Post-mortem Changes:

ISHITA KANODIA

Signs of Death: (in order)

I] Immediate (somatic death):

i) insensibility & loss of voluntary power → earliest sign of death
ii) cessation of respiration
iii) cessation of circulation

II] Early (cellular death):

iv) Pallor & loss of elasticity of skin
v) Changes in eye
vi) Primary flaccidity of muscles
vii) Cooling of the body
viii) Postmortem lividity
ix) Rigor mortis.

III] Late (decomposition & decay):

x) Putrefaction
xi) Adipocere formation
xii) Mummification.

Suspended Animation (Apparent Death):

→ signs of life are not found as the functions are interrupted for sometime or are reduced to minimum.
→ life continues & resuscitation is successful.
→ may be produced voluntarily (yoga-practitioners)
→ seen in: vagal inhibition, severe syncopal attacks, newborn infants, drowning, electrocution, sunstroke, cholera, narcotic poisoning, hypothermia.

Changes in the Skin:

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→ skin becomes pale & ashy-white, loses elasticity (within few minutes)

→ lips ⇒ dark-red to black, dry, hard

Changes in the Eye:

1) Loss of Corneal Reflex

2) Opacity of Cornea: may occur in certain diseases before death (cholera, wasting)

→ occurs due to drying (delayed for 2 hours if lids are closed after death)

→ if lids are open for a few hours after death:

- film of cell debris & mucus forms 2 triangles on the sclera at each side of the iris with base of triangle towards margin of cornea & apex towards medial/lateral canthus of eye which becomes brown & then black ⇒ Tache noir.

3) Flaccidity of Eyeball: eye looks sunken & become softer (due to ↓ in intraocular tension)

4) Pupils: slightly dilated (due to relaxation of muscles of iris) soon after death

→ later ⇒ constricted (due to algor mortis of constrictor muscles & evaporation of fluids)

→ pupils do not react to light after death.

5) Retinal vessels: fragmentation/segmentation of blood columns in retinal vessels ⇒ Kevorkian Sign

→ appears few minutes after death & persists for about half-an-hour.

→ retina is pale for first 2 hours

Algor Mortis [Cooling of Body / Loss of Body Heat]:

→ after stoppage of circulation ⇒ convectional transport of heat inside body stops.

→ initially ⇒ heat is lost from superficial layers of body only ⟶ low velocity of heat transport inside the body ∴ takes time for heat to be conducted from deeper layers to more superficial layers ⟶ finally ⇒ temperature gradient reaches the core.

→ internal organs mainly cool by conduction.

→ Postmortem temperature plateau: for about ½ - 1 hour after death ⟹ rectal temperature shows little/no fall.

→ Next 12 - 16 hours ⟹ linear rate of cooling (0.4 - 0.6 °C/hour)

→ Temperature of the body does not drop significantly when it is within 4°C of the environment.

→ Curve of cooling ⟹ Sigmoid / inverted S-shaped

→ rectum is the ideal place to record temperature except in cases of sodomy. (rectal temperature is 0.6°C higher than oral temperature)

$$\text{Time of death (hrs)} = \frac{\text{Normal body temperature} - \text{Rectal temperature}}{\text{Rate of temperature fall per hour}}$$

Factors Affecting Rate of Cooling:

1) Difference in temperature between body & environment:
more temperature difference ⟹ more rapid loss of body heat

2) Build of the cadaver: more surface area ⟹ more heat loss

3) Physique of cadaver: fat is a bad conductor of heat
∴ fat bodies cool more slowly.

4) Environment of the body: Body kept in well-ventilated room loses heat more rapidly.
→ Moist air is a better conductor of heat than dry air ∴ more humidity facilitates faster cooling

5) Covering on/around the body: rate of cooling is slow when body is clothed

MLI: determination of body temperature is important only in cold & temperate climates

Postmortem calogicity: temperature of the body remains raised for first 2 hours or so after death.

→ occurs when:
- regulation of heat production has been severely disturbed before death (as in sunstroke, nervous disorders)
- there has been a great increase in heat production in muscles due to convulsions

(in tetanus & staychnine poisoning)

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- there has been excessive bacterial activity (septicaemia, cholera, other fevers).

Postmortem Hypostasis:

aka postmortem staining / cadaveric lividity / liver mortis.

bluish-purple / purplish-red discolouration (due to deoxyhemoglobin) which appears under the skin in the most superficial layers of dermis (rete mucosum) of the dependent parts of the body after death due to capillo-venous distension.

Mechanism: caused by:
- stoppage of circulation
- stagnation of blood in blood vessels
- tendency of blood to sink by force of gravity.

→ blood tends to accumulate in toneless capillaries & venules of dependent parts of the body.

→ upper portions of the body drained are pale.

→ intensity of colour depends upon the amount of reduced hemoglobin in blood

→ some backflow of blood from venular end of capillaries adds to blueness of blood after death.

Development: begins shortly after death (~ ½-1 hour in normal individuals, 1-4 hours in anemic persons)

1) 0.5-2 hours: mottled patches of about 1-2 cm diameter (can be mistaken for bruises)

→ patches are initially seen on upper surface of body → areas then combine, enlarge & slide down to produce extensive discolouration

→ plasma also accumulates (good site for bacterial growth)

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2) 4 hours: well-developed like a sheet but not fixed

3) 8 hours: (6-12 hours) fixation occurs ⇒ Maximum PM staining ⇒ Primary lividity

[Fixation ⇒ if body is turned over to a different position, the postmortem staining would not disappear]

→ Causes of fixation: • clotting of blood within capillaries

• capillaries become permeable → blood leaks through them → stains tissues

• rigor mortis obliterates the big vessels

4) >8 hours: PM staining persists till putrefaction sets in.

→ if body is shifted within 8 hours ⇒ old patches of lividity disappear & new patches develop ⇒ Secondary lividity.

Influencing factors:

i) Volume of blood in circulation at the time of death

ii) Length of time for which blood retains its fluidity after death

iii) More intensity in conditions where blood does not readily coagulate:

• asphyxia • CO poisoning • Septicaemia • amniotic fluid embolism

Various Colours of PM staining:

Colour	Cause of Death
Black	Mummified bodies, opiate poisoning
Bluish-green	H_2S
Bluish-violet	Asphyxia
Bright pink	Hypothermia, refrigerated bodies
Bright red	HCN, burns, refrigerated bodies
Cherry red	CO poisoning
Chocolate	Acetanilide, Aniline, Bromates, Chlorates
Dark brown/yellow	Phosphorus toxicity

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Distribution: depends on position of the body

(1) Body lying on its back (supine): Neck ⟶ entire back (upto flanks)

→ Areas of direct pressure do not show staining (∵ pressure prevents capillaries from filling) & appear pale / free from colour ⇒ Vibices [occipital scalp, shoulder blades, buttocks, calves, heels]

- this is also caused by pressure of one area of the body by another ⇒ Contact pallor / mirror-image blanching.

(2) Body lying in prone position:

→ Connective tissues in front are relatively loose (as compared to those at the back) ⟶ colour of lividity is intense & Tardieu spots are common.

→ petechial haemorrhages & cutaneous blood blisters may develop in areas of hypostasis

→ minute blood vessels in nose may rupture ⇒ bleeding.

(3) Body Suspended (as in hanging):

→ PM staining develops on dependent parts of hands & legs, undersurface of chin, breasts & genitals ⇒ Glove & stocking pattern.

Internal Hypostasis: hypostasis occurs in dependent parts of internal organs also

Body in supine position: hypostasis in – posterior parts of cerebrum & cerebellum
– dorsal portion of lungs
– posterior wall of stomach
– dorsal portion of heart, liver, kidney, spleen
– lowermost coils of intestine.

Muscular Changes: < Primary flaccidity / Rigor mortis

Primary flaccidity: death is only somatic (no cellular death)

→ lasts for 1-2 hours

→ all muscles of the body begin to relax soon after death.

→ **Contact flattening:** body flattens over areas which are in contact with the surface on which it rests

→ muscles are relaxed only as long as ATP is sufficiently high to permit splitting of actin-myosin cross-bridges.

Rigor Mortis: (Death stiffening / Cadaveric Rigidity) individual cell death takes place.

Mechanism: physico-chemical change occuring in muscles

→ Normally ⇒ relaxation of muscles is achieved by energy-dependent (ATP-driven) pumping of calcium back across the membrane of the sarcoplasmic reticulum.

→ After death ⟶ lack of ATP ⟶ increased calcium levels in sarcomere ⟶ muscle contraction ⟶ actin-myosin crossbridges cannot break

→ Actomyosin complex is rigid & causes hardness of muscles

→ contraction persists until it is physically disrupted by the onset of putrefaction.

Order of Appearance: It does not start in all muscles simultaneously ⇒ Nysten's rule

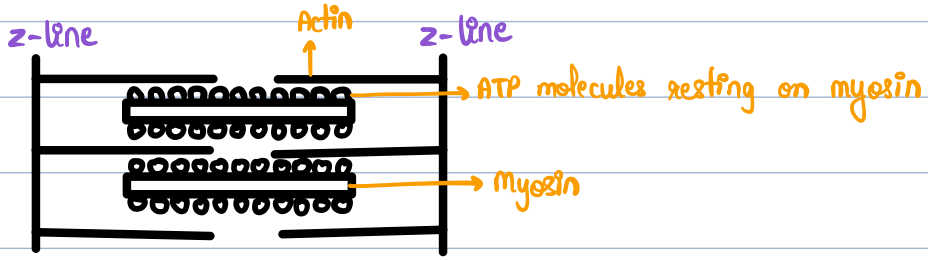
→ first appears in involuntary muscles

- appears first in myocardium in 1 hour ⟶ other involuntary muscles ⟶ voluntary muscles

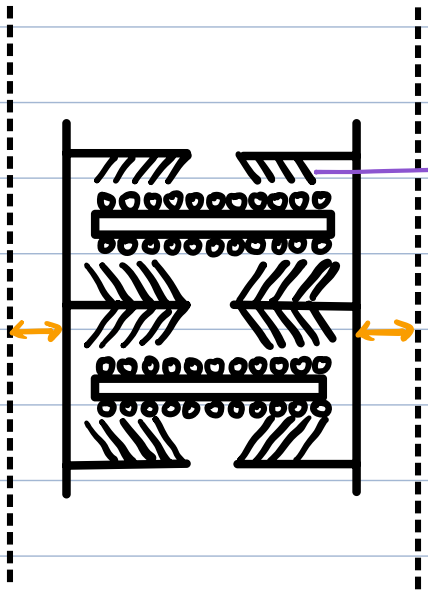
→ appears in proximo-distal fashion

Rule of 12:
- Rigor mortis appears in the entire body in proximodistal fashion in ~12 hours
- Persists for another 12 hours
- Disappears in the next 12 hours in the same proximodistal fashion ∴ body is relaxed again in 36 hrs (secondary relaxation)

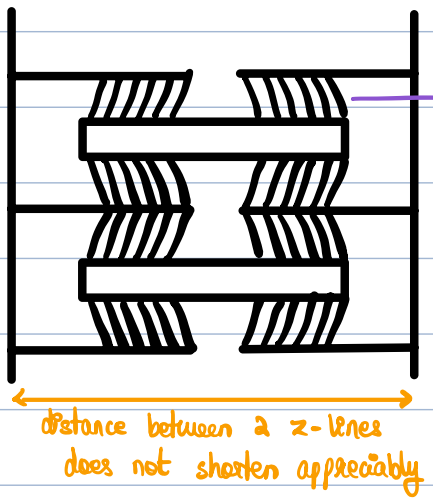
Relaxed muscle during life



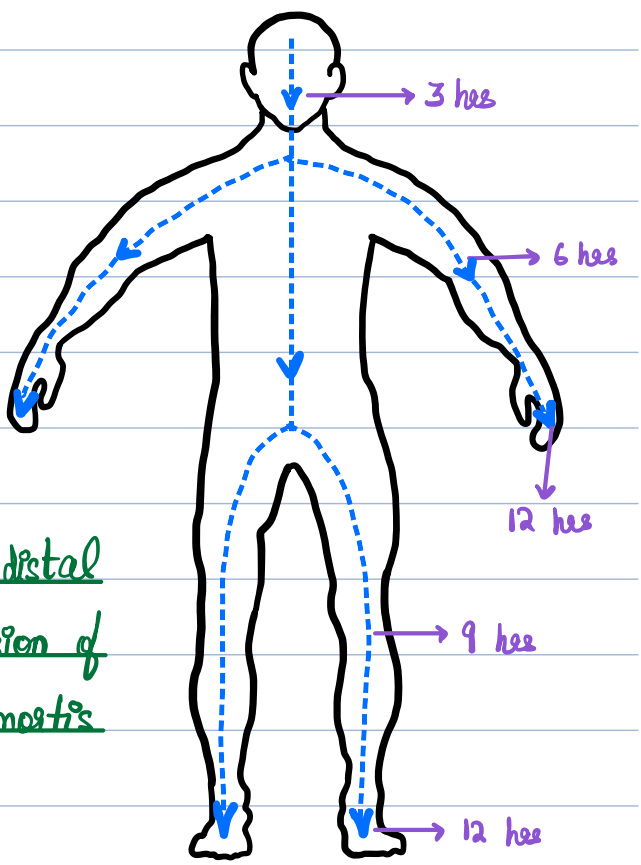
Contracted muscle (during life)



Muscle in rigor mortis (after death)



Proximodistal progression of Rigor mortis



Conditions altering onset & duration of Rigor Mortis:

1] Age: fetus < 8 weeks does not show RM (∵ muscles begin to form at 8 weeks)
→ In children & elderly ⇒ RM is feeble, develops more rapidly & passes away early.

2] Nature of death:
→ RM is sometimes absent in septicemia
→ RM appears early & passes away early in: • bacterial infections (putrefaction begins early)
• death preceded by convulsions (DDT, OP, strychnine)
• violent deaths (electrocution/ firearm/ lightning)
→ RM appears late & passes away late in: • Asphyxia • CO poisoning

3] Muscular state:
→ Slow onset, long duration, substantial strength: muscles healthy & at rest before death.
→ Slow onset, short duration, weak strength: emaciated weak persons
→ Rapid onset, short duration: • Convulsions • Exercise • Exhaustion

	Rigor Mortis	Cadaveric Spasm
Production	freezing & exposure to temp. > 65°C produces RM	Cannot be produced by any method after death.
Mechanism	Known ⇒ reduction of ATP.	Not clearly known.
Predisposing factors	None	Sudden death/ excitement/ fear/ exhaustion/ nervous tension/ etc.
Time of onset	1-2 hours after death	Instantaneous
Muscles involved	All muscles of the body (voluntary & involuntary)	Usually restricted to a single group of voluntary muscles.
Muscle stiffening	Not marked (can be overcome)	Marked (great force required to overcome)
Molecular death	Occurs	Does not occur

	Rigor Mortis	Cadaveric Spasm
Body heat	Cold	Warm
Electrical stimuli	Muscles do not respond	Muscles respond
Muscular reaction	Acidic	Alkaline
MLI	Indicates time of death	Indicates mode of death (suicide/homicide/accident)

MLI of Rigor Mortis:

1] It is a sign of death
2] Helps in estimating time of death (but, not reliable)
3] Indicates position of the body at the time of death

Conditions Simulating Rigor Mortis:

1] Heat stiffening: when body is exposed to temperatures > 65°C.
→ rigidity is much more marked than in RM.
→ seen in deaths from burning, high voltage electric shocks & from falling into hot liquid.
→ there is flexion of limbs due to muscle contraction

2] Cold stiffening: when a body is exposed to freezing temperature before acid metabolites appear in the muscles → freezing of body fluids & solidification of subcutaneous fat → tissues become frozen & stiff.
→ If body is placed in a warm atmosphere ⇒ stiffness disappears & after sometime ⇒ normal RM develops.

3] Cadaveric spasm/Instantaneous Rigor: MLI:

i) Sometimes, in case of suicide ⟹ weapon of suicide (knife, pistol, etc.) is seen firmly grasped in victim's hand
   - ordinary rigor does not produce the same firm grip (can be differentiated from an attempt to simulate cadaveric spasm to conceal murder)

ii) If the victim dies due to assault ⟹ some part of clothing of assailant or hair of assailant may be grasped firmly in the hands.

iii) In case of drowning ⟹ grass/weeds/leaves may be found firmly grasped in the hands of the victim ⟹ sign of antemortem drowning.

Decomposition: < Autolysis / Putrefaction

Autolysis: self-digestion of tissues

→ death → cell membrane becomes permeable → release of cytoplasmic enzymes (digestive enzymes – lysozymes, hydrolases) → autodigestion & disintegration of organs (without bacterial influence)

→ accelerated by heat; slowed by cold temperature

→ First affected: organs rich in enzymes (glandular organs)

→ Dead foetus in utero → autolysis → maceration of foetus

→ in case of intracranial lesions ⟹ autolytic digestion of brain starts before death

→ Earliest external sign of autolysis: whitish, cloudy appearance in cornea.

Putrefaction: disintegration of body tissues after death (a.k.a decomposition)

→ putrefaction sets in after disappearance of rigor mortis. (starts earlier in hot season)

Mechanism: organisms from GI tract / respiratory tract / external skin → multiplication of bacteria begins in 4 hours → multiplication reaches peak within 24–30 hours

→ bacterial growth is favoured by fall in oxygen concentration in tissues & rise in H^+ ion concentration after death → bacteria spread through blood vessels → destruction of soft tissues.

→ Chief destructive bacterial agent ⇒ Clostridium welchii ⇒ marked haemolysis, liquefaction of postmortem clots, disintegration of tissues, gas formation in blood vessels & tissue spaces (C. welchii produces lecithinase most importantly)

→ Other organisms: • Streptococci • staphylococci • Lactobacilli
• B. coli • B. proteus

Features: i) Changes in colour of tissues
ii) evolution of foul-smelling gases
iii) Liquefaction of tissues.

I] Colour Changes:

1) External: Early stage ⇒ haemoglobin diffuses through blood vessels & stains surrounding tissues reddish-brown → colour changes gradually as various Hb derivatives are formed to greenish-black.

→ first external sign of putrefaction in a body lying in air ⇒ greenish discolouration of skin over the region of the caecum.

• internally, this is seen on undersurface of liver, anterior peritoneal surface of right lobe of liver & adipose tissue around gallbladder (areas of contact with hepatic flexure & transverse colon)

• more clearly appreciated in fair-skinned people than in dark-skinned.

• Occurs in ~ 18 hrs in summer & ~ 36 hrs in winters.

• Mechanism: Haemoglobin $\xrightarrow{H_2S}$ Sulfhaemoglobin
(H_2S formed by bacteria which break up sulfur-containing amino acids.)

→ green discolouration soon spreads to the entire abdomen, external genitalia neck, face, arms & legs.

2) Marbling of skin: superficial veins are stained greenish-brown / purplish-red (depending on the total amount of sulfhaemoglobin formed.

→ haemolysis of red cells → staining of vessel walls & tissues due to SulfHb → linear branching pattern in affected blood vessels → marbled appearance

→ starts in 24 hours; prominent in 36-48 hours.

II] Evolution of foul-smelling gases:

→ proteins & carbohydrates are reduced to simples compounds (amino acids, methane, CO, CO_2, H_2, H_2S, mercaptans, NH_3, PH_3).

- many of these gases are inflammable.

→ Foul-smell: mainly due to production of ptomaines (cadaveric alkaloids)

- cadaverine (decarboxylation of lysine)
- putrescine (decarboxylation of ornithine)

→ Postmortem blisters are formed from collection of gases between epidermis & dermis

- first formed on areas which contain more plasma due to hypoplastic edema.
- time of development: ~ 36 hrs ⇒ summer
  ~ 48 hrs ⇒ winters

→ Eyes are softened & may bulge from sockets

→ Face is swollen & discoloured

→ Expulsion of urine & faeces in 2-3 days.

→ Sutures of skull are separated with flowing out of liquefied brain.

→ Swelling of entire body.

→ due to presence of gas in abdomen, the diaphragm is forced upwards ⇒ compressing the lungs & heart & bloodstained froth exudes from the mouth & nostrils ⇒ postmortem purge.

III] Maggots: produced in 1-2 days

→ they have proteolytic enzymes which dissolve the tissue.

→ unusually large accumulation of maggots on one area of the body ⟹ may indicate a pre-existing antemortem wound.

→ Skin slippage ⟹ due to release of hydrolytic enzymes resulting in slippage of epidermis over dermis.

Distribution: order of putrefaction: abdomen → chest → neck → face → legs → shoulders → arms

→ putrefactive changes are more advanced & marked in the dependent body parts.

→ Order of putrefactive changes in internal organs: Larynx & trachea → stomach, intestine, pancreas, spleen → liver, lungs → brain → heart → kidneys, bladder → prostate, uterus → skin, muscles, tendons → bones

Honeycomb liver: foamy appearance of liver

→ C. welchii forms characteristic small clumps in a tissue space & produce gas, which increase in size soon → small yellowish-grey dendritic figures in the parenchyma → greenish discolouration extends to the whole organ → organ finally becomes coal-black.

→ gall bladder resists putrefaction for a long time.

Conditions affecting rate of Putrefaction:

1] Temperature: putrefaction begins above 10°c, optimum at 21 – 38 °c.

→ increase in temperature by 10°c ⟹ rate doubles.

2] Moisture: necessary for putrefaction (drying of the body prevents putrefaction).

3] Air: free access of air hastens putrefaction partly.

4] Clothing: hastens putrefaction initially (by maintaining body temperature)

5] Manner of Burial: putrefaction is less if body is buried soon after death.

6] Age: bodies of newborn infants who have not been fed decompose very slowly.

7] Sex: No effect.

8] Condition of the body: fat & flabby bodies putrefy more rapidly than lean bodies.

9] Cause of death: Death due to septicemia, peritonitis, asphyxia, etc. ⇒ putrefy rapidly.

→ Putrefaction is delayed after death due to wasting diseases, anemia, chronic heavy metal poisoning, etc.

Putrefaction in Water: Casper dictum: a body decomposes in air twice as rapidly as in water & 8 times as rapidly as in earth.

→ putrefaction is more rapid in warm, fresh water than in cold, salt water.

Skeletonisation: last stage of putrefaction when a body is reduced to a skeleton.

→ time varies considerably.

→ Body exposed to carnivores, flies, maggots, insects, rodents ⇒ few days

→ Body in water ⇒ attacked by aquatic animals (clothing is protective).

Buried bodies: factors modifying rate of skeletonization:

- access to air
- acidity of soil
- amount of soil water
- climatic & seasonal variations.

→ buried bones decay according to the acidity of the soil.

Adipocere / Saponification: modification of putrefaction

→ fatty tissues of the body change into a substance known as adipocere

→ seen most commonly in bodies immersed in water or in damp, warm environment.

Mechanism: occurs due to gradual hydrolysis & hydrogenation of pre-existing fats into higher fatty acids, which combine with Ca^{2+} & NH_4^+ ions to form insoluble soaps, which being acidic ⇒ inhibit putrefactive bacteria.

→ Body fatty acid content: At death ⇒ 0.5 %

Within 1 month ⇒ 20%

By 3 months ⇒ 70%.

→ process starts under the influence of intrinsic lipases & is continued by bacterial enzymes (lecithinases mainly).

→ water is essential for the process.

Factors influencing adipocere formation:

→ water is essential for the process.

→ delayed by cold, hastened by heat.

→ warm, moist, anaerobic environment ⇒ favours adipocere formation.

→ more frequent in females, obese, mature newborn children, corpses submerged in water for a long period.

→ foetuses under 7 months do not show this change.

Properties:

→ distinct offensive / sweetish smell. (penetrating ammoniacal odour in early stages)

→ fresh adipocere: soft, moist, whitish, translucent

→ old adipocere: dry, hard, cracked, yellowish, brittle.

→ inflammable, floats in water, dissolves in alcohol & ether.

Distribution: formed in any site where fatty tissue is present.

→ Usual sites: face, buttocks, abdomen, breasts.

→ epidermis disappears as adipocere is formed & dermis becomes darkened.

→ multiple whitish-grey rounded outgrowths of 1-10 mm

→ intestines & lungs are usually parchment-like in consistency & thinness.

Mummification: (modification of putrefaction) ⟹ dehydration or drying & shrivelling of cadaver occurring from evaporation of water [natural appearances of body & general facial features are preserved].

Features:

→ begins in the exposed parts of the body (face, hands, feet & then extends to entire body including internal organs).

→ skin: • shrunken, contracted, dry, brittle, leathery, rusty brown-black
• tightly stretched across anatomical prominences

→ entire body loses upto 60-70% weight

→ arms are often abducted at the shoulder, flexed at the elbows & hands are clenched into fists (flexion is often seen in lower limbs also) ⟹ due to shrinkage of muscles & tendons.

→ mummified tissues: dry, brown, leathery

→ internal organs: shrunken, hard, dark brown-black

Factors Necessary for Production of Mummification:

→ absence of moisture in air.

→ continuous action of dry/warmed air.

Time required: 3 months - 1 year (for complete mummification).

Embalming: treatment of dead body with antiseptics & preservatives to prevent putrefaction & preserve the body.

→ proteins are coagulated, tissues are fixed, organs are bleached & hardened, blood is converted into a brownish mass.

→ produces a chemical stiffening similar to rigor mortis (embalming rigidity is permanent & normal rigor does not develop).

→ decomposition is inhibited for several months.

→ embalming alters the appearance of the body, tissues, organs ⟹ difficult to interpret any injury / disease.

→ required when a body has to be transported to distant places

Typical Embalming Fluid:

• Formalin	preservative
• Sodium borate	buffer
• Sodium citrate	anticoagulant
• Glycerin	Wetting agent
• Sodium chloride	Controls pH
• Eosin	Cosmetic
• Soluble wintergreen	Perfume
• Water	Vehicle.

Estimation of Postmortem Interval:

→ Interval between death & time of examination of a body ⟹ Postmortem interval.

→ Importance: – to know when the crime was committed
– to confirm / disprove an alibi
– to check on a suspect's statements.

→ longer postmortem interval ⟹ wider range of estimate of time since death.

Estimation of Time:

1] Algor mortis

2] Postmortem lividity

3] Rigor mortis

4] Progress of decomposition, adipocere & mummification

5] Entomology of the Cadaver: Myiasis = condition caused by infestation of body by fly maggots.

Developmental Stage	Summers	Winters
Flies lay eggs	within minutes	within hours
Pupa	3 days	8 days
Adult	5 days	5 days
TOTAL =	10-11 days	22 days

6] G.I.T.: amount of contents & their extent of digestion ⟹ useful if hour at which the deceased took his last meal is known.

→ average emptying time of stomach = 3 hours

→ digestion is an active antemortem process which does not continue after death.

7] C.S.F.: after death ⟹ amount of potassium increases constantly in relation to temperature of the body during first 20 hours.

8] Blood: progressive increase of lactic acid.

→ acid phosphatase ↑

→ amylase ↑

→ LDH ↑

→ Sodium level ↓

9] Pericardial fluid

10] Synovial fluid: linear ↑ in potassium.

11] Vitreous humour: ↑ in potassium concentration

→ linear relationship between vitreous potassium concentration & time elapsed after death upto 120 hours.

→ ↓ in sodium & chloride concentration.

→ ↓ in vitreous glucose concentration.

12] Muscle enzymes: linear ↑ in myofibrillar protease activity.

→ linear ↓ in creatinine phosphokinase activity.

13] Hair

14] Scene of Death