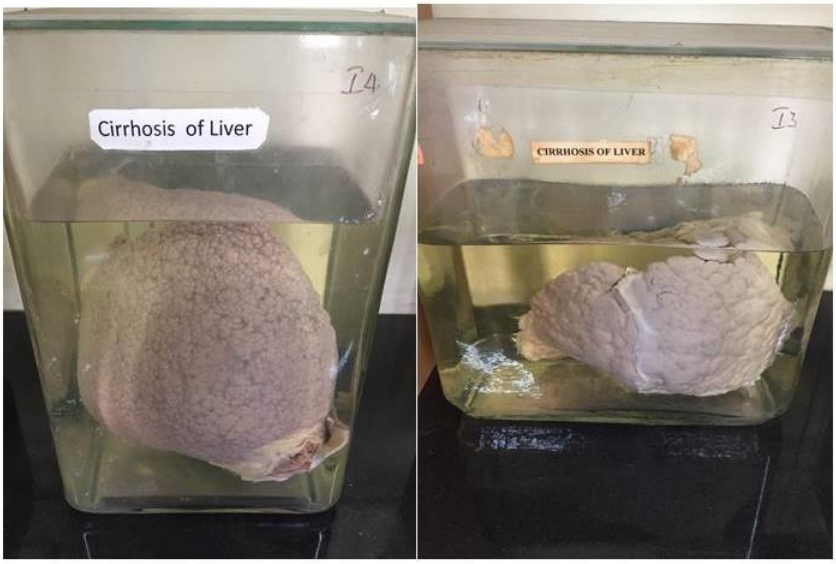
- When energy passes through this organ during electrocution:

-Heart stops contracting.

-Ventricular fibrillation.

- If high voltage current seizes quickly, then the heart may start beating again by itself.
- At autopsy, there could be petechial haemorrhages or contusion on the surface of heart.
- Small contusion in heart can cause serious disturbance of normal rhythm or stoppage of cardiac action and death.
- Large contusions can prevent adequate cardiac emptying and lead to heart failure.

HEPATIC PATHOLOGY



I1-I4 : CIRRHOSIS OF LIVER

Cirrhosis is a consequence of chronic liver disease characterized by replacement of liver tissue by fibrosis, scar tissue and regenerative nodules (lumps that occur as a result of a process in which damaged tissue is regenerated), leading to loss of liver function.

GROSS INSPECTION

Grossly, with the naked eye, a cirrhotic liver appears nodular, "hub-nailed", on the external surface and nodular on the cut surface. Variation in size, color, shape and consistency is relevant and may help in the identification of the etiology. The liver is usually indurated shrunken and yellowish-tan but it may be enlarged and yellow as in alcoholic fatty cirrhosis, rusty as in hemochromatosis or large and green as in biliary obstruction .

MACRONODULAR CIRRHOSIS: Larger nodules separated by wider scars and irregularly distributed throughout the liver usually due to an infectious agent such as viral hepatitis which does not diffuse uniformly throughout the liver

MICRONDULAR CIRRHOSIS: Small rather uniform 2 m nodules seperated by thin fibrous septa usually due to a chemical agent as alcohol which diffuse uniformly throughout the liver.

I5 : FATTY LIVER

Fatty Change in Liver Gross

• The liver is enlarged and yellow with tense, glistening capsule and rounded margins.

• The cut surface bulges slightly and is pale-yellow and greasy to touch

Fatty Change in Liver Microscopy

i. Fat in the cytoplasm of the hepatocytes is seen as clear area which may vary from minute droplets in the cytoplasm of a few hepatocytes (microvesicular) to distention of the entire cytoplasm of most cells by coalesced droplets (macrovesicular) pushing the nucleus to periphery of the cell.

ii. When steatosis is mild, centrilobular hepatocytes are mainly affected, while the progressive accumulation of fat involves the entire lobule.

iii. Occasionally, the adjacent cells containing fat rupture and produce fatty cysts. iv. Infrequently, lipogranulomas may appear consisting of collection of macrophages, lymphocytes and multinucleate giant cells.

v. Special stains such as Sudan III, Sudan IV, Sudan Black and Oil Red O can be employed to demonstrate fat in the tissue.

LIVER INJURIES

J1 TO J6: LACERATION OF LIVER

Laceration occurs due to blunt force violence, forcible fall, vehicular accidents etc. The fracture of ribs can pierce the liver and lacerate.

Lacerations of internal organs are due to:

a)Direct injury

b) Stretching of visceral attachments

c) Development of traction and shear strains

d)Hydrostatic forces developing in visceral fluid

e)Pneumatic force in viscera with air

Types of liver lacerations:

1) Trans capsular: over the convex surface of liver at the site of impact –Branch out in a cobweb fashion.

2) Sub capsular: over convex surface of liver at the site of impact.

3) Non communicating or central laceration: in deep substance of liver.

4) Coronal lacerations:over superior surface due to distortion.

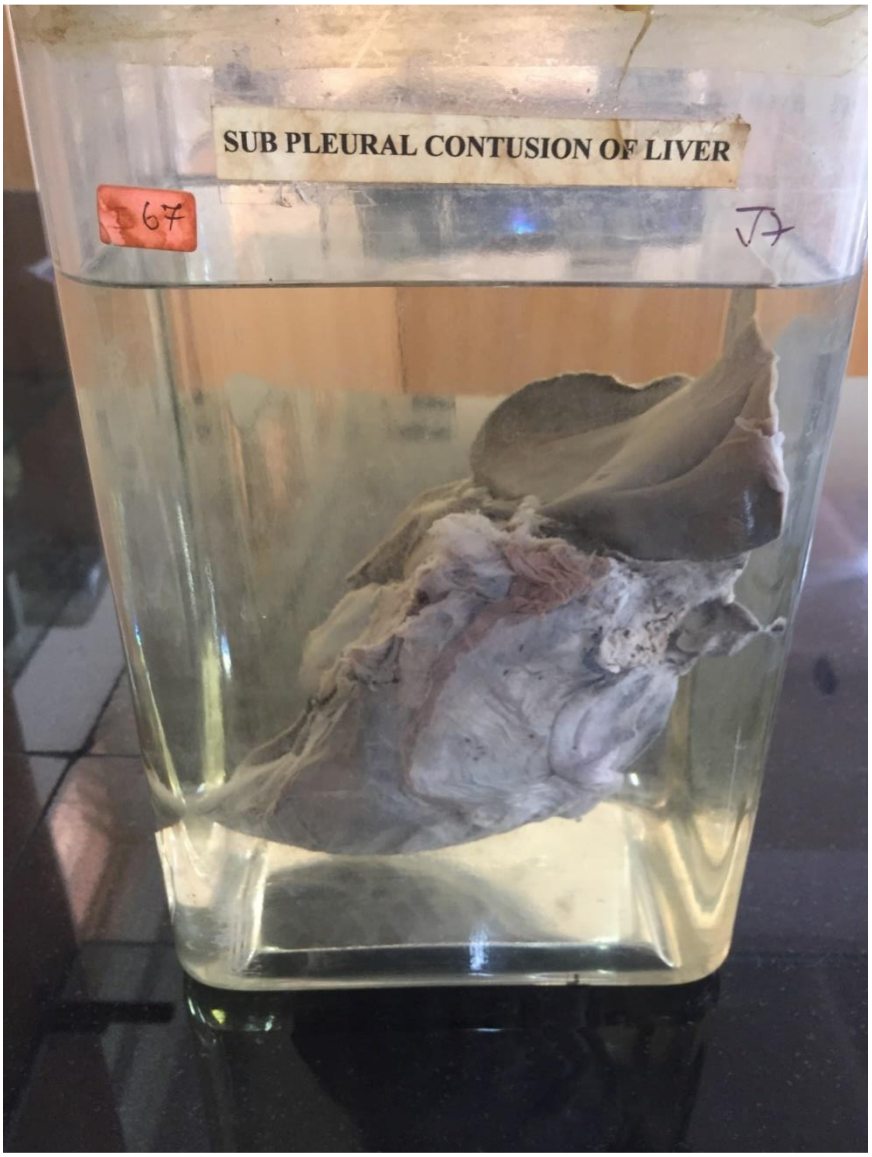
5) Inferior surface laceration due to distortion.

6) Contrecoup laceration at posterior surface with coup lacerations,contusion over anterior surface .

Right lobe is five times more prone for lacerations usually directed anteroposterior or obliquely.Liver lacerates with trivial trauma if enlarged due to diseases like tumours, fatty change,abscess,malaria ,etc.

Liver in children is large and abdominal organ and may lacerate or show subcapsularhematoma due to sudden pressure over chest during delivery.

Death can be due to shock and hemorrhage.Liver tissue may embolise leading to death by fatal pulmonary embolism.



J7. CONTUSION OF LIVER

- ✓ Can occur due to blunt force impact, leading to effusion of blood in to the tissues, due to rupture of blood vessels.
- ✓ Can cause death from shock and internal hemorrhage.
- ✓ The pooled blood can be a source of bacterial growth by clostridial group.

J8. LIVER-STAB INJURY (BY DOUBLE EDGED WEAPON). J9. LIVER STAB INJURY (BY SINGLE EDGED WEAPON)

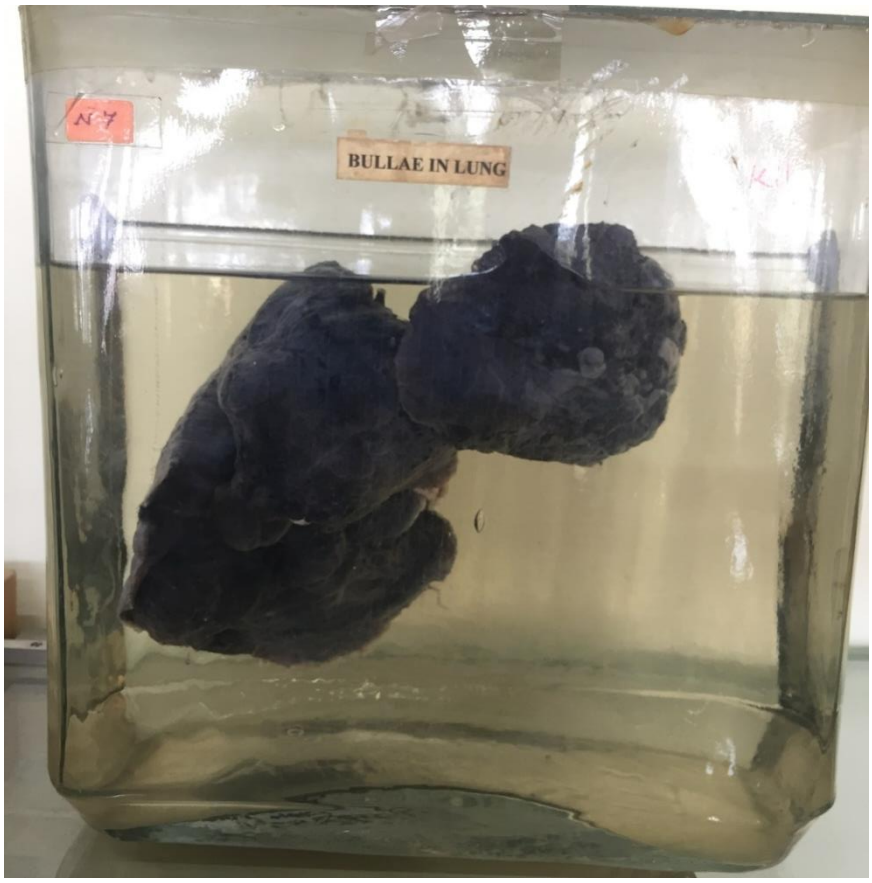
The shape of stab wound suggests the kind of weapon used. Wedge shaped entry wound is caused by a single edged penetrating weapon. The measurement and shape will help to some extent to trace the weapon. Double edged weapon causes elliptical entry wound.

The injury results in profuse bleeding leading to death by shock and hemorrhage.

LUNG INJURIES

K1 to K10:CONTUSION OF LUNG

- ✓ Can occur due to blunt force impact, leading to effusion of blood in to the tissues, due to rupture of blood vessels.
- ✓ Associated with rib and fracture of sternum.
- ✓ Can be an therapeutic artefact, caused during cardiopulmonary resuscitation.



K11BULLAE IN LUNG

- Bulla is a pathological entity caused by the confluence of two or more of the terminal elements of the bronchial tree.
- They may be found subsequent to any condition that interferes with normal respiratory mechanism that produces increased intra-alveolar pressure.
- A single bulla is a rarity: they vary in size from that of a large vesicle to that of a large grape fruit.
- There are two views as to the formation of bullae: both believe bullae to be the result of the increased intra-alveolar pressure that is present.
- There is no clinical sign or symptom characteristic of bullae.

K12LUNG SPINDLE SHAPED STAB INJURY (DOUBLE EDGED WEAPON)

- The shape of stab wound suggests the kind of weapon used. Spindle shaped entry wound is caused by a double edged penetrating weapon. The measurement and shape will help to some extent to trace the weapon.

- The stab wound could be either be penetrating or perforating type.This is a perforating type of stab wound, hence the description shall include wound of entrance, depth, direction of wound tract and specific termination.

- The injury results in pneumothorax

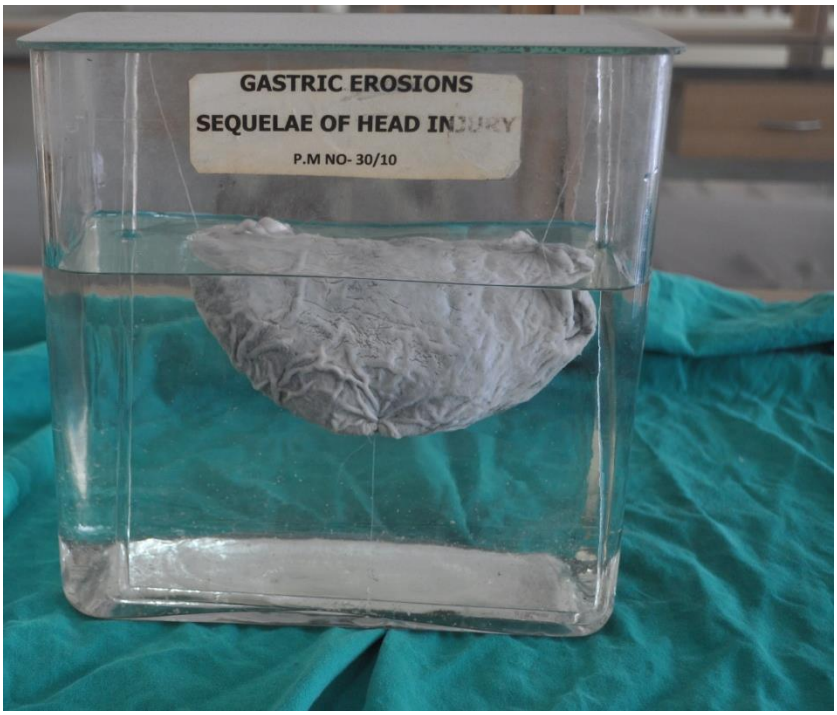
K13, K14: LACERATION OF LUNG

Caused by – Blows from blunt object.

- Fall on hard surface.
- Machinery accidents.
- Traffic accidents.

Mechanism of causation:

- Direct injury
- Development of traction shears
- Stretching of the visceral attachment
- Hydrostatic forces



L1GASTRIC EROSIONS SEQUELAE OF HEAD INJURY. (PM No:30/10)

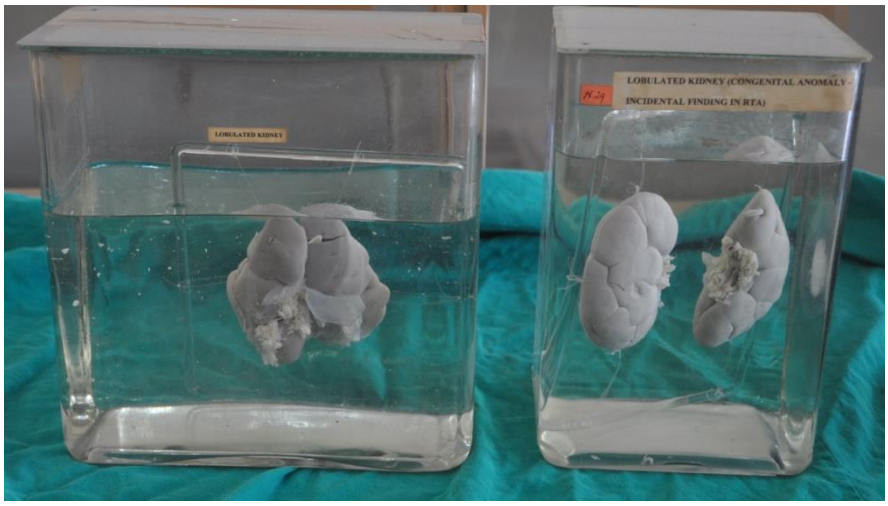
- Occurs due to elevated intracranial pressure. Can lead to ulceration of stomach mucosa termed as **Cushing ulcer**, named after Harvey Cushing.

- It is a gastric ulcer associated with elevated intracranial pressure. It is also called von Rokitansky-Cushing syndrome. Apart from in the stomach, ulcers may also develop in the proximal duodenum and distal esophagus

- The mechanism of development of Cushing ulcers is thought to be due to direct stimulation of vagal nuclei as a result of increased intracranial pressure. Alternatively, it may also be a direct result of Cushing reaction. Efferent fibers of the vagus nerve then release acetylcholine onto gastric parietal cell M3 receptors, causing insertion of hydrogen potassium ATPase vesicles into the apical plasma membrane. The end result is increased secretion of gastric acid with eventual ulceration of the gastric mucosa.

M1 AIRWAY DISSECTED-(INDICATION BURNS/DROWNING: PM NO.81/11)

- Dissection of airway is done to look for presence of froth in cases of drowning
- In cases of burns, it is to look for presence of soot particles.



N1 N2 :LOBULATED KIDNEY

Persistent fetal lobulation is a normal variant seen occasionally in adult kidneys. It occurs when there is incomplete fusion of the developing renal lobules. Embryologically, the kidneys originate as distinct lobules that fuse as they develop and grow.

KIDNEY AND SPLEEN INJURIES

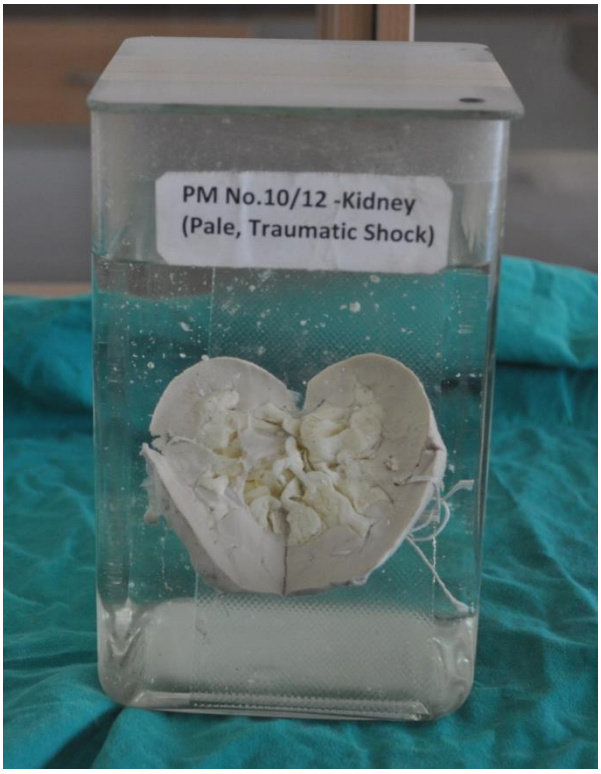
N3, N4: KIDNEY (CONTUSION /LACERATION)

Caused by – Blows from blunt object.

- Fall on hard surface.
- Machinery accidents.
- Traffic accidents.

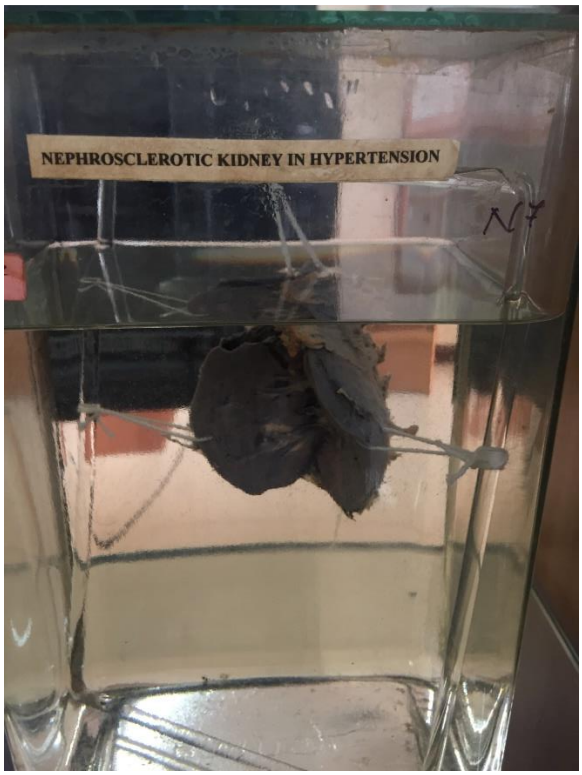
Mechanism of causation of laceration:

- Direct injury
- Development of traction shears
- Stretching of the visceral attachment
- Hydrostatic forces



N5 KIDNEY (PALE ,TRAUMATIC SHOCK: PM NO.10/12)

- The specimen of kidney, on gross examination appears pale due to loss of blood leading to death due to hypovolemic shock.
- **Hematogenic shock or traumatic shock:** It occurs when there is reduction in the circulating blood volume due to blood or fluid loss or hemorrhages more than 35 percent of the total blood volume, fluid loss due to neonatal calf diarrhea or colitis-X, acute intestinal obstruction and dehydration. E.g. trauma, extensive burn, rough handling of visceral organs during surgery.

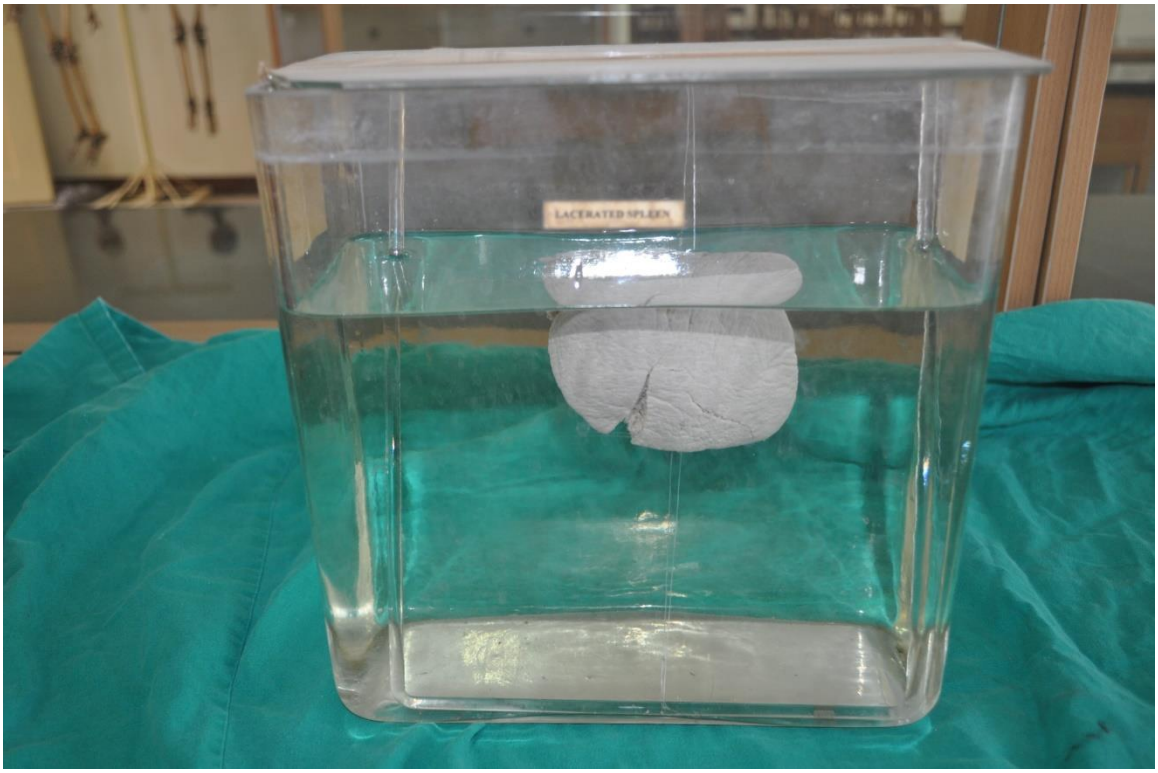


N7 NEPHROSCLEROTIC KIDNEY IN HYPERTENSION

- Chronic hypertension results in arterial nephrosclerosis with thickening of large and small blood vessels in the kidney by atherosclerosis.
- Disease of larger blood vessels can result in segmental infarcts of the kidney (small or large), which produce the typical pattern of arterial nephrosclerosis with small indentations in the renal cortex that are visible on gross examination.
- These indentations result from the loss of parenchymal tissue owing to small, discrete infarcts and the contraction of fibrous scar tissue, producing a football-like appearance.
- The thickening of afferent arterioles to glomeruli can ultimately result in loss of glomerular function and devascularization of glomeruli.

N8 HORSE SHOE KIDNEY

Horseshoe kidney, also known as renarcuatus (in Latin), **renal**fusion or super **kidney**, is a congenital disorder affecting about 1 in 500 people that is more common in men, often asymptomatic, and usually diagnosed incidentally.



N9, N10:LACERATION OF SPLEEN

These are due to blunt force violence to abdomen. More common in left hypochondriac area, fall, traffic accidents & injury by fractured ribs, they bleed profully dangerous and often fatal.

Site: Lacerations are more common over concave surface and often associated with other abdominal injuries.

Lacerations are often transcapsular.

Shape: They are often multiple & look like 'Y' 'H' or 'L'.

Trivial trauma & contraction of abdominal wall can result in rupture of enlarged vascular diseased spleen.

Spleen is enlarged in Malaria, Leukemia, Anaemia etc.,

Subcapsular bleeding results in slow accumulations of blood, pressure of the clot prevents further bleeding.

Death is delayed & occurs later due to rupture of clot.

N11 SPLEEN -INCISED WOUND

- An incised wound is basically three dimensional, having sharp edge, and length of the wound will be greatest of the three dimensions.
- Produced by the object having a sharp cutting edge, such as knife, razor, scalpel and sword.
- Margins: Clean cut, well defined and usually everted. The edges are free from contusions and abrasions.
- Width: width is greater than the edge of the weapon causing it.
- Length : Length is greater than width and depth.
- Shape: is usually spindle shaped due to greater retraction of the edges in the centre.
- Hemorrhage: More as the vessels are cut cleanly.

TOXICOLGY

O1 to O10, O12,O13,O16,O17,O19,O20 – NON POISONOUS SNAKE

Trait	Poisonous	Non-poisonous
Head scales	• Small (viper) • Large (pit viper) • 3rd labial touches the eye and nasal shields.	• Large
Belly scales	Large and cover entire breadth	Small, do not cover the entire breadth.
Fangs	Hollow like hypodermic needle	Short and solid
Teeth	Two long fangs	Several small teeth
Tail	Compressed	Not much
Habits	Usually nocturnal	Not so

O11 CENTIPEDE

Commonest genus: Scolopendra

Neurotoxic venom is injected through its venom ducts. Bite through centipede is typically pointed in shape.

Bite site are characterized by local burning pain, erythema, swelling, inflammation, superficial necrosis, lymphadenopathy and lymphangitis.

Systemic manifestations: anxiety, dizziness, vomiting, headache, convulsions, cardiac arrhythmia, rhabdomyolysis.

Treatment: Supportive. Ice packing over the site of bite. Steroids, antinistamines, analgesics & Inj. T.T.

O14, 015:SCORPION

Scientific name of red scorpion: Mesobuthustamulus

It is poisonous eight legged Arthropod. It has a hollow sting in the last joint of tail at its tip. The sting is communicated by a duct with poisonous gland. The venom is injected on sting.

The scorpions are light yellow to black in color and can grow upto 5 to 8 cms in length.

Venom: is colorless, clear toxalbumen.

Types: 1) Hemolytic which mimics viper bite

2) Neurotoxic which mimics strychnine poisoning.

The venom is more potent than that of snakes but mortality is less especially in adults as little quantity is injected. Mortality can occur in children.

Symptomatology: Hemolysis, swelling, severe pain at the site with one hole by sting with reddening. In neurotoxic venom, there are convulsions, nausea, vomiting, restlessness, fever, paralysis respiratory depression, coma & death by paralysis.

Treatment: Application of Ice packs
Local anesthetic injection
Antihistamine
Calcium gluconate – controls local swelling
Steroids
Atropine- prevents pulmonary oedema
Barbiturates –to reduce excitement and convulsions

O18, O24: COMMON KRAIT. O28, O29: BANDED KRAIT

Common Krait
Scientific Name: Bungaruscaeruleus
Features:

- Hexagonal large scales-dorsal aspect.
- Subcaudals undivided
- Round pupils
- 4th infralabial scale is the largest.
- Venom :Neurotoxic.

O21, O22 O23:COMMON COBRA

Scientific name: Naja naja

The colour of snake is brown. They can be dark in colour. It can grow upto 1½ to 2 mtrs. It has a hood with a single or double spectacle mark or it may have an oval spot, surrounded by an ellipse. There may be small golden spots.

There may be two black spots or three black bands over ventral aspect of hood. At the junction of hood and body here is usually a white band.

The fangs of cobras are grooved.

In cobra bite the mark can be 1.2 cms deep. The bite site can contain fluid & haemolysed blood.

Symptomatology (Ophitoxaemia)

At the site of bite: Symptoms may start in 5 to 10 minutes in the form of reddish wheal, tenderness present with pain.

Other symptoms: Weakness in limbs, sleepy, intoxicated nausea & vomiting, paralysis of lowerlimbs, then trunk, head, ptosis develops, excessive salivation may be present, paralysis of tongue & larynx result in difficulty in swallowing and speech. Person dies due to respiratory muscle failure or respiratory centre failure.

Total Dose: 12 mg

Total Period: 30 min to 6 hours

Snake Venom: It is the saliva from modified salivary gland. Cobra venom is transparent yellow and is viscous. Becomes turbid when exposed to light venom contains mainly Peptides, enzymes & proteins of lower molecular weights. The enzymes are transaminase, Hyaluronidase, phospholipase – A, B, C & D, proteinases, lipases, ribonuclease, deoxyribonuclease etc., It also contains 5 hydroxytryptamine& acetylcholine. In cobra, venom is neurotoxic & causes Curare like effect & paralysis.

Cobra lays about 45 to 50 eggs and they hatch in about 58 days.

O25, O26, 027:RUSSELL'S VIPER

Scientific name: Vipera russelli

It has flat, triangular head with a white 'V' mark pointing forwards; its body is whitish with dark semilunar spots. Tail is narrow & short. It is known to hiss loudly & continuously. It has three rows of diamond shaped black or brown spots. Over back, the outer two rows consisting of spots ringed with edges.

The fangs are canalised

The bite mark can be 2.5 cms deep

The venom is white or yellow.

The snake venom has proteins of low molecular weight, enzymes, it is mainly haemolytic& causes intravascular haemolysis& depression of coagulation mechanism.

Fatal Dose – 15 mg

Fatal period – about 1 to 2 days

Symptomatology: (ophitoxaemia)

Same in Russell's viper &echiscarinata. Many have no symptoms because little or no venom is injected.

TREATMENT IN SNAKE BITE POISONING

Snakes are most timid reptiles and they often attack for self defence. Sometimes the sight of snakes itself can cause shock and death.

I. First Aid:

1. Assure the frightened nervous patient
2. Use tourniquet (a board bandage, cloth) over & above the site of bite to occlude superficial veins. Loosen for about 1 min. once in 10-15 min time.
3. Immobilise the limb to prevent spread of venom
4. Make crises cross superficial infusions about 1 cms x 0.5 cms deep over the site of bite to drain blood and lymph.
5. Section – suck the site of bite by using suction, suction bulb, in some tribal areas chick's anus is used. Suck for about 1 to 1½ hrs.
6. Clean the would nicely & keep the wound open.
7. Ice packing can cause vasoconstriction and decrease absorption to some extent.

II Polyvalent antisnake venom: This is prepared in Haffkine Institute, Bombay & at Kasauli by Hyperimmunising horse serum. The venom of – 4 common snakes are used. They are Cobra, common Krait, Russell's Viper & Saw sealed viper. The serum obtained is lyophilized by drying it from frozen state under high vaccum. It is available as a white power in an ampule & retains potency for 10 years. It is better to use it with in 4 to 8 hours after bite. The powder is dissolved in distilled water. Before giving sensitivity test is done by injecting 1:10 dilution of 9.05 to 0.1 ml intradermally. In sensitive people, wheat of 1 cms or more diameter with eythemia develops in 5 to 20 minutes.

Desensitisation: It can be done in sensitive persons by injecting 0.5 to 1 cc intradermally& dose doubled every ½ hr until full dose is administrered.

Dose: 20 ml IV & repeat after 1 hour or early if symptoms persist. Further dose every 6 hourly until the symptoms disappear.

III. Inject TT to prevent tetanus.

IV. Inject antihistamine, steroids – broad spectrum antibiotics.

V .If antisnake venom is not available, antivenin 40 ml. IV & repeated if necessary. It is of use in Cobra & Russell's viper bite.

VI. In viper bite inject antisnake venom at the site to prevent sloughing & necrosis.

VII. Sedative in viper to relieve pain & Nervousness.

VIII. Infusion of normal saline, blood plasma in severe poisoning.

IX. If patient survives on a later date surgical debridement of bite site to remove blebs, vesicles & superficial sloughing.

X. Pentoneal dialysis to remove poison.

XI. Please remember incantation (Chanting of Sanskrit slokas etc.,) eating sacred tree leaves are of no use.

O30, O35:RICINUS COMMUNIS

TYPE: Organic Irritant Poisons (I).

Active principle: Ricin

Symptoms: Of GIT irritation, colicky pain in abdomen, dehydration, oliguria, uraemia, collapse and death.

Fatal Dose:10-20 Seeds (6mg ricin)

Fatal Period:3 – 5 days

Treatment: Gastric lavage, emetics & demulcents. Administration of glucose & saline.2 – 5 g of sodium bicarbonate is given 8 hourly.

P.M. Changes: Mucosa of the GIT is congested, softened and inflamed with occasional erosions and submucous haemorrhages. Fragment of seeds may be found in stomach and intestines. Dilatation of heart,haemorrhages in the pleura, edema and congestion of the liver, kidneys, spleen and lungs.

Medico-legal Importance: Accidental poisoning usually occurs in children from eating the seeds by mistake. Homicidal poisoning may occur when administrated in food in the form of powder seeds.

O31 CALOTROPIS GIGANTEA

TYPE: Organic Irritant Poisons (I).

Active principles:calotropin, calatoxin, uscharin, calactin

Symptoms: When taken internally, gives rise to an acrid, bitter taste and a burning pain in the throat and stomach. These are followed by salivation, stomatitis, vomiting, diarrhoea, dilated pupils, convulsion; collapse and death.

Fatal Dose: Uncertain.

Fatal Period: Half-an-hour to eight hours.

Treatment: Stomach wash should be given. Saline cathartics may be helpful. Demulcents are administered. Morphine like drugs is used to control pain.

P.M. Changes: Signs of irritation in the stomach & intestines may be seen.

Medicolegal Importance: Juice may be taken orally or applied on an abortion stick to procure abortion. It may be mixed with milk for infanticide. Used to produce artificial bruise

O32 NERIUM ODORUM

TYPE: Cardiac Poisons.

Active principles: Oleandrin, Nerin, Folinerin, Rosagenin

Symptoms: Oleander poisoning includes gastrointestinal irritation and digitalis like action on the heart. There is also difficulty in swallowing & articulation, abdominal pain, vomiting, profuse frothy salivation and diarrhea. The pulse is slow & become rapid& weak.

Fatal Dose: Uncertain.

Fatal Period:26-36 hours.

Treatment: Evacuation by washing the stomach treatment of symptoms. If cardiac arrhythmia develops, anti- arrhythmic drug should be given.

P.M. Changes: Post-mortem appearances are not characteristic. There may be congested patches in the stomach and upper portion of the small intestine.

Medico-legal Importance: As abortifacient, cattle poison, Quack remedy.

O33 DATURA FASTUOSA

Type: Neurotoxic poison (Deliriant)

Active principles: Hyoscine**,** Hyoscyamine, Atropine

Mode of action: Peripherally anticholinergic, centrally stimulation later depression.

Signs & Symptoms: 8Ds:

- Dryness of mouth , with nausea and vomiting
- Dysphagia
- Dilated pupils with blurred vision,
- Dry hot skin
- Drunken gait
- Delirium with confusion & hallucinations
- Drowsiness leading to coma
- Death due to respiratory failure

Treatment:

- Decontamination
- Physostigmine – 1 to 2mg IM or IV repeated after half an hour if necessary
- Diazepam
- Supportive measures

Medicolegal importance:

- Accidental
- Suicidal
- Stupefying agent(road poison)
- Homicidal agent

O34 ARGEMONE MEXICANA

Sessile, spiny thistle shaped leaves

Seeds resemble mustard seeds, used as adulterant with mustard oil.

Active principles: Plant -2 alkaloids, Berberine, Protopine

Oil – 2 alkaloids, Sanguinarine, Dihydrosanguinarine

Signs & Symptoms: Causes epidemic dropsy

- loss of appetite, marked edema of the limbs
- Myocardial damage and dilatation of the heart
- Bluish mottling of the skin
- Dimness of vision due to glaucoma
- Death may be due to severe damage to the heart

Treatment: Stop consumption of the adulterated oil. Thiamine

O36 STOMACH IN FORMALIN POISONING (PMNO 78/10)

O37 TONGUE IN FORMALINPOISONING

- Formalin is an aqueous solution of formaldehyde containing 37-40% formaldehyde and 10-15% methanol. This is generally referred to as 100% formalin.
- The commercial preparation usually contains 3.7% of formalin. Accidental exposure to formalin is common as an occupational hazard.
- Formaldehyde is a corrosive that can produce late sequelae, similar to the more common ingestion of acids and alkalies.
- Renal failure is a frequent complication in severe poisoning.
- Skin and mucous membrane may appear whitened.
- At autopsy, the smell of formalin may be noticed on opening the body and stomach. The mucous membrane of the stomach may be red, inflamed and eroded with extravasation of blood, or may be leathery, fixed and hard to touch.
- The duodenum may present the same appearance as that of the stomach.
- Histological details may be well preserved. The kidneys may reveal microscopic evidence of tubular necrosis.
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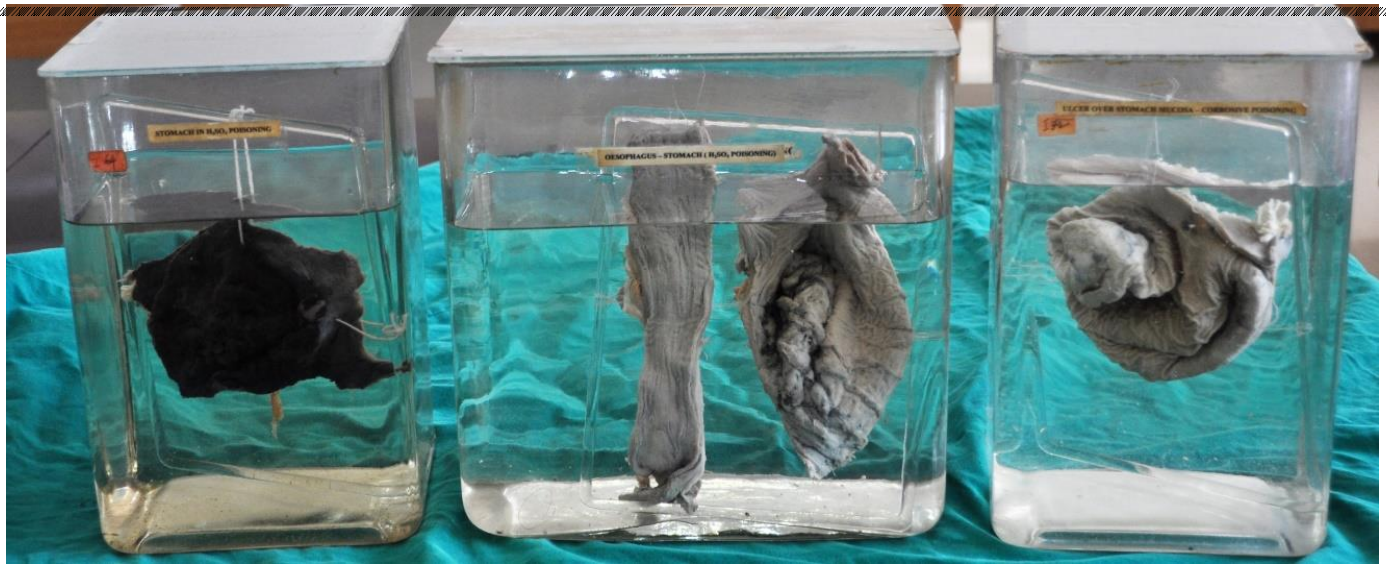


O38 STOMACH IN SULPHURIC ACID POISONING

O39 OESOPHAGUS-STOMACH IN SULPHURIC ACID POISONING

O40 ULCER OVER STOMACH MUCOSA - CORROSIVE POISONING

Sulphuric acid Poisoning- autopsy features:

- ✓ Corroded areas of skin and mucous membranes appear brownish or blackish.
- ✓ Teeth appear chalky white.
- ✓ Stomach mucosa shows the consistency of *wet blotting paper*.
- ✓ There may be inflammation, necrosis, or perforation of the GI tract.

FETAL DEVELOPMENT AND ANOMALIES

MALE /FEMALE GENITAL TRACT

P1 FOETUS LENGTH: 8.5CMS (2.5-3 MONTHS)

STAGES OF DEVELOPMENT

End of first month

Length one cm; weight 2and a half grams. The eyes are seen as two dark spots and mouth as a cleft. Nucleated red cells begun to form in the placenta.

End of second month

Length four 4cm; weight 10 grams. The hand and feet are webbed. The placenta begins to form. The anus is seen as a dark spot. First ossification centre in a fetus appears in the clavicle (4 to 5 weeks), followed by Maxilla (6 weeks)

End of third month

Length nine cm; weight 30 gms. The eyes are closed and the papillary membrane appears. Nail appears and the neck is formed.

 **P2**

FOETUS - 4 MONTHSP3 FOETUS - 18 WEEK

<u>MALE FOETUS – 4 MONTHS OLD</u>:

Length from crown to heel is 16 cms.

Weight about 120 – 130 gms

The sex organs are developing and sex can be determined.

Lanugo hair present over the body

<u>At Autopsy</u>: One can find meconium in duodenum and convolutions in Brain.

<u>HAASE RULE</u>: This helps to calculate roughly and quickly age of foetus.

<u>Up to 5 months</u>: Age of Foetus in months:-

Square Root of crown to heel length of foetus.

Eg: If foetus is 16 cms long, the age is = 4 months

After 5 months(Morison's rule):-

Age of the foetus in months = $\frac{\text{Crown to heel length}}{5}$

Eg: If length of foetus is 40 cms

Age of foetus in months = 8 months

P4 FOETUS - 5 MONTHS

End of fifth month

Length is 30 cm; weight 700 g. nails are distinct and soft. Light hairs appear on the head. Skin is covered with vernix caseosa. Meconium is seen at the beginning of small intestine

P5 FOETUS- 6 MONTHS

End of sixth month

Length 30cm; weight 700 gm. Eyebrows and eyelashes appear. Skin is red and wrinkled and subcutaneous fat begins to be deposited. Vernix Caseosa is present. Meconium is in transverse colon. The testes are seen close to the kidneys.

P6 FOETUS WITH PLACENTA-7 MONTHS

End of the seventh month

Length 35cm; crown rump length 23cm; foot length 8cm; weight 900 to 1200g.Nails are thick. Eyelid are open and pupillary membrane disappears. Skin is dusky red, thick and fibrous. Meconium is found in the entire large intestine. Testes are found at the external inguinal ring. Gall bladder contains bile and caecum is seen in the right iliac fossa. Ossification centre is present in the talus

P7 FEMALE FOETUS-8 MONTHS

The Crown to heel length is 40 cms

Weight: 1.5 to 2 kg

Nails grow upto tip of the fingers.

Scalp hair are dark, thicker, about 1 to 2 cms length

Skin is tight & smooth.

Placenta weight :500 to 550 gms.

Centre for calcaneum : Appears at the end of 5 months of IUL

Centre for talus : Appears at the of 6 months of IUL

Sternum shows centre for Manubrium

Ist piece of body = 5^{th} month

IInd&IIIrd piece of body = 7^{th} month

Viability: It is defined as the physical capability of foetus to lead a separate existence after birth without much assistance by virtue of certain amount of development as a whole.

Age for viability is 7 month (210 days)

But in some exceptional cases viable even after 180 days.

P8 FOETUS - 9 MONTHS

End of ninth month

Length 45cm; weight 2.2 to 3 kg. Scalp hair is dark and 4 cm, long. Meconium is seen at the end of large intestine. Scrotum is wrinkled and contains both testes. Placenta weighs 500 gms. Ossification centres usually present in the lower end of femur.

End of tenth month

Length 48-52 cm, crown rump length 28 to 32 cm; weight 2.5 to 5 kg; average about 3.4kgs. The length is much less variable than the weight. The circumference of the head is 33 to 38 cm. Sixfrontanels are usually present in the neonatal skull. Anterior frontanella measures 4x 2.5cm. The surface of the brain shows convolutions and the grey matter beginning to form. The scalp hair is dark, 3 to 5cm long. The face is not wrinkled. The skin is pale and covered with vernix caseosa. The rectum contains dark brown or greenish or black meconium. The umbilical cord is 50 to 55cm long, and is one cm thick

P9 RESPIRED LUNGSP10 UNRESPIRED LUNGS

Sl no	Feature	Before respiration	After respiration
[A]	**GROSS**		
1	COLOR	Uniformly reddish brown, bluish or deep violet depending on the degree of anoxia	Mosaic, mottled or marbled
2	Volume	Small; do not cover heart. Collapsed on the hilum	Larger; cover heart
3	Surface	Smooth	Uneven
4	Margins	Sharp	Rounded
5	Lowermost	At the level of 4-5 ribs	At the level of 6-7 ribs
6	Consistency	Liver like, dense, firm, non-crepitant	Crepitant, elastic, soft, spongy
7	Blood within lungs	Less	More. Twice that of present in unrespired lungs 70g
8	Weight of both lungs	35g	70g
9	Weight of both lungs [In relation to body weight]	1/70gof body wt	1/35of body wt
10	Specific Gravity	1.04-1.05 Heavier than water	0.94 Lighter than water
11	Hydrostatic test	-ve. Sinks in water	+ve. Floats in water
12	Thoracic cavity	Not occupied fully by lungs	Almost occupied fully by lungs
13	Pluera	Loose, wrinkled	Taut, stretched
[B]	Cut Section		
14	Oozing of blood	Little frothless blood exudes on pressure	Abundant frothy blood oozes on cut section
15	Bronchi and bronchioles	Empty	Contain blood stained froth
16	Alveoli	Not inflated	Inflated
[C]	MLI		
17	MLI	Indicated stillborn or deadborn child	Indicates live born child

P11,P12,P13; OSSIFICATION CENTRE FOR FOOT , STERNUM, KNEE

Dissection to demonstrate the ossification centres

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

Centres of ossification may be demonstrated as follows

For ossification centres in the various divisions of the sternum, the bone is placed on a wooden board and sectioned in its long axis with a cartilage knife which exposes centres of ossification in the various divisions of the sternum. For the ossificationcentre in the lower end of the femur and the upper end of the tibia, the leg is flexed against the thigh and a horizontal incision made across and into the knee joint.

A number of cross sections are made through the epiphysis starting from the articular surface and continuing until the largest cross section of the ossification centre is reached. In the lower end of the femur, this is seen as brownish red nucleus which is surrounded by a bluish white cartilage. This centre of ossification in the upper end of the tibia is found in some cases, but in others it appears after birth. To expose the ossific centres in the bones of the foot, the heel of the foot is placed on a sponge and firmly held by one hand, and with the other hand an incision is made through the interspace between the third and fourth toes and carried downwards through the sole of the foot and heel. Centres of the calcaneum and talus which appear at fifth and seventh months of intrauterine life are exposed.

P14 DECAPITATED HEAD

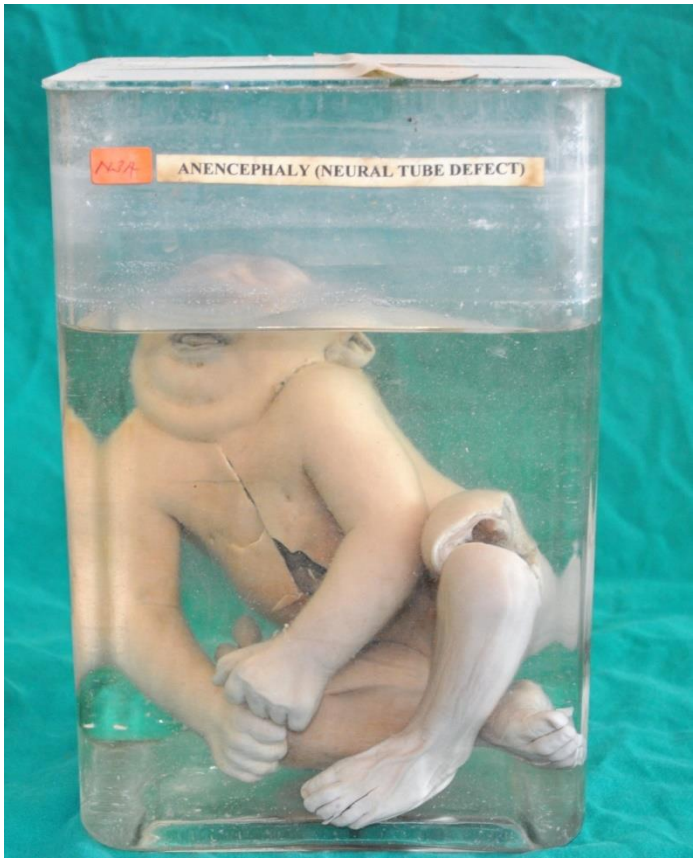
- In forensic practice, decapitated bodies are usually associated with accidental explosions or vehicle crashes
- Decapitation has been reported to occur also in suicidal and homicidal deaths.
- In suicides, decapitation can be found occasionally in hanging deaths and trainpedestrian fatalities.
- Postmortem decapitation must be considered highly suggestive of a homicidal mode of deathcharacterized by absence of vital signs around the wounds of decapitation.
- Decapitation of infant could be associated with criminal abortion with instrumentation.

P15 : UTERUS WITH MUMMIFIED FOETUS

The foetus can undergo mummification changes if following conditions are met with.

1. Foetus dies of deficient blood supply
2. No air enters into womb.
3. There is scanty liquor amni

The foetus will have same features of mummification that are seen in adult. It is a dead born foetus if it is delivered with mummification changes.



P16 ANENCEPHALY (NEURAL TUBE DEFECT)

Anencephaly is a serious birth defect in which a baby is born without parts of the brain and skull. It is a type of neural tube defect (NTD). As the neural tube forms and closes, it helps form the baby's brain and skull (upper part of the neural tube), spinal cord, and back bones (lower part of the neural tube). Anencephaly happens if the upper part of the neural tube does not close all the way. This often results in a baby being born without the front part of the brain (forebrain) and the thinking and coordinating part of the brain (cerebrum). The remaining parts of the brain are often not covered by bone or skin.

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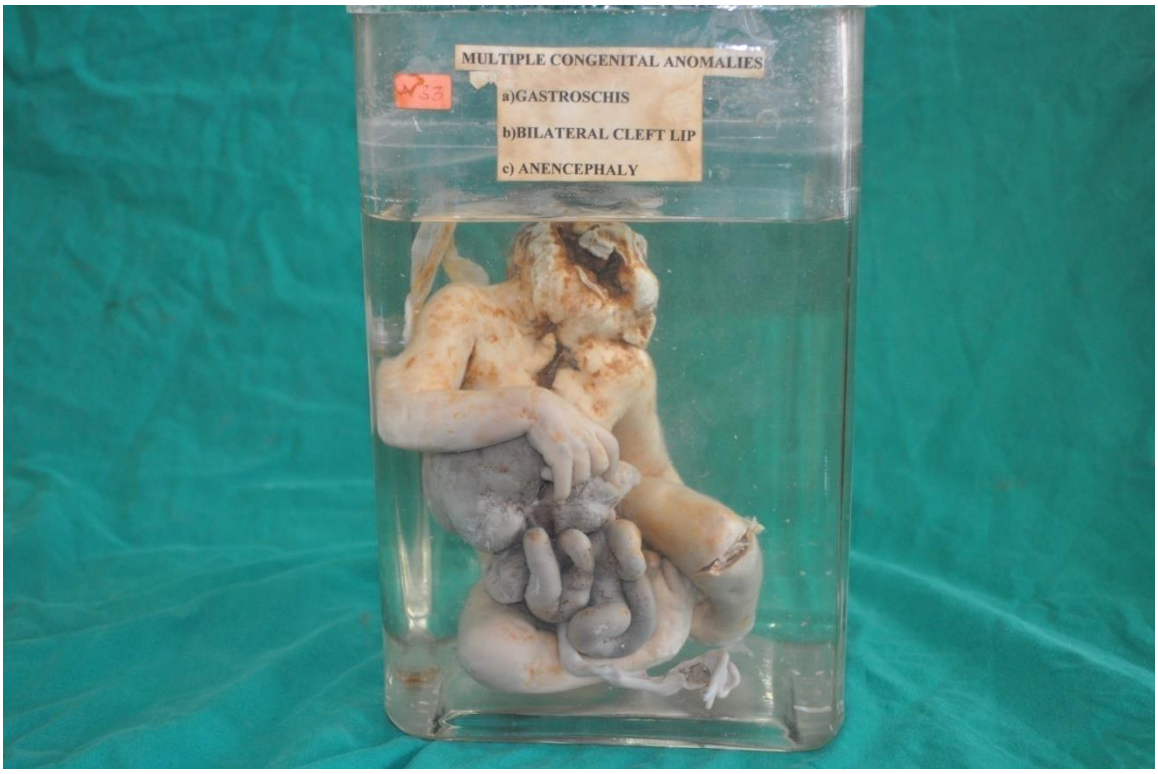


P17 MULTIPLE CONGENITAL ANOMALIES (A)GASTROSCHISIS (B)BILATERAL CLEFT LIP (C)ANENCEPHALY

A gastroschisis is a birth defect in which an opening in your baby's abdominal wall allows the stomach or intestines to protrude outside of the body and float in the amniotic fluid. The amount of abdominal contents outside the baby varies from very small - just a few loops of bowel - to quite large, involving most of the intestines and stomach. It is not known why this happens, but the opening occurs when the abdominal wall muscles develop incorrectly. The majority of babies with this problem are born to mothers in their late teens or early 20s.

P18 DOLICOCEPHALUS

Dolichocephaly is a condition in which one has an abnormallylong head. A "perfectly round" head measures the same distancebetween the ears as it does from the nose to the back of thehead. In dolichocephaly, the front to back measurement is longerthan the side-to-side measurement.

Normally, as an infant's brain grows, sutures between the bones of the skull expand and lead to thedevelopment of a normal head shape. If one or more of thesesutures close early, it will cause the skull to expand in the directionof the sutures that remain open, which can result in an abnormal head shape.

In dolichocephaly, the sagittal sutures close together too early. Thisclosure prevents the skull from expanding in width, and the skullbegins to expand towards open sutures

The result is that the skull takes on a long, narrow, and boat-shaped appearance. A ridge may be noticeable by the closed sagittal sutures.

In severe cases, dolichocephaly can result in increased pressure on the growing brain, leading tobrain impairment and mental retardation.

P19 RACHISCHISIS NEURAL TUBE DEFECT

Rachischisis (sometimes known as **complete spina bifida**) refers to a severe form of spina bifida where there is a cleft through the entire spine.

Pathology

There is often a severe or complete defect of the neural tube involving the entire spine from the cervical region through to the sacrum.

Associations

A rachischisis often occurs in association cranial neural tube defects such as:

- anencephaly
- acrania
- iniencephaly

Q1 NORMAL UTERUS WITH TUBES AND OVARIES

Features	Nulliparous uterus	Parous uterus
Size	Smaller (7 cm x 5cm x2 cm)	Larger (10 cm x 6 cm x 2.5 cm)
Weight	40 gms	80-100 gms
Ratio between body length and cervix	1:1	2:1
Upper surface of fundus	Less convex and is in the same line of broad ligament	More convex and is at a higher level than the line of broad ligament.
Uterine cavity	Triangular with less space	Large and spacious cavity
External os	Small, round	Transverse slit like opening
Internal os	Circular, well defined	Ill defined, margin wrinkled.

Q2 PLACENTA WITH UMBILICAL CORD

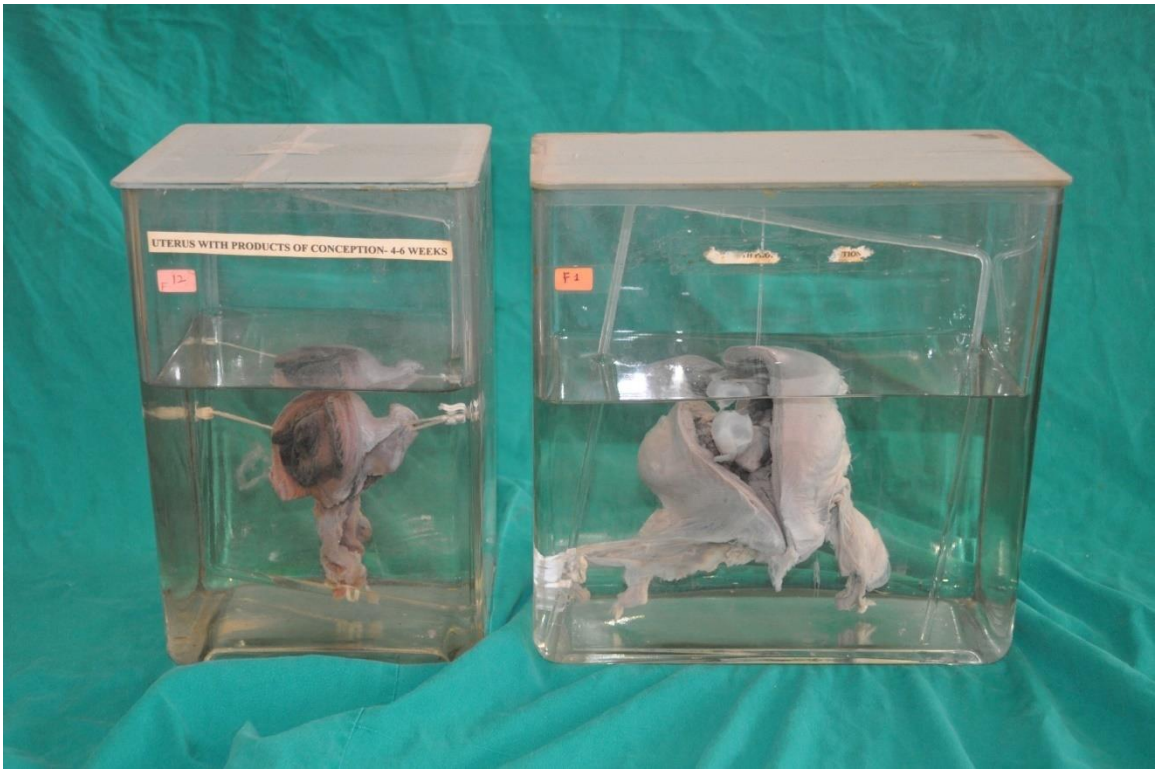
Examination of the umbilical cord and the placenta at autopsy

If the cord and the placenta are present, they should be examined. Placenta should be weighed to evaluate maturity and any abnormality should also be observed. Placenta is enlarged in syphilis and hemolytic diseases, placenta is smaller is case of placental insufficiency.

The changes in the cord are an important indicator of separate existence. Any knots, ruptured varices, number of arteries are noted. The severed end of the cord may help in concluding whether the cord has been actually cut or broken. The cut ends with a sharp instrument like scissors or knife appear clean cut but occasionally may appear ragged if the instrument is relatively blunt.

Any evidence for malformations or birth injuries should be searched for meticulously, which may reveal obvious incompatibility with life.

Changes in the umbilical cord Changes in umbilical cord begin to appear in the cut end to its base at the umbilicus soon after birth. Even when the putrefaction has rendered the evaluation of the breathing extremely difficult, vital signs in the cord may be helpful in indicating live birth if there has been a sufficient survival period. The portion of the cord attached to the child shrinks and dries within 24- 48 hours and an inflammatory ring or a reddening ring appears at its base and the adjacent skin from 36 to 48 hours. By the second or the third day, it shrivels up, mummifies and falls off on the fifth or sixth day, leaving a raw area, which heals and cicatrizes within 10-12 days.



Q3UTERUS WITH PRODUCTS OF CONCEPTION 4-6 WEEKSQ4 UTERUS WITH PRODUCTS OF CONCEPTION Q5 UTERUS WITH PRODUCTS OF CONCEPTION

The fundus of the Uterus shows fully formed Embryo.

It has chorionic villi buried in the endometrium,, parts of the foetus have not yet formed.

Age about 1 month.

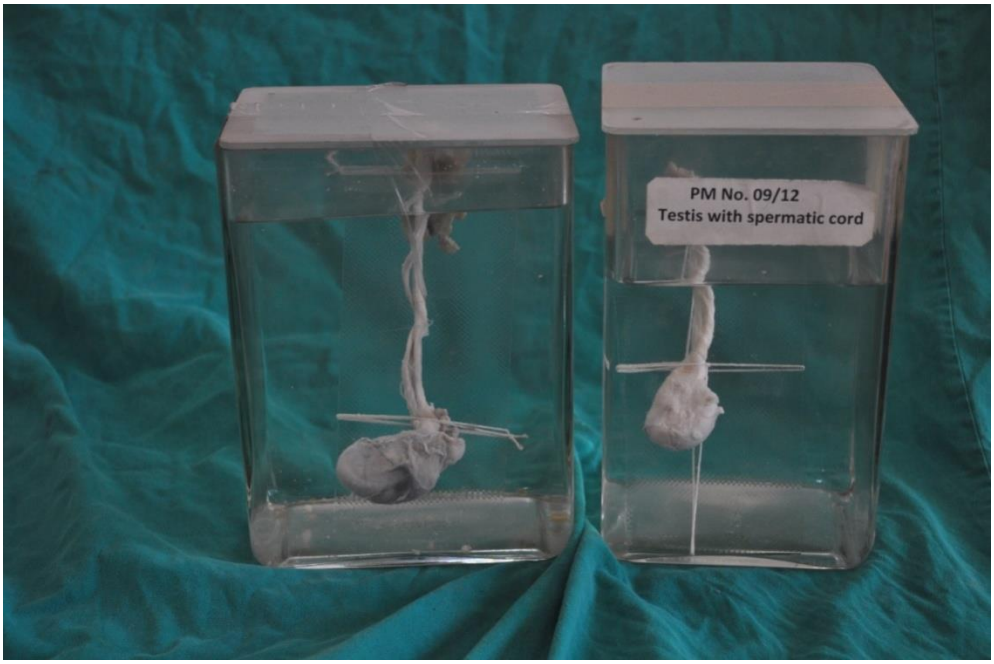
Q6 FIBROID UTERUS, Q7 UTERUS WITH INTRAMURAL FIBROID

Uterine fibroids are the most common pelvic tumor, occurring in about 70% of women by age 45. Most fibroids in the uterus are

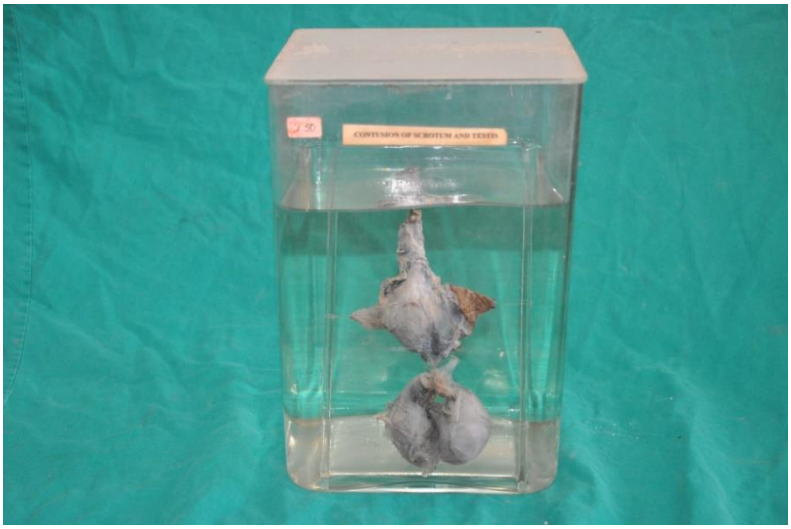
- Subserosal (most common)
- Intramural
- Submucosal (least common)

Occasionally, fibroids occur in the broad ligaments (intraligamentous), fallopian tubes, or cervix.

Some fibroids are pedunculated. Most fibroids are multiple, and each develops from a single smooth muscle cell, making them monoclonal in origin. Because they respond to estrogen, fibroids tend to enlarge during the reproductive years and decrease in size after menopause.
Fibroids may outgrow their blood supply and degenerate. Degeneration is described as hyaline, myxomatous, calcific, cystic, fatty, red (usually only during pregnancy), or necrotic. Although patients are often concerned about cancer in fibroids, sarcomatous change occurs in < 1% of patients.



Q8TESTIS WITH SPERMATIC CORD Q9TESTIS WITH SPERMATIC CORD (PMNO.09/12)

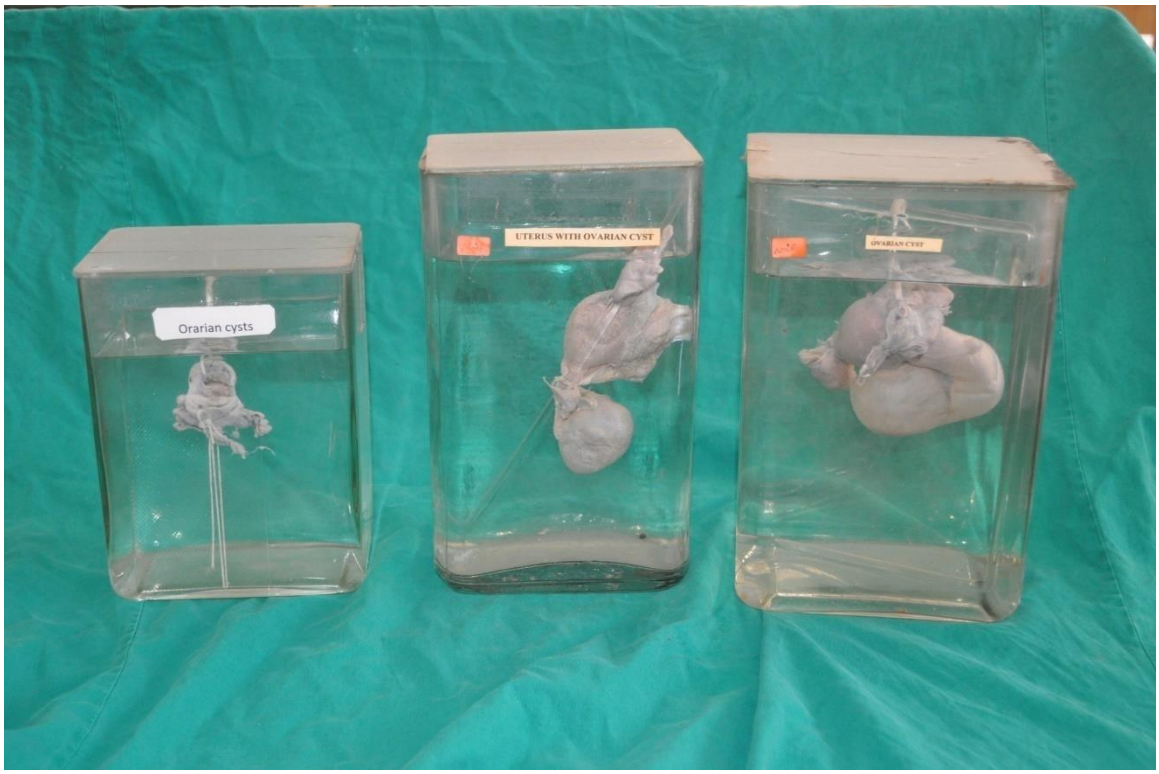


Q10 CONTUSION OF SCROTUM AND TESTIS

- Associated with blunt force impact to genital region.
- In cases of testicular injury, blunt scrotal trauma is responsible for 75% of the reported cases; of these, most result from sports injuries, vehicle accidents and assault
- Testicular Trauma can result in death due to Shock and Systemic Inflammatory Response Syndrome

Q11 UTERUS WITH COPPER 'T' ,Q12 COPPER T – UTERUS

- An Intrauterine Device (IUD) is a small object that is inserted through the cervix and placed in the uterus to prevent pregnancy.
- A small string hangs down from the IUD into the upper part of the vagina.
- The IUD is not noticeable during intercourse.
- IUD's can show pharmacological efficacy for about 1-10 years.
- They work by changing the lining of the uterus and fallopian tubes affecting the movements of eggs and sperm and so that fertilization does not occur.



Q13, Q14, Q15: OVARIAN CYST

Ovarian cysts are fluid-filled sacs or pockets in an ovary or on its surface.

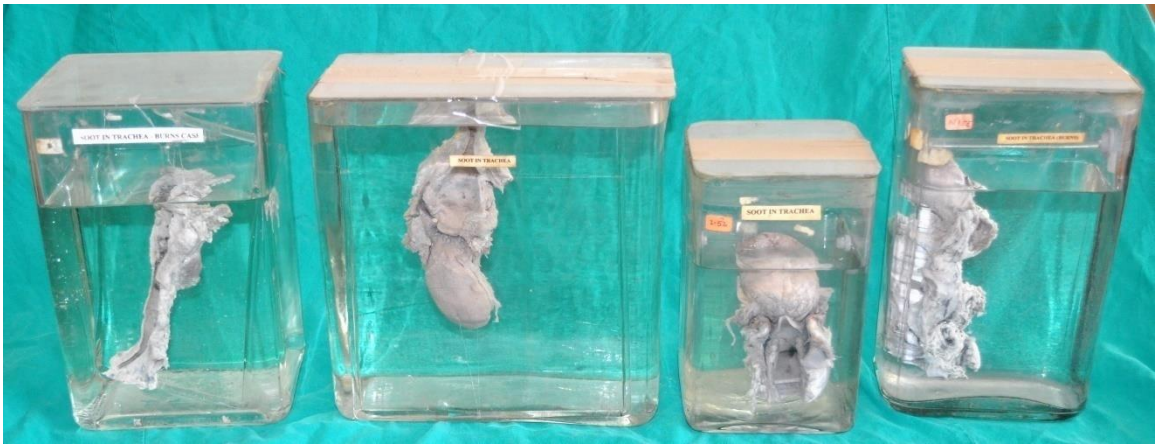
Functional cysts: If a normal monthly follicle keeps growing, it's known as a functional cyst. There are two types of functional cysts:

- **Follicular cyst.** Around the midpoint of your menstrual cycle, an egg bursts out of its follicle and travels down the fallopian tube. A follicular cyst begins when the follicle doesn't rupture or release its egg, but continues to grow.
- **Corpus luteum cyst.** When a follicle releases its egg, it begins producing estrogen and progesterone for conception. This follicle is now called the corpus luteum. Sometimes, fluid accumulates inside the follicle, causing the corpus luteum to grow into a cyst.

Other cysts

- **Dermoid cysts.** Also called teratomas, these can contain tissue, such as hair, skin or teeth, because they form from embryonic cells. They're rarely cancerous.
- **Cystadenomas.** These develop on the surface of an ovary and might be filled with a watery or a mucous material.
- **Endometriomas.** These develop as a result of a condition in which uterine endometrial cells grow outside your uterus (endometriosis). Some of the tissue can attach to your ovary and form a growth.

NECK STRUCTURES



R1,R2,R3,R4 :SOOT IN TRACHEA

Internal examination findings in death due to flame burns

- The nasal and mouth cavities may show presence of Carbon soot.
- Presence of carbon particles in the terminal bronchioles & alveoli on histopathological examination is absolute proof of life during fire.
- Presence of soot in alveoli & elevated CO saturation together absolute proof that victim was alive.

R6 NECK STRUCTURES WITH TRACHEOSTOMY WOUND

- Tracheostomy is a creation of permanent or semi- permanent opening in the trachea. The main indication is in upper airway obstruction i.e. Trauma Foreign body, Infections, Malignant lesions, etc.
- Tracheostomy wound at autopsy- may be confused as stab wound (iatrogenic artefact)

R7 CHOKING

- It is a form of asphyxia caused by an obstruction within the air passages.
- Impaction of solid bodies – a large bolus of food, piece of meat, coin, etc
- Asphyxial features may be marked.
- Microscopically lung shows intraalveolar oedema with desquamated epithelial cells

R8 TRAUMATIC ASPHYXIA – SUBMUCOSAL PETECHIAE IN TRACHEA AND EPIGLOTTIS

- Traumatic asphyxia results from respiratory arrest due to mechanical fixation of the chest, so that the normal movements of the chest wall are prevented.
- The lungs are usually dark, heavy and have subpleural petechial hemorrhages.
- The right heart and all the veins above the aorta are markedly distended.
- Internal organs are congested.

R9 LIGATURE MARK

- ✓ It is a type of pressure abrasion where there is crushing of superficial layers of the epidermis.
- ✓ Movement of object is perpendicular to the skin.
- ✓ Eg: Ligature mark – Hanging &Strangulation .
- ✓ Ligature mark is oblique, above thyroid cartilage in hanging .
- ✓ In cases of strangulation it is horizontal and at or below the level of thyroid cartilage.

SKELETAL / MUSCULAR INJURIES

S1 CRUSH INJURY HAND

A crush injury is defined as compression of the extremities causing muscular and neurological disturbance] and in the upper limb is sustained when the fingers, hand or wrist are caught between two surfaces (sharp, blunt, smooth or irregular) forcibly producing damage to the skin and its enclosed contents of soft tissues and bone.

The degree of damage is proportional to the amount of force applied per square inch and the duration the compression is in place.

Crush syndrome is a condition characterized by systemic manifestations of crush injuries, consisting of Rhabdomyolysis, electrolyte and acid-base abnormalities,hypovolemia and acute renal failure.

S2 BURR HOLE

Trepanning, also known as **trepanation**, **trephination**, **trephining** or making a **burr hole** is a surgical intervention in which a hole is drilled or scraped into the human skull, exposing the *dura mater* to treat health problems related to intracranial diseases or release pressured blood buildup from an injury.

It may also refer to any "burr" hole created through other body surfaces, including nail beds.

It is often used to relieve pressure beneath a surface. A trephine is an instrument used for cutting out a round piece of skull bone.

S3 DEPRESSED FRACTURE

- Caused due to forceful localized impact causing multiple linear fractures radiating from the site of impact with depression at the site of impact where the bone breaks into pieces.

- Depressed comminuted fractures called ‘fracture ala signature’ since the weapon which has caused the fracture may leave its impression.

S4 OBLITERATED SUTURE WITH PACCHIONIAN DEPRESSION

- Pacchionian depression: Any of various small pits on the inner surface of the skullalong the sagittal sinus that contain arachnoid granulations.
- Skull suture closure helps in estimation of the age.
- Sagittal suture fuses early and start from back to proceed to the front, unites by 30-50 years.
- Coronal suture by 40-60 years.
- Lambdoid suture by 45 years.
- Squamous part of the temporal bone unites with parietal bone around 60 – 70 years.

S6 STERNUM - STAB WOUND,S11 STAB INJURY THROUGH RIGHT 3rd INTERCOSTAL SPACE

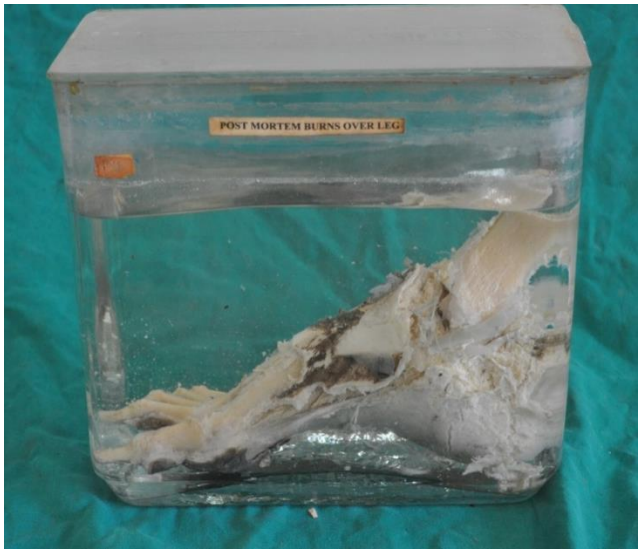
- The shape of stab wound suggests the kind of weapon used. Spindle shaped entry wound is caused by a double edged penetrating weapon. The measurement and shape will help to some extent to trace the weapon.
- The stab wound could be either be penetrating or perforating type.
- The weapon entering the chest cavity can lead to injury to heart or lungs
- The injury could result in pneumothorax or hypovolemic shock.

S7,S9: FRACTURE OF STERNUM

- Results due to blunt force impact sustained to chest as in road traffic accidents.
- External cardiac massage can lead to sternal fracture – therapeutic artefact. It may cause bruising of the anterior chest wall, haemorrhage into subcutaneous tissues and pectoral muscles. Symmetrical, parasternal fractures of several ribs and sometimes of sternum can occur associated with contusion or tears of the lungs.
- Can result in cases of traumatic asphyxia .

S8 STERNAL END OF CLAVICLE: PENETRATING INJURY BY PELLET

- Pellets would travel as a single mass upto to a distance of 45 cm after which they start to disperse.
- In intermediate range (2 – 4 metres),pellets begin to spread & individual pellet holes may be detected. Main entrance becomes smaller and individual pellet wound increases in number.
- All shots penetrate separately, in an area of 10 -15cm in distant range (> 4 meters).



S10 POST MORTEM BURNS OVER LEG

Trait	Ante-mortem Burns	Post-mortem Burns
Line of redness	Present	Absent
Blister	Contains serous fluid with proteins & chlorides. Base is red & inflamed	Air & thin clear fluid. Base is dry, hard & yellow
Vital reaction	Cellular exudation & reactive changes in the tissue	Absent
Healing	Present	Nil

S12 SPLIT LACERATION OF SCALP: There is a crescentic split laceration over middle of scalp measuring 2.5x0.5cmsx bone depth, the shape is suggestive of an object with a limited striking surface

SPLIT LACERATION : is also known as incised looking wound because it looks like incised wound. But if seen through hand lens one can appreciate irregular edge and crushing of tissues and hair bulbs.

The two criteria's for split lacerations are:

1) Skin coming in contact with two hard objects.
2) Absence or less amount of subcutaneous tissue and fat or where in skin is in close contact with a bone.

The other sites for split lacerations are forehead,eyebrow,maxillary area,chin,shin of leg,knee cap.

Death is due to respiratory failure secondary to pressure over vital centres.

S13RETROSTERNAL CONTUSION

- ✓ An effusion of blood in to the tissues, due to rupture of blood vessels caused byblunt trauma.
- ✓ Caused by any blunt force such as fist, stone, stick, bar, whip, boot etc
- ✓ Bruise is usually situated in the subcutaneous tissues often in the fat layer.
- ✓ Could be associated with fracture of sternum.

T6 ENTRY WOUND OF RIFLED FIRE- ARM CLOSE RANGE – POWDER TATTOOING. (PM NO. 77/11)

- Firearm is fired within the range of flame and powder blast.
- The wound appears as a circular hole surrounded by scorching, singeing and smudging.
- Abrasion collar, grease collar and tattooing seen.

TOXICOLOGY SPECIMENS NOTES

1. POPPY SEEDS:

These are white, harmless seeds of papaver somniferum.
They have nutritive value and used in food & sweet preparation. It is also used as demulcent.

Demulcents: These are the substances which form a protective layer over the gastric mucosa & also delay absorption.

Other eg.: Egg white, milk, starch etc.

People have a false belief that sweet prepared with poppy seeds induces sleep. But it is not true & does not contain any sedative or hypnotic.

2. MUSTARD SEEDS:

These are used widely domestically for cooking.

It can be used as house hold emetic in poisoning case.

Emetics: Substances which can inducevomiting.

Dose: A table spoon (15 g) mustard powder in 200 ml of water.

Mustard oil is used in cooking.

The seeds of Argemone Mexicana are used as adulterant with mustard seeds.

3. STRYCHNOS NUXVOMICA SEEDS:

The fruit of strychnos nux vomica contains 3 to 5 seeds.
The seeds present in ripe fruit are poisonous.
It is a spinal poison.

The seeds are flat, circular discs or convex on one side and concave on the other side usually 2.5cm in diameter, 6 to 8mm in thickness.

Ash grey or light brown with shining surface with radiating silky fibres. They are hard and cannot be digested.

They contain two alkaloids:

1. Strychnine - 1.5%
2. Brucine - 1.55%

Also contain a glucoside called loganin.

Strychnine is colourless, odourless, rhombic prisms& have a bitter taste. Strychnine is 10 to 20 times more potent than brucin.

Medicolegal importance:

1. Rodenticide
2. For killing stray dogs
3. Respiratory stimulant
4. Arrow poison
5. Aphrodisiac
6. Cattle poison

The seeds are not poisonous if swallowed. Other seeds which are not poisonous if swallowed are castor seeds, croton seeds, abrus precatorious seeds .

Site of action: It depresses anterior horn cells inhibiting postsynaptic potentials in spinal cord & prevents effects of glycine (probably it is inhibitory transmitter)
It stimulates cerebral cortex.

Fataldose: 50 to 100 mg (one crushed seed)

Fatal period: 1 to 2 hours

The signs& symptoms mimic that of tetanus.

Treatment:

1) First isolate patient in a quiet place
2) Short acting barbiturates - Pentobarbital Sodium, sodium amytal are antidote .
   Dose = 0.3 to 0.6 gms IV

3) Inj.Diazepam is a better drug.
4) Stomach wash with dilute $KMnO_4$.

Difference between Strychnine and Tetanus :

Trait	Strychnine	Tetanus
1) Onset	Sudden	Gradual
2) History	Consumptionofpoison	H/O injury
3) Convulsions	Allmusclescontract at thesame time	Notsame time
4)Lowerjaw	Notusuallyaffected	usuallystarts late
5) Muscular coordination	Muscles are relaxed in betweenattacks.	Musclesremainrigid.
6) Fatal Period	1-2 hrs	> 20 to 24 hrs
7) Chemical analysis	Detected	Microscopy and culture ofClostridiumtetani can be done.

4. CAPSICUM ANNUM:(CHILLIES)

It is widely domestically used. But it is an organic irritant poison. It has pungent taste and odour. The seeds resemble the seeds of datura.

Active principles: Capsicin & Capsaicin.

Signs &symptoms:

It causes skin irritation, burning sensation in mouth & upper GIT.

It causes pain, congestion & inflammation.

There may be vomiting & loose motion.

If falls in eyes, it produces congestion, pain swelling &watering.

Criminal uses:

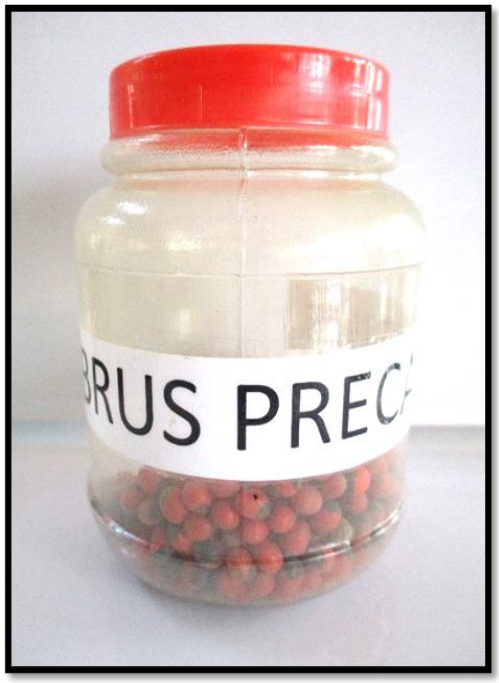
- To throw into eyes to facilitate robbery
- For torturing & falsely charging of rape, assault it is rubbed over breast, put into vagina, rectum etc.
- Hydrabad Goli - Small spherical balls prepared out of chilli powder are used for torturing criminals, introduced into rectum, vagina, urethra etc., to get confession.
- Extract of miniature 'Chilli' grown in Meghalaya is used in USA in a pistol like apparatus for protection of women- Pepper spray
- Used for treating arthritis

This poison does not cause death .

Treatment:

1. Symptomatic
2. Antacid
3. Demulcents

5. ABRUS PRECATORIUS:



It is also known as jequirity, Indian liquorice, rosary bead, gunja or rati.

Seeds: are egg-shaped, bright scarlet colour with a large black spot at one end.

Active principles: Abrin (toxalbumin), Abrine (aminoacid), Haemoglutinin, Abralin (glucoside).

Signs and Symptoms:
Severe irritation of upper GI tract, abdominal pain, nausea, vomiting, bloody diarrhea, weakness, cold perspiration, trembling of hands, weak rapid pulse, miosis and rectal bleeding.

Fatal dose: 90 to 120mg (1 to 2 seeds)

Fatal period: 3 to 5 days

Treatment:

1. Gastric lavage.
2. Activated charcoal.
3. Purgative.
4. Injection of antiabrin.
5. The needle should be dissected out.
6. Sodium bicarbonate 10 g orally per day.

Medicolegal importance:

1. The seeds are used for killing cattle and rarely for homicide.

Suis- The seeds are decorticated, and alone or mixed with datura, opium and onion, are made in to paste with spirit and water, and small sharp pointed spikesor needles or "suis" are prepared, which are then dried in the sun. The needles are 15mm long. Two needles are inserted by their base in to holes in a wooden handle. A blow is struck to the animal with great force which drives the needle in to the flesh.

2. Homicide- The person is slapped with needle between two fingers.
3. Malingering, to produce conjunctivitis.
4. Taken internally, disturb the uterine function and prevent conception.
5. Abortifacient and as arrow poison.

6. GANJA:

It is prepared from the flower tops of female plant of cannabis sativa. It has a rusty-green colour and a characteristic odour. It is mixed and smoked with tobacco in a pipe or hukka.

Active principle: Tetra Hydro Cannabinols. It contains 25% to 40% of the active principle.

Other preparations:

Bhang (Siddhi, Sabji): It is prepared from the dried leaves and fruit shoots. It is the mildest and contains 15% of the active principle.

Majoon: Sweet prepared with bhang. It increases the appetite and sexual desire.

Charas or hashish: It is the resin exuding from the leaves and stems of the plant, and it contains 20 to 40% of the active principle.

Signs and Symptoms:

Euphoria, passivity, disorientation in small doses. Impaired memory, disturbed thought in moderate doses.

Depersonalisation and marked sensory distortion in high doses.

Fatal Dose: Charas-2g, Ganja-8g, Bhang-10g/kg. THC 30 mg/kg.

Fatal period: Several days.

Treatment:

1. Stomach wash/Emesis, activated charcoal and cathartic.
2. 100ml of 50% glucose, 2mg naloxone and 100mg thiamine iv.
3. 5 to 10mg diazepam if the patient is violent/aggressive.
4. Assurance.
5. Haloperidol for flashbacks.
6. Psychotherapy.

Hashish psychosis: In chronic poisoning, rarely, they become insane and may suffer from auditory and visual hallucinations and delusions of persecution.

Run amok: In chronic poisoning, the person may develop a psychic disturbance marked by a period of depression, followed by violent attempts to kill people. He first kills a person whom he may have real or imaginary enmity and then kills anyone that comes in his way until the homicidal tendency lasts.

Medico-legal importance:

1. Accidental ingestion or inhalation.
2. Majun and Charas- road poison.
3. Taken by criminals before committing a criminal act.
4. Aphrodisiac.

7. ACONITE :

(MONK'S HOOD, BLUE ROCKET,MITHA ZAHAR)

It is a cardiac poison. The common varieties are Aconitum napellus & Aconitum ferox .

It grows in Himalayas, all parts are poisonous.

Roots contain maximum poison &contains aconitine & other alkaloids such as pseudoaconitine, aconine, picraconitine etc.

The dry root is conical or tapering with scars of broken rootlets. It has arched or shriveled longitudinal wrinkles.

The colour is dark brown. But in fresh roots, it is white inside, which becomes pink on exposure to air.
It is odourless. It is a classical example of sweet taste.

The root is mistaken for horse radish root which is long cylindrical, yellowish white externally & whitish internally.
It does not change colour & has pungent odour.

Signs &Symptoms:

Root can produce tingling and numbness. The odour has effect similar to narcotic effect.
The pollen causes pain, swelling & inflammation .

Other symptoms are tingling numbness, sweet taste with burning sensation, later there may be excessive salivation, nausea, loose motion, difficulty in swallowing, feels thirsty.

Headache, giddiness, profuse sweating, subnormal temperature, weakness of limbs , cramps & convulsions may develop.

Hippus:

Pupils alternatively dilate & contract & lastly remain dilated.

Visual acuity is decreased withdiplopia , pulse slow &irregular, low BP.

Death is due to paralysis of heart or respiratory centre failure.

Fatal Dose:

1 to 2 mg of aconitine
1 to 2 g of root

Fatal period: Few hours

Treatment:

1) Gastric lavage with weak solution of iodine in potassium iodide or tannic acid. It precipitates the poison.

2) Animal charcoal

3) Injection Atropine 0.5 to 1 mg

4) Symptomatic

PM Findings:

There are findings of asphyxia

The poison is destroyed fast in the body hence must be analysed early.

Medicolegal use:

1. Accidental as it is mistaken for horse radish roots or by taking herbal medicine containing aconite or when it is used in alcohol to increase its potency.

2. For suicidal purpose.

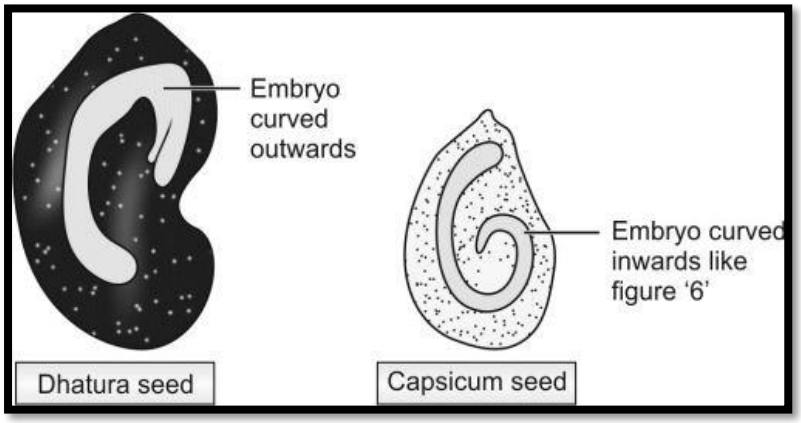
3. For homicidal purpose, it is given along with betel leaves.

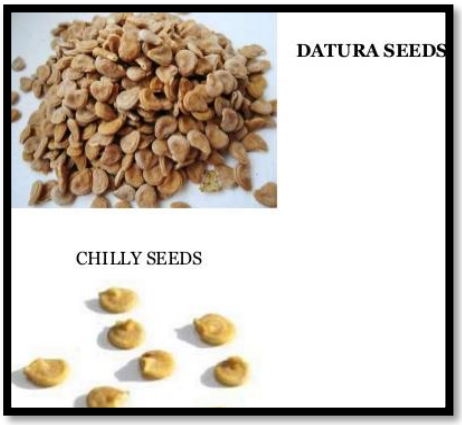
4. Abortifacient.

5. Cattle poison & arrow poison.

Difference between Datura &Chilliseeds :

Trait	Datura Seed	Chilli Seed
1) Size	Large & Thick	Small &Thin
2)Shape	Kidney Shaped	Rounded & Oval
3) Colour	Dark or Yellowish Brown	Yellowish
4) Margins	Laterally compressed & double edged at convex border.	Convex border is sharp.
5) Surface	Small pits present	Smooth
6) Smell	Odourless	Pungent
7) Taste	Bitter	Pungent
8) Embryo	Longitudinal section embryo is carved outward at hilum	Embryo is curved inward at hilum like 6





8. ARGEMONE MEXICANA SEEDS WITH CAPSULE:

This is a small shrub which grows on wasteland all over India, in winter season. It is identified by sessile, spiny thistle like leaves (leaves with spiky growths). The flowers are small and yellow in colour. All parts are poisonous.

The seeds present in elliptic capsule, 2 to 4 g. The seeds are dark brown in colour, smaller than Mustard seeds, spherical & have minute projections when pressed, burst whereas mustard seeds collapse

Active principles:

Plant has two alkaloids, berberine & protopine .
The oil has two alkaloids called sanguinarine & dihydrosanguinarine.

Oil is used as adulterant of mustard oil.

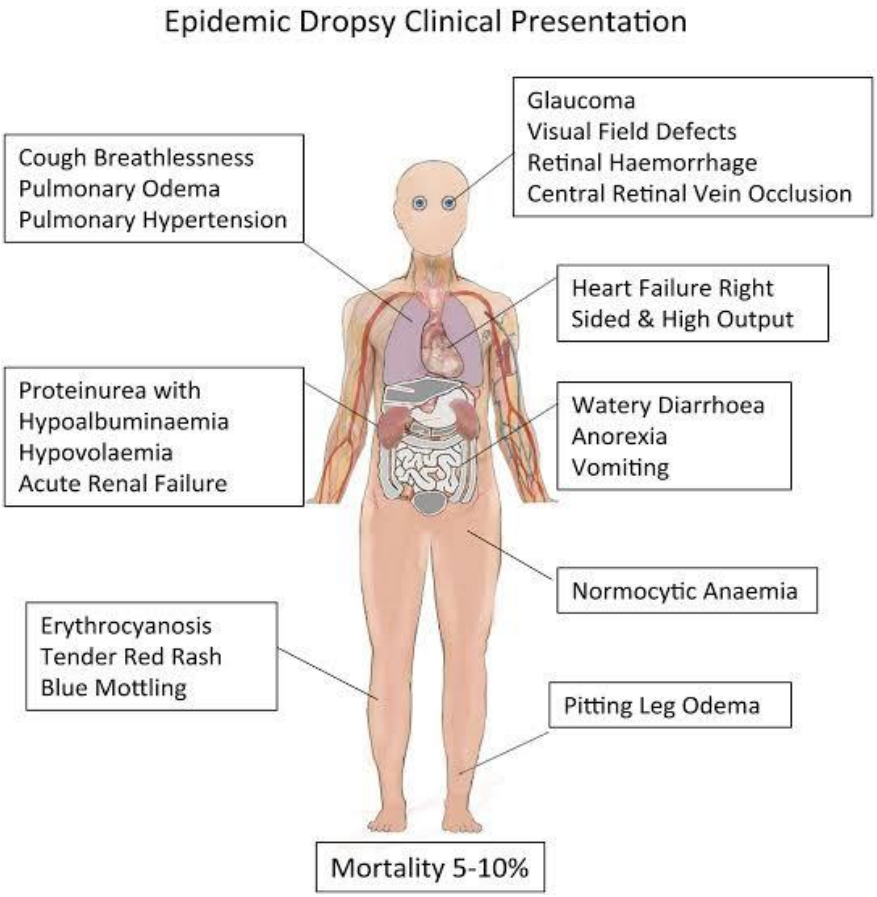
Signs &symptoms:

Slow onset, loss of appetite, discomfort , oedema of legs, diarrhoea, generalised weakness, myocardial damage & dilatation of heart may be present.
BP low with feeble & rapid pulse.

Skin: Bluish, mottling due to dilated vessels. Tingling sensation & Hyperaesthesia and tenderness of muscles.

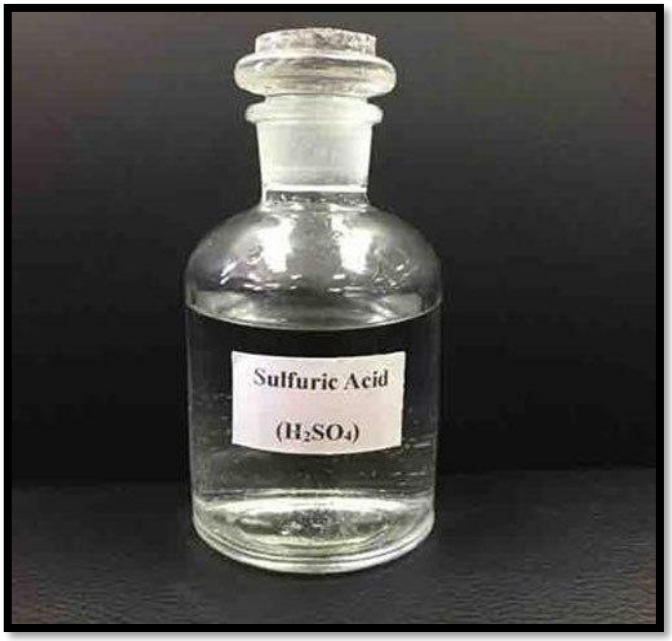
Eyes: Eye sight is reduced due to increased intraocular pressure of glaucoma. Subcutaneous haemangioma or telangiectasia may be seen .

Epidemic dropsy is a clinical state resulting from use of edible oils adultered with argemone mexicana seed oil.



9. SULPHURIC ACID:

(OIL OF VITRIOL,BATTERY ACID,H_2SO_4)



It is a mineral acid, results in coagulative necrosis, mainly acts locally.

Dilute H_2SO_4 acts as irritant , well diluted H_2SO_4 , acts as stimulant. Concentrated H_2SO_4 acts as corrosive.

It is heavy, colourless, oily &hygroscopic .

It carbonises the organic tissues.

Signs &Symptoms:

The teeth are chalky white because enamel is destroyed.

It is the only mineral acid where in pupils are dilated.

Tissues are softened, corroded and blackened, perforation can occur.

Fatal dose:10 to 15 ml.

Fatal period: 18 to 24 hrs.

If concentrated, can kill within half to 1 hour.

Causes of death:

1) Neurogenic shock.

2) Perforation.

3) Toxaemia.

4) Suffocation due to oedema of glottis & larynx.

VITRIOLAGE : (Vitriol throwing)

It is throwing of corrosives on another, especially over face with the intention of disfiguring, usually thrown on female out of sexual jealousy to disfigure.

Strong acid or alkali is collected in a delicate bottle, electric bulb and thrown, when it hits the person, it bursts sprinkling the corrosive. Plant juice (Calotropis, marking nut) are also used.

It produces deep painless burns because cutaneous nerves are damaged. They produce permanent scars and disfigure a person Hence it is treated as grevious injury .

Treatment:

1) Wash with soap water if acid is used. Use weak acid in case of alkali.

2) Use thick paste of Magnesium oxide or carbonate if acid is used.

3) Wash eyes with weak solution of Sodium bicarbonate or olive oil.

Rarely death can occur due to shock, toxaemia, infection or laryngeal oedema .

10. NITRIC ACID:

(AQUA FORTIS, HEAVY WATER RED SPIRIT OF NITRE and HNO_3)

It is a mineral acid when fresh, it is colourless but becomes yellowish after some time .
It is heavy, has peculiar choking odour.

Action: It acts locally & destroys organic matter by oxidation & produces yellowish discolouration of tissues called xanthoproteic reaction. The acid acts on organic matter and produces picric acid which stains the tissues yellow.

Signs &symptoms: It destroys tissues locally and are yellowish in colour. It produces eructations and distension of abdomen. The teeth are stainedyellow.

Fatal Dose: 10 to 15 ml.

Fatal period: 18 to 24 hrs, early if concentrated acid is consumed .

Causes of Death: Same as in sulphuric acid(H_2SO_4) treatment.

Uses:

1) Taken accidentally, or for suicidal purpose.
2)Abortion.
3) Vitriolage.

11. HYDROCHLORIC ACID :

(HCl, MURIATIC ACID, SPIRITSOF SALTS)

It is a mineral acid, with pungent odour, colourless & fuming.
Signs & Symptoms: Tissues become grey or greyish white. Later it turns brown or black.

Chronic Poisoning :
It is the only acid with chronic poisoning features .
This is due to constant exposure to fumes .
Patient c/o coryza, conjunctivitis, corneal ulcer, pharyngitis, laryngitis, bronchopneumonia, gum inflammation & loosing of teeth.

Fatal Dose: 15 to 20 ml

Fatal Period: 18 to 24 hrs. Less if acid is concentrated

Uses:
1) Taken for committing suicide or accidentally.
2) Abortifacient.
3) For vitriolage.

12. OXALIC ACID:

(SALT OF SORREL,ACID OF SUGAR)

It is an organic acid. It is colourless, transparent prismatic crystals form and these crystals resemble crystals of $ZnSO_4$ or $MgSO_4$.

Normally about 20 mg of oxalates are excreted in urine daily.

Action: It is a corrosive poison & it is the only acid which retains corrosive property even on dilution .

Signs & symptoms: Large dose produces shock and death, it produces hypocalcaemia because it combines with calcium. So there are convulsions, tetany and irritability .

There is burning sour taste. If patient survives for some days, the oxalates present in renal tubules leads to renal damage & there is tubular nephrosis or necrosis.
This results in uraemia in 2 to 15 days.

Vomit contains altered blood with mucus. It is coffee ground coloured. The mucosa is bleached & whitened. Stomach shows brownish black streaks. Rarely stomach is softened, corroded & blackened.

Kidneys are swollen, oedematous & congested , tubules contain oxalate crystals.

Uses:

1) Commonly used as a bleaching agent in calico printing, to clean metal or leather .
2) To remove stains from clothes.
3) Criminal use: to erase signature in forgery .

Oxalic acid can lead to acute poisoning, fulminating poisoning & delayed poisoning.

Treatment:

1) Careful stomach wash by calcium lactate or calcium gluconate (when there is not much damage to GIT).

2) **Antidote:** preparation of calcium which convert oxalic acid into insoluble calcium oxalate, lime water, calcium lactate, calcium gluconate, milk can be used.

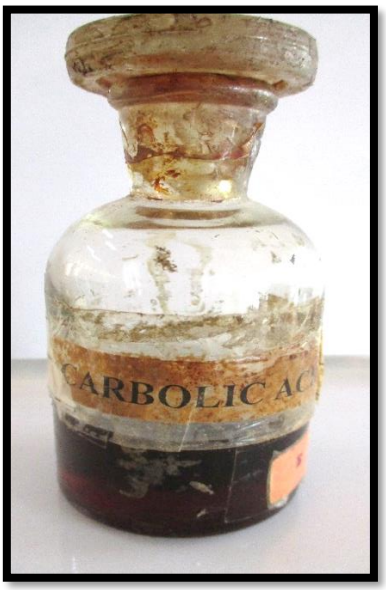
3) I.V10 ml of 10% calcium gluconate.

4) Parathyroid extract 100 units IM.

5) Demulcents.

6) Enema.

13. CARBOLIC ACID (PHENOL, PHENYL, C_6H_5OH)



It is an organic acid. In pure form, it is available as short, colourless, prismatic needle like crystals. But turns in to pink soapy liquid when exposed to air. It has its own characteristic smell called carbolic smell. It has a burning sweetish taste.
Commercially available as dark brown liquid and widely used as disinfectant and antiseptic solution.

Action: It has both local and remote action. It is converted in to hydroquinone and pyrocatechol. Small amounts are excreted through the lungs unchanged and to some extent detoxified in the liver.

Fatal dose: 10 to 15 g.

Fatal period: 3 to 5 hours.

Signs and symptoms:

Poisoning by this acid is known as **Carbolism**.

1) Local effect: Skin burning and numbness due to damage of nerve endings. It precipitates proteins and coagulates all contents. It produces an opaque white eschar that turns brown within a few days. The tissues may be necrotic, gangrenous, with a brownish-white or greenish-white discoloration.

2) On GIT: First tingling sensation, later anaesthesia and corrosion of mucosa. Stomach is hardened, leathery &anaesthetised with congestion and petechial hemorrhages.

3) On Respiratory system: Pulmonary and laryngeal oedema, inflammation, bronchitis & bronchopneumonia may develop.

4) Other systemic effects: Depression of CNS, mainly respiratory centers. Headache, dizziness, confusion, tinnitus, muscular spasm.The temperature is subnormal, pupils are constricted.Face shows sweat & dusky cyanosis. Strong smell of phenol present. There can be convulsions and lock jaw, blood is dark & semifluid.

5) On kidneys: Urine scanty, albumen present with hemoglobin.

Carboluria: Hydroquinone, pyrocatechol with unchanged phenol are excreted in urine. Partly free & some with unstable combination with H_2SO_4& glucuronic acid. Further oxidation gives green urine. This is called carboluria. Kidneys are inflamed and congested.

Oochronosis: Hydroquinone & Pyrocatechol may get deposited into cornea & cartilages.

Causes of Death: Syncope, Asphyxia because of respiratory center failure, Oedema of larynx, Bronchopneumonia.

Treatment:

1. Emetics can be given, but often fails because mucosa is anaesthetised.

2. Stomach wash can be given safely because stomach is hardened & leathery. Give stomach wash with luke warm water with animal charcoal, olive oil, MgO soap solution etc., continue till no smell of phenol comes.

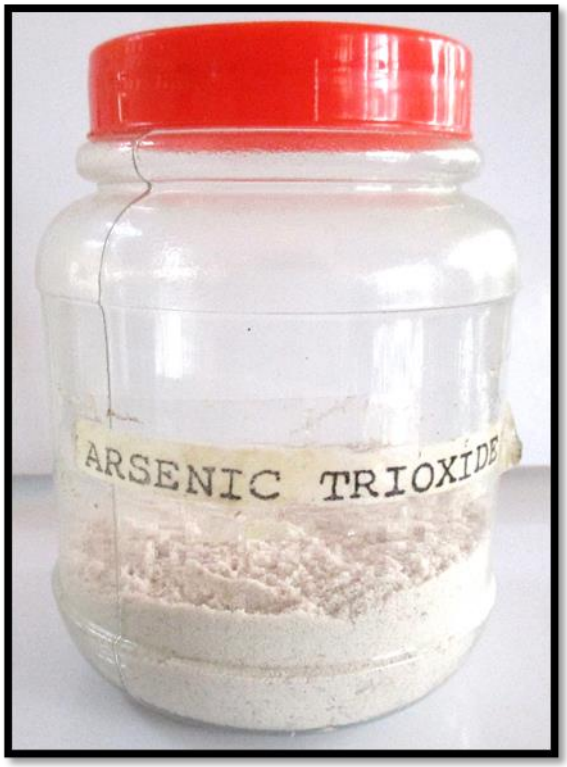
3. 30 g of Epsom salt ($MgSO_4$) or Medicinal liquid paraffin to be left in stomach.

4. Demulcents

5. Symptomatic

<u>Medico-legal importance</u>:
Used for committing suicide, taken accidentally, rarely for abortion.

14. ARESNIC TRIOXIDE



It occurs in two forms- a. white, smooth heavy crystalline powder, b. white and opaque solid mass similar to porcelain.

Action: It interferes with cellular respiration by combining with the sulphydryl groups of mitochondrial enzymes, especially pyruvate oxidase, and certain phosphatases.

Signs and symptoms:

1. **The Fulminant type**: Massive doses of arsenic when rapidly absorbed cause death in one to three hoursfrom shock and peripheral vascular failure or may be delyed for many days.
2. **The Gastroenteric type**:

• GIT: Burning and colicky pain in the esophagus, stomach and bowel. Intense thirst and projectile vomiting. The stools are expelled frequently and involuntarily, and are dark-coloured, stinking and bloody, but later become

colourless, odourless and watery resembling rice-water stools of cholera. A garlicky odour of breath and faeces may be noted.

- Renal: Oliguria, uremia.
- CVS: Acute circulatory collapse
- CNS: Headache, vertigo, tremors, convulsions, coma, general paralysis.
- Skin: Delayed loss of hair, skin eruptions.

3. **Narcotic form**: Giddiness, formication and tenderness of muscles, delirium, coma and death.

Fatal dose: 0.1 to 0.2 g.

Fatal period: 1 to 2 days.

Treatment:

1. Stomach wash with large amount of warm water and milk.
2. Butter and greasy substances prevent absorption.
3. Freshly precipitated, hydrated ferric oxide orally in small doses.
4. B.A.L 400 to 800mg on first day, 200 to 400mg on 2nd and 3rd days, 100 to 200mg for 7 to 10 days.
5. Penicillamine 100mg per kg daily up to 1 to 2 g
6. DMSA or DMPS
7. Demulcents lessen irritation.
8. Castor oil or magnesium sulphate to prevent intestinal absorption of arsenic.
9. Glucose-saline with sodium bicarbonate.
10. Hemodialysis in case of renal failure.

Chronic poisoning:

1. CNS: Polyneuritis, anaesthesia, paraestehsia, encephalopathy.

2. Skin: **Rain drop pigmentation**- Finely mottled brown pigmentation on the skin flexures, temples, eyelids and neck. Hyperkeratosis of the palms and soles with irregular thickening of the nails and the development of transverse white lines in the fingernails called **Aldrich-Mees lines** is seen.
3. Eyes: Congestion, watering of the eyes, photophobia.
4. GIT: Nausea, vomiting, abdominal cramps, diarrhea, salivation.
5. CVS and Kidneys: Chronic nephritis, cardiac failure, dependent edema.
6. Hepatic: Hepatomegaly, jaundice, cirrhosis of liver.
7. Hematologic: Bone marrow suppression, hypoplasia, anemia, thrombocytopenia, leukemia.
8. General: Anemia and weight loss, loss of hair, brittle nails.
9. RS: Cough, hemoptysis, dyspnea.

Medicolegal importance:

1. It is a popular homicidal poison- given orally, mixed with food. It is cheap, easily obtained, colourless, tasteless and odourless.
2. Rarely suicide.
3. Accidental- chronic poisoning from drinking well water containing arsenic.
4. Ingested or applied locally to abortion sticks to produce abortion.
5. Fed to animal mixed with cattle fodder.

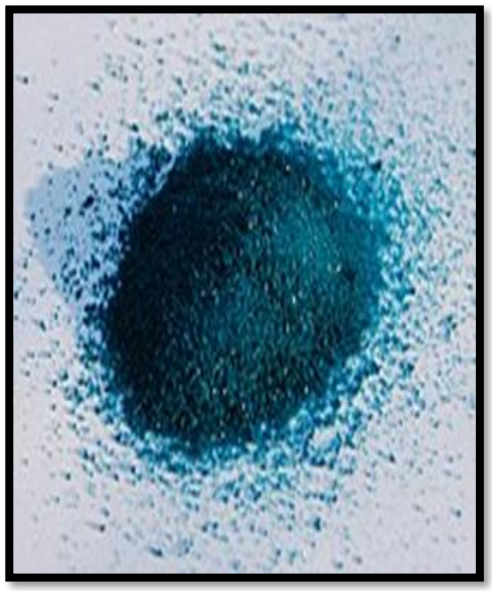
15. COPPER SULPHATE:($CuSO_4$ -Blue Vitriol)

($ZnSO_4$ -White Vitriol, H_2SO_4 -Oil of Vitriol)

The metal copper is not poisonous, but its saltsare poisonous (all metals in pure metallic form are not poisonous except lead)

Copper sulphate - Blue crystals

Copper subacetate - Bluish green mass or powder



Normally copper is present in body.
$CuSO_4$ is an example of a poisoning used in treatment of another poison. It is used as an antidote in phosphorus poisoning.

Signs & Symptoms: It is an irritant poison. So, produces irritation and pain. Vomitus & Stool are bluish in colour. It is hepatotoxic so jaundicedevelops. It is nephrotoxic, there is oliguria, haematuria with albumen, and later uraemia.

Fatal dose: $CuSO_4$ - 30 g, Cu subacetate-15 g

Fatal period: 1 to 4 days

Treatment:

1) No emetics are used as it itself acts as an emetic.
2) Stomach wash with 1% potassium ferrocyanide forms insoluble cupric ferrocyanide.
3) Chelating agents - EDTA, BAL.
4) Demulcents form insoluble albuminate of Cu.
5) Castor oil to remove from intestine.

PM findings: Skin is yellowish if jaundice has developed. Greenish blue froth coming out of mouth and nostrils. Mucosa of mouth will be bluish with inflammation. Spontaneous bleeding at places, degenerative changes in proximal tubules of kidneys may be present.

Chronic poisoning: It is due to inhalation of powder. It leads to gradual anaemia, green line over gums, nausea and vomiting, colicky pain, diarrhoea, malaise. There may be degeneration & atrophy of muscles.

It can cause haemochromatosis, also called Bronzed diabetes or pigmented cirrhosis. Copper is mainly excreted through faeces (other poisons- endrin & lead), traces is excreted in body fluids.

Medico Legal importance:

Used as cattle poison, Abortifacient, Suicide

Accidental poisoning: Accidental poisoning occurs when food, especially vegetables are cooked in copper vessels, which are not covered with layer of tin. Vegetables' acids act on copper vessels and produces copper subacetate (Verdigris) usually such a poison occurs in marriage, fair, where cooking is done in copper vessels.

16. LEAD MONOXIDE: (PbO)

It is pale brick red or orange, scaly mass. Quacks use it for treating syphilis, used by painters & is also seen in hair dye.

Other preparations of lead are lead tetroxide (sindhur), lead sulphide (surma), lead acetate (sugar of lead), lead subacetate, lead carbonate etc.

Lead compounds are present in petrol (lead tetra-ethyl) paints, calico printing - painted toys.

Metal lead is also poisonous, Acute lead poisoning is mainly due to lead acetate.
The poisoning is of three types: 1) Acute 2) Subacute 3) Chronic

Plumbism:
(Chronic lead poisoning, saturnism, saturnine poisoning)

It is due to chronic exposure to lead dust and fumes to person employed in factories using lead compounds. It is also seen in painters, plumbers, enamel workers, glass blowers etc. It can be due to drinking water from lead utensils, chronic use of hair dyes, depilators (hair remover) & cosmetics containing lead. It is also known in India due to eating of ghee stored in copper or brass vessels, tinned vessels. It also occurs if food is cooked in tinned vessels and also due to use of vermillion (red lead) in women over hair line.

Signs & symptoms:

1) **Lead line: (Burtonian line)** It is a blue line at the junction of gum & teeth, more commonly seen in upper jaw, usually at diseased and dirty teeth. It is due to formation of lead sulphide. The decomposing food stuck in the teeth & crevices produce hydrogen sulphide which reacts with lead & liberates lead sulphide. The patient will have foetid breath, sweetish metallic taste dyspepsia and sallow earthy complexion. Similar line is seen in mercury, copper, bismuth, iron and silver poisoning.

2) **Colic &constipation: (Dry belly ache)** Severe colicky pain is noticed, patient gets relief on applying pressure over abdomen, associated with metallic taste, anorexia & constipation, colicky pain is of sudden onset & often at night.

3) **Anaemia:** The typical peripheral blood smear helps to diagnose plumbism. The RBCs show marked punctuate basophilia (called basophilic stippling of RBCs). Hypochromic anaemia, reticulocytosis. Platelet count is reduced. Fluorescent RBCs - 75 to 100%seen under ultraviolet light. Nucleated RBCs are also seen. Anaemia is probably due to rapid destruction of RBCs & inhibition of haeme synthesis by interference with the incorporation of iron into protoporphyrin.

4) **Lead palsy:** It affects extensors of fingers and wrist except supinator longus giving wrist drop and claw shaped hand. There may be paralysis of extensors of foot giving rise to foot drop, tibialis anterior will be spared. The muscles which are used are involved & is a result of motor type of peripheral neuropathy due to axonal degeneration. These patients complain of tingling numbness, joint pain, tremors and weakness later.

5) **Lead encephalopathy:** It is commonly seen in children, infants and rarely seen in adults. There is severe ataxia, vomiting, lethargy, stupor, convulsions and coma. There will be intense headache, loss of concentration, memory, optic neuritis, insomnia, hallucinations, delirium, insanity, coma etc.

6) **CVS involvement:** Arteriosclerotic changes, enhanced rise in BP.

7) **Renal system involvement:** Urine shows albumin, abnormal increase in lead excretion, coproporphyrin III etc. Interstitial nephritis is seen

17. PHOSPHOROUS :

There are two varieties of phosphorus,
White phosphorus and Red phosphorus.

Images		
Features	**White phosphorous**	**Red phosphorous**
Color and appearance	White, waxy, crystalline &translucent	Violet-red &amorphous
Taste and odor	Garlicky odour and taste	Odourless and tasteless
Luminosity	Luminous	Not luminous
Toxicity	Highly toxic	Non toxic

Mechanism of Action :

Yellow phosphorus is a protoplasmic poison and affects cellular oxidation.

It is also hepatotoxic and cardiotoxic

It causes fatty infiltration and necrosis of liver and kidney

Locally it produces severe irritation or burn injuries of skin and mucosa.

Clinical Features :

A.<u>Acute poisoning :</u>

Three stages are usually recognised in acute phosphorus poisoning extending over a period of 8 to 10 days.

First stage: It is one of acute irritation of GIT with vomiting, diarrhoea and abdominal pain. There is garlicky odour. Vomitus and stool may be luminous in the dark. Fumes may evolve from the stools and called as **smoky stool syndrome**.

Second stage: If patient survives, the acuteness of symptoms may subside and condition appears to improve.

Third stage: The symptoms of first stage re-appear with increased severity. Manifestation of hepatic failure in form of tender and enlarged liver, jaundice, pruritus and encephalopathy. There are purpuric hemorrhagic areas and cramps.
Convulsions may appear at later stage. Renal failure develops with oliguria, hematuria, and albuminuria.
Bone marrow depression.
Biopsy reveals decreased cellular mass with degenerative changes.

Local application causes corrosive burns.

B. <u>Chronic poisoning</u>:

- Occurs due to long term exposure.
- Nausea, vomiting, diarrhea, eructation and abdominal discomfort.
- Wasting and weakness of muscle.
- Anemia.
- Jaundice.

Phossy jaw or glass jaw develops: It is osteomyelitis of jawbone (lower jaw) due to chronic phosphorus poisoning.

Management :

- Do not give milk or oily or fatty food/drink because it will enhance the absorption of phosphorus.
- Gastric lavage with potassium permanganate (1:5000).
- Potassium permanganate oxidizes phosphorus into less toxic phosphoric acid and phosphate.
- Intravenous fluid support
- Vitamin K for hypoprothrombinemia..
- Blood/products for correction of coagulation cascade
- Glucose for hypoglycemia..
- Calcium gluconate for hypocalcemia.
- Benzodiazepines for convulsions.

Autopsy Findings :

- Petechial hemorrhages may be noted over skin.
- Jaundice.
- Garlicky odor.
- Gastric mucosa is yellowish or greenish-white in color and is softened.
- Gastric contents emit garlicky odor and luminous in dark.
- Liver shows necrobiosis. Liver is enlarged, doughy inconsistency, uniformly yellow and contains many hemorrhagic areas in parenchyma.
- Heart, kidneys and voluntary muscle fibers shows fatty degeneration.
- On microscopy, hepatocellular necrosis and cholestasis are seen.

Medicolegal Importance:

- Accidental poisoning – few
- Suicidal or homicidal poisoning – rare..
- Yellow phosphorus rolled up in wet cloth was employed to set fire to postal letterboxes during the Indian civil disobedience movement in 1932.

Post mortem Appearance :
Stomach Contents may smell like kerosene and mucosa is congested with submucous petechial hemorrhages.

Lungs show gross congestion , edema and sub-pleural petechiae Brain is congested , edematous with petechiae.

Medicolegal Importance : Suicide is very common.

18. MERCURY:

(Quick silver)

Only metal which is liquid at room temperature .

Metallic mercury is having bright silvery appearance and is volatile at room temperature.
The fumes are odourless and invisible.

It forms mercuric and mercurous compounds. Mercuric iodide is amongst the poisonous compounds of mercury and is in scarlet-red powder form.

Mechanism of Action :

Mercury compounds act by inactivating sulfhydryl causing interference with cellular metabolism.

Clinical Features :

Acute poisoning

- Inhalation: Breathlessness.

- Injection: Subcutaneous or intramuscular injection of mercury causes abscess formation with ulceration.
- Ingestion: Metallic taste, Abdominal pain, Vomiting.

Chronic poisoning: also called as **Hydrargyrism.**

Management :

• Gastric lavage with egg white or albumin or milk to bind the mercury.
• Demulcents.
• Laxative.
• X-ray follow up.
• Chelation with BAL, DMPS.

Autopsy Findings :

• Emaciated body.
•Mouth, throat, stomach appear grayish with softening and corrosion with hemorrhagic areas.
• Liver and heart – fatty degeneration.
• Kidneys – pale, swollen with edema of renal cortex with necrosis of renal tubules.

Medicolegal Importance :

• Poisoning occur due to accidental consumption.
• Homicide and suicide – rare.
• Use to procure criminal abortion.

19. FOLIDOL (OP COMPOUND) :

Derived from phosphoric acid

Signs & Symptoms:

1. Muscarinic manifestations:

Diarrhoea, Urination, Miosis, Bronchospasm, Emesis, Lacrimation, Salivation. (DUMBELS)

2. Nicotinic manifestations :

Fasciculations, Cramps, Weakness, Hypertension, Tachycardia,
Headache, Tremors, Drowsiness, Ataxia, Convulsions, Coma.

Mechanism of action:

Potent inhibitor of ACETYL CHOLINESTERASE which is responsible for hydrolyzing acetyl choline. They bind to acetyl cholinesterase molecule at the active site and phosphorylate the serine moiety and inhibits it results accumulation of acetyl choline.

Fatal dose: 175 mg. Orally or 80 mg I.M

Fatal period: within few hours if not treated.

Treatment:

Patient removed from the source of exposure.

- Airway.
- Stomach wash.
- Activated charcoal lg/kg.
- Atropine sulphate to arrest muscarinic effects.
- Cholinesterase reactivators like di-acetyl monoxime or pralidoxime iodide.
- Control of convulsions and treatment of pulmonary edema.