35th Edition

The Essentials of Forensic Medicine & Toxicology

As per the Competency-based Medical Education Curriculum (NMC)

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SIGNS OF DEATH AND CHANGES FOLLOWING DEATH

Forensic taphonomy is the interdisciplinary study and interpretation of postmortem processes of human remains in the dispositional context, **i.e., the history of changes of a body following death.** A knowledge of the signs of death help to differentiate death from suspended animation. The changes which take place may be helpful in estimation of the approximate time of death. **Order of appearance of signs of death:**

(I) Immediate (somatic death).
(1) Insensibility and loss of voluntary power.
(2) Cessation of respiration.
(3) Cessation of circulation.
(II) Early (cellular death).
(4) Pallor and loss of elasticity of skin.
(5) Changes in the eye.
(6) Primary flaccidity of muscles.
(7) Cooling of the body.
(8) Postmortem lividity.
(9) Rigor mortis.
(III) Late (decomposition and decay).
(10) Putrefaction.
(11) Adipocere formation.
(12) Mummification.

Insensibility and Loss of Movement: This is the earliest sign of death, but it can lead to error if precautions are not taken. They are found in cases of prolonged fainting attack, vagal inhibitory phenomenon, epilepsy, trance, catalepsy, narcosis, electrocution, etc.

Cessation of Respiration: This must be complete and continuous. The stethoscope is placed over the upper portions of the lungs and larynx where the faintest breath-sounds can be heard. **Complete stoppage of respiration for more than 5 minutes usually causes death.** Respiration may stop for a very short period without causing death. It may occur. (1) as a purely voluntary act, (2) Cheyne-Stokes breathing, (3) drowning, and (4) newborn infants.

Feather test, mirror test and Winslow's test are of historical importance only.

Cessation of Circulation: The stethoscope is placed over the precordial area where the heartbeat can be heard readily. Under normal conditions, **stoppage of heartbeat for more than 5 minutes is irrecoverable and is accepted as evidence of death.**

Magnus test, Icard's test, diaphanous test, fingernail test, and heat test are of historical importance only.

SUSPENDED ANIMATION (apparent death): **In this condition signs of life are not found, as the functions are interrupted for some time, or are reduced to minimum.** However, life continues and resuscitation is successful in such cases. **The metabolic rate is so reduced that the requirement of individual cell for oxygen is satisfied through the use of oxygen dissolved in the body fluids.** In freezing of the body, or in severe drug poisoning of the brain, the activity of brain can completely stop and in some cases start again.

Types: (1) Suspended animation may be produced voluntarily. **Practitioners of yoga can pass into a trance, death-like in character.** (2) Involuntary suspension of animation **lasting from a few seconds to half-an-hour or more may be found** in vagal inhibition, severe syncopal attacks, newborn infants, drowning, electrocution, sunstroke, cholera, narcotic poisoning, after anaesthesia, shock, hypothermia, cerebral concussion, insanity, etc. The patient can be resuscitated by cardiac massage or electric stimulator and artificial respiration.

CASE: (1) A physician was called to examine an elderly man at his home. The doctor saw the man lying in bed motionless, quickly checked the man's heart beat and pulse and declared the man dead. In the autopsy room, while the body was being placed on the table, the doctor heard a gurgling sound and noticed a slight swallowing movement. The man was rushed to a hospital and lived for two more months.

(2) Two doctors, both experienced physicians, were driving behind an omnibus which ran over a child. They went to the scene and found the child lying under the front wheel of the bus; she was black in the face and appeared to be dead. Of this they were quite certain. It was impossible to extricate the child until the vehicle was reversed to set her free, a step which the driver took only after considerable persuasion. As soon as she was released, the child immediately showed signs of life. She was taken to an adjoining shop, and a few minutes later, she was found about to be given a drink. This the doctors forbade as they felt sure that rupture of viscera had occurred. The child was taken to hospital and after a day, made a complete and uneventful recovery.

(3) A man aged 83 years attempted suicide by smothering. He was certified to be dead and was taken to the mortuary. His relatives had come with wreaths. The undertakers also arrived, but were surprised to see the "dead man" seated on top of the coffin and complaining of hunger. He subsequently walked home unaided.

All postmortem changes are dependent on ambient temperature. High temperature accelerates the changes.

CHANGES IN THE SKIN: Skin becomes **pale and ashy-white and loses elasticity** within a few minutes of death. The lips appear dark-red to black, dry and hard due to drying.

CHANGES IN THE EYE: (1) Loss of Corneal Reflex: This is found in all cases of deep insensibility, e.g. epilepsy, narcotic poisoning, general anaesthesia and therefore not a reliable sign of death.

(2) Opacity of the Cornea: This may occur in certain diseases (cholera, wasting diseases) before death. The opacity is due to drying and is delayed for about two hours if the lids are closed after death. **If the eyelids are open for a few hours after death, a film of cell debris and mucus forms two yellow triangles on the sclera at each side of the iris, with base towards the margin of cornea and apex towards medial or lateral canthus of the eye, which become brown and then black called "tache noire" Figs. (7–1 and 7–2)** within 3 to 4 hours, upon which dust settles and the surface becomes wrinkled (artefact).

Fig. (7–1). Tache noire.

Fig. (7–2). Tache noire.

(3) Flaccidity of the Eyeball: The eyes look sunken and become softer within minutes due to reduction of intraocular tension. During life, the intraocular tension varies between 14 to 25 gm; soon after death it is less than 12 gm; within half an hour it is less than 3 gm., and becomes nil at the end of two hours.

(4) Pupils: **Soon after death, pupils are slightly dilated,** because of the relaxation of muscles of the iris. Later, they are constricted with the onset of rigor mortis of the constrictor muscles and evaporation of fluid. As such, their state after death is not an indication of their antemortem appearance. **Occasionally, rigor mortis may affect ciliary muscles of iris unequally, so that one pupil is larger than the other.** If different segments of the same iris are unequally affected, the pupil may be irregularly oval or have an eccentric position in the iris. **The pupils react to atropine and eserine for about an hour after death, but they do not react to strong light.** The shape of the pupil cannot be changed by pressure during life, but after death, if pressure is applied by fingers on two or more sides of the eyeball, the pupil may become oval, triangular or polygonal.

(5) Retinal Vessels: **Fragmentation or segmentation (trucking or shunting) of the blood columns (Kevorkian sign) in the retinal vessels appear within minutes after death, and persists for about an hour.** This occurs all over the body due to loss of blood pressure but **it can be seen only in retina by ophthalmoscope.** The retina is pale for the first two hours. At about six hours, the disk outline is hazy and becomes blurred in 7 to 10 hours. These changes are of little practical value.

(6) Chemical Changes: **A steady rise in the potassium values occur in the vitreous humour after death up to 100 hours.**

COOLING OF THE BODY (ALGOR MORTIS)

Loss of Body Heat: The cooling of the body ('chill of death') after death is a complex process, which **does not occur at the same rate throughout the body.** The body will not cool according to Newton's law. After stoppage of circulation, convectional transport of heat inside the body stops. **Heat is generated by residual metabolic process (glycogenolysis) of dying tissues and by metabolic activity of intestinal bacteria, due to which body temperature does not fall for some time.** With the start of cooling, a temperature gradient develops from the surface to the core of any part of the body. **Exchange of heat between the core and surface of the body occurs only by conduction. At first heat is lost from superficial layers of the body only.** Due to the low velocity of heat transport inside the body, it takes some time for heat to be conducted from the deeper layers to the more superficial layers, until finally, the temperature gradient reaches the core. **Internal organs cool primarily**

by conduction. Conductive heat exchange occurs due to the temperature difference between the body and surroundings, e.g. clothing, covering, air, water, etc. **The body heat is mostly lost by conduction (absorption of heat by objects in contact with the body) and convection (movement of air).** At non-contact areas heat exchange occurs by convectional mechanism, which exceeds that of contact surface. **Heat exchange by radiation (loss in the form of infrared heat rays) is extensive for the first hour, but decreases later,** depending on the rapid decrease in skin temperature. Only a small fraction of heat is lost by evaporation of fluid from the skin. **For about half to one hour after death, the rectal temperature falls little or not at all. (postmortem temperature plateau; isothermic phase) Fig. (7–3). This is followed by a linear rate of cooling (between 0.4 to 0.6°C per hour) for the next 12 to 16 hours.** Then the cooling rate is relatively uniform in its slope. Then it gradually becomes slower, and when the temperature is within about 4°C of the environment, rate of cooling becomes very slow. The human body rarely reaches the ambient (atmospheric) temperature unless the latter is at or near freezing, probably because enzyme and bacterial action starts during early decomposition. **The curve of cooling is sigmoid or inverted 'S' shaped in pattern,** because of some residual enzymatic activity and due to retention of heat for some time. In serious illness, circulation begins to fail before death, and hands and feet become cooler than the rest of the body; this coolness gradually extends towards the trunk. In sudden death, the cooling starts after death.

Measurement of temperature: A laboratory thermometer 25 cm. long, with a range of 0 to 50°C. which can be read in single degrees is used. **The rectum is the ideal place to record temperature except in cases of sodomy.** The thermometer should be inserted 8 to 10 cm. and left there for two minutes. The temperature can also be recorded by making a small opening into the peritoneal cavity and inserting the thermometer in contact with the inferior surface of the liver. **The external auditory meatus or the nasal passages also can be used** to record temperature. Where the nose is used, the probe or bulb should be passed up to the cribriform plate, and in the ear, should be placed on or through the tympanic membrane. A small electronic thermocouple with a digital readout is better. The time of this reading is recorded and temperature of environment is recorded at the same time. **Reading should be made at intervals of 1 to 2 hours, in order to obtain the rate of fall of temperature.**

A rough idea of approximate time in hours of death can be obtained by using the formula:

$$\frac{\textbf{Normal body temperature — rectal temperature}}{\textbf{Rate of temperature fall per hour}}$$

Variations in rectal temperature: The rectal temperature is between 36.5 to 37.5°C. Rectal temperature is about 0.6°C to 1°C higher than oral. **There are individual and daily variations up to 1 to 1.5°C. being lowest in the early morning and highest in the late evening. During sleep, the rectal temperature is 1/2° to 1°C. lower.** It cannot be assumed that the body temperature is normal at death. In cases of fat or air embolism, certain infections, septicaemia, heatstroke and in pontine haemorrhage, thyrotoxicosis, psychotic (emotional) stress, administration of neuroleptic medication, CO poisoning, intoxication with heroin and cocaine, drug reactions, etc. a sharp rise in temperature occurs. Exercise or struggle prior to death may raise the rectal temperature up to 1.5° to 2°C. Low temperature occurs in cases of collapse, congestive cardiac failure, hypothermia, hypothyroidism, administration of muscle relaxants, cholera, secondary shock, etc.

Factors Affecting Rate of Cooling: (1) The difference in temperature between the body and the medium: The temperature fall is rapid when the difference between body and air temperature is great. In India, during summer, the temperature of the environment may be higher than that of the body temperature, and as such the cooling is very slow. **In tropical climates the heat loss is roughly 0.4°C to 0.6°C and in temperate countries 1°C per hour. (2) The build of the cadaver**: The rate of heat loss is proportional to the weight of the body to its surface area. Thus, children and old people cool more rapidly than adults. **(3) The physique of the cadaver**: Fat is a bad conductor of heat. Fat bodies cool slowly and lean bodies rapidly. **(4) The environment of the body**: A body kept in a well-ventilated room will cool more rapidly than one in a closed room. Moist air is a better conductor of heat than dry air, so that cooling is more rapid in humid atmosphere than in dry atmosphere. Body heat is lost three times faster in water than in dry, cold area of some temperature because, water is a far better conductor of heat. **Cooling in still water is about twice as fast as in air, and in flowing water, it is about three times as fast.** Bodies cool more slowly in water containing sewage effluent or other putrefying organic material than in fresh water or sea water. **(5) Covering on or around the body:** The rate of cooling is slow when the body is clothed, as clothes are bad conductors of heat. A breadspread covering may at least halve the rate of cooling.

Because of the above external variable factors, an accurate formula cannot be devised to define rate of heat loss. The rectal temperature of an average-sized naked body reaches that of environment in about 15 to 20 hours. **If the body is exposed to a source of heat for a few hours shortly after death, its temperature will rise. A body in zero weather may undergo freezing and become stony-hard from formation of ice in**

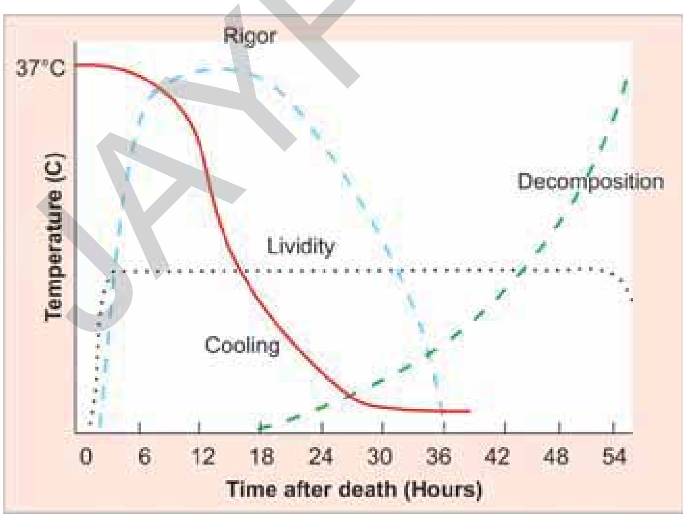

Fig. (7–3). Chart showing the major changes to estimate time since death.

cavities and blood vessels. The ice inside the skull may expand and cause separation of sutures.

Medicolegal Importance: Determination of temperature of the body is important only in cold and temperate climates, where more people die indoors. They are often useless in warm or tropical climate zones and outdoor deaths. In tropical zones, the postmortem fall in temperature may be minimal. It helps in the estimation of the time of death, which is not reliable.

Postmortem Caloricity: In this condition, the **temperature of the body remains raised for the first two hours or so after death. This occurs:** (1) when the regulation of heat production has been severely disturbed before death, as in sunstroke and in some nervous disorders, (2) when there has been a great increase in heat production in the muscles due to convulsions, as in tetanus and strychnine poisoning, etc., and (3) when there has been excessive bacterial activity, as in septicaemic condition, cholera and other fevers.

POSTMORTEM HYPOSTASIS

This is the bluish-purple or purplish-red (due to deoxyhaemoglobin) discolouration, which appears under the skin in the most superficial layers of the dermis (rete mucosum) of the dependent parts of the body after death, due to capillo-venous distention. It is also called **postmortem staining, subcutaneous hypostasis, livor mortis,** cadaveric lividity, suggilations, vibices and darkening of death.

Mechanism: **It is caused by the stoppage of circulation, the stagnation of blood in blood vessels, and its tendency to sink by force of gravity.** The blood tends to accumulate in the toneless capillaries and venules of the dependent parts of the body. Filling of these vessels produces a bluish-purple colour to the adjacent skin. The heavier red cells have a tendency to settle first imparting a deeper colour to the affected parts. **The upper portions of the body drained of blood are pale. The intensity of the colour depends upon the amount of reduced haemoglobin in the blood.** In cases of large amount of reduced haemoglobin before death, the blood has deep purplish-red colour. **The colour of the hypostasis may vary from area to area in the same body.**

In the recently dead or dying tissues, oxygen dissociation takes place, which is continued until equilibrium is reached between the tension of the oxygen in the capillaries and the surrounding tissues. There may also be backward flow of venous blood from the venular end of the capillaries, which adds to the blueness of the blood after death. **It is not possible to distinguish the postmortem discolouration from that produced by cyanosis in the living.**

Development: Postmortem lividity **begins shortly after death, but it may not be visible for about half to one hour after death in normal individuals, and from about one to four hours in anaemic persons. Dull-red patches of 1 to 2 cm. diameter appear in 20 to 30 minutes to two hours. These patches then deepen, increase in intensity and become confluent in 1 to 4 hours. In the early stage these patches can be mistaken for bruises** Table (7–1). In doubtful cases, a portion should be removed for microscopic examination.

Christison refers to two cases, in one of which two persons were convicted, and in the other three narrowly escaped conviction, upon a mistake of this kind.

Table (7–1). Hypostasis related to the time of death [hours post-mortem (hpm)]

Stage	Lower limit	Upper limit
Beginning	0.25	3
Confluence	1.0	4
Maximum	3.0	16
Thumb pressure	1.0	20
Complete shifting	2.0	6
Incomplete shifting	4.0	24

Computed by Mallach from literature data.

The areas then enlarge and combine to produce extensive discolouration. In some bodies, isolated patches of lividity remain separate from the large areas of lividity resembling contusions. Frost erythema, hypothermia induced red-purple spots seen over prominent parts of the body such as shoulder, knee or elbow joints may sometimes be mistaken for hypostatic patches. **Circulatory stasis in the aged, and sometimes the effect of cold, may resemble the effects of violence.** Such marks are usually found on ears, shins, forearms and hands, where the circulation is comparatively poor and the skin is exposed. **When lividity first develops, if the end of the finger is firmly pressed against the skin and held for a second or two, the lividity at that part will disappear and the skin will be pale or white. When the pressure is released the lividity will reappear.** The plasma tends to cause oedema of the dependent parts and contributes to the cutaneous blisters of early putrefaction. In the early stages mottled patches of hypostasis may be seen on the upper surfaces of the body, especially the legs and thighs due to uneven dilatation of the vascular bed. These patches soon join together and slide down to the dependent parts. **It is usually well developed within four hours and reaches a maximum between 6 to 12 hours (primary lividity) and persists until putrefaction sets in** Fig. (7–3). It is present in all bodies, but is more clearly seen in bodies of fair people than in those of dark. It may not be appreciated in old and anaemic persons.

Shifting of lividity: **If the body is moved within few hours after death, patches of lividity will disappear and new ones will form on dependent parts, (secondary lividity),** but lividity to a lighter degree remains in the original area, due to staining of the tissues by haemolysis **(complete shifting)**. This may take from a few minutes to up to one hour. In **incomplete shifting,** after turning the body over, lividity appears slightly in the downward facing parts.

Intensity: **It is intense in asphyxia, where the blood may not readily coagulate, and in cases of sudden death with a short agonal period and a great circulating blood volume. It is less marked in death from haemorrhage, anaemia and wasting diseases due to reduced amount of blood and pigment.** It is less marked in death from lobar pneumonia, and other conditions in which the blood coagulates quickly. **Sometimes, bluish hypostasis becomes pink along the upper part of the horizontal margin, the lower parts remaining dark.** This is due to the haemoglobin being oxygenated where the erythrocytes are less densely packed in the upper layers of hypostasis.

The extent of lividity. It mainly depends upon: (1) the volume of blood in circulation at the time of death, and (2) the length of time that the blood remains fluid after death. **Hypostatic congestion resembling postmortem hypostasis may be seen a few hours before death in case of a person dying slowly with circulatory failure,** e.g. cholera, typhus, tuberculosis, uraemia, morphine and barbiturate poisoning, congestive cardiac failure, deep coma, and asphyxia. In such cases, hypostasis will be marked shortly after death. Lividity can be marked shortly after death in a person dying slowly from circulatory failure.

Petechial haemorrhages: Numerous coarse or fine petechial haemorrhages may be present in hypostatic areas in deaths during an anoxic state. They are very common in narcotic poisoning and in sudden collapse due to acute cardiac arrest. **Petechiae may be seen in hypostatic area in 18 to 24 hours due to rupture of small vessels.**

The distribution of P.M. hypostasis: The distribution of the stain depends on the position of the body. (1) **In a body lying on its back** (supine) it first appears in the neck, and then spreads over the entire back extending up the flanks and sides of the neck, with the exception of the parts directly pressed on, i.e. occipital scalp, shoulderblades, buttocks, posterior aspects of thighs, calves and heels. **Any pressure prevents the capillaries from filling, such as the collar band, waist band, belts, wrinkles in the clothes, etc. and such areas remain free from colour and are seen as strips or bands called vibices**. This is also caused by pressure of one area of the body with another; in which case "mirror image" blanching may be seen **(contact pallor, contact blanching)**. **Such pale areas should not be mistaken for marks due to beating, or when they are present on the neck, due to strangling.** Hypostasis is usually well-marked in the lobes of the ears and in the tissues under nails of the fingers. **In asphyxial deaths, the nail beds will retain the cyanotic colour present, when the body was first refrigerated. As the vessel walls become permeable due to decomposition, blood leaks through them and stains the tissues. At this stage, hypostasis does not disappear,** if finger is firmly pressed against the skin. The pattern of lividity may be modified by local changes in the position of the body, e.g., if the head is turned to one side and slightly flexed on the neck for some hours after death blood may gravitate into a linear distribution determined by the folds formed in the skin and subcutaneous tissues. If such a body is examined after the neck has been straightened, the linear discolouration of the stains may be mistaken for marks due to beating. (2) **If the body is lying in prone position,** the lividity appears in the loose connective tissues in front, the colour is intense and Tardieu spots are common. **Sometimes, the congestion is so great that minute blood vessels are ruptured in the nose, and cause bleeding Fig. (7–4).** In persons who die in prone position, petechiae, ecchymoses and cutaneous blood blisters may develop after death, in areas of deep hypostasis especially in the shoulders or over the chest, which may be mistaken for asphyxial death. They are more common in cyanotic, congestive types of death and become more prominent as postmortem interval lengthens. (3) **If the body has been lying on one side,** the blood will settle on that side. (4) **If the body is left with the head downwards,** the confluent petechiae and ecchymoses may be so marked that they virtually blacken the face and neck. In sudden infant death syndrome and in drunken persons and epileptics who die face down on a pillow or other surface, pale areas are often seen on the face around the nose and mouth due to pressure against the supporting surface. This should not be mistaken for suffocation. (5) **If the body is inverted as in drunken persons who slide out of bed, hypostasis will appear in the head and neck. The eyes may suffuse, and numerous haemorrhages may appear in the conjunctivae and hypostatic areas.** This may give rise to suspicion of suffocation or strangulation. (6) If the body has been suspended in the vertical position as in hanging, hypostasis will be most marked in the legs, hands and external genitalia, and if suspension be prolonged for a few hours, petechial haemorrhages are seen in the skin. (7) **In drowning,** postmortem staining is usually found on the face, the upper part of chest, hands, lower arms, feet and the calves, as they are the dependent parts. If the body is constantly moving and changing its position, as after drowning in flowing water, the staining may not develop. (8) **Sometimes, blotchy areas of lividity appear on the upper surface of the limbs due to some irregularity of capillary dilatation at the time of death.** The internal jugular veins are markedly engorged due to the blood which has drained from the head. This blood cannot drain away below to the heart, as the valves in the subclavain veins prevent the drainage of blood into the upper limbs. As a result of this, the tributaries of the superficial veins in the neck cannot be effectively drained, due to which **isolated areas of lividity may develop on the front and sides of the neck resembling bruises. In certain cases, isolated patches of lividity remain separate from the large areas of lividity resembling contusions.**

Fig. (7–4). PM hypostasis on front of body due to body lying in prone position.

Fixation: It was thought that **fixation of p.m. hypostasis** was due to clotting of blood in blood vessels, but it is not correct. **The physical factors for fixation of p.m. staining are:** (1) Blood cannot pass out of the capillaries after formation of p.m. hypostasis. (2) Rigor mortis obliterates the big vessels, and as such the blood cannot pass through these vessels to settle in venules and capillaries in a new area. (3) After full development of rigor mortis, venules and capillaries are compressed and

Table (7–2). Difference between postmortem hypostasis and congestion

Trait	Hypostasis	Congestion
(1) Redness:	**Irregular and occurs on a dependent part.**	**Uniform all over the organ.**
(2) Mucous membranes:	**Dull and lusterless.**	**Normal.**
(3) Exudate:	**No inflammatory exudate.**	**Exudate may be seen.**
(4) Hollow viscus:	**Stomach and intestine when stretched show alternate stained and unstained areas according to the position of coils.**	**Uniform staining.**

almost empty and cannot be easily distended by the resettling blood. **Hypostasis becomes fixed when blood leaks into the surrounding soft tissues due to haemolysis and breakdown of blood vessels. This usually occurs in 6 to 12 hours or more,** but the condition of blood at the time of death exerts a considerable influence. Non-displacement and non-shifting of lividity is due to haemoconcentration by loss of fluid which penetrates the wall of those vessels related to the hydrostatic pressure. Fixation occurs earlier in summer and is delayed in asphyxial deaths and in intracranial lesions. **Some authors are of the opinion that hypostasis does not get fixed.**

Calorimetry shows an increasing paleness of the hypostasis between 3 to 5 hours, from a wavelength of 575 nm at 3 hours at an average rate of 2 nm per hour. Vanezis claims that there is a linear relationship between the fading colour of the hypostasis, and time during the first 24 hours, after which the relationship in unpredictable.

Haemorrhages: Tiny, often spot-like, sometimes confluent oval to round, bluish-black haemorrhages **(death spots, postmortem ecchymoses)**, are exclusively limited to areas of hypostasis as a result of mechanical rupture of subcutaneous capillaries and venules. **They are seen commonly in the back of the shoulders and neck, and sometimes on the front of the chest, even when the body is lying on its back.** They are common in cyanotic congestive types of death, and appear more prominent with the increase in postmortem interval, and may blacken the face and skin. They are more prominent when the body lies with the head downwards.

Absence of hypostasis: Hypostasis may be sparse or even **absent in deaths where considerable blood loss of at least 65% of the circulating blood volume in adults and 45% in infants, occurs before death due to trauma. In severe anaemia lividity will be absent.**

Internal Hypostasis: Hypostasis also occurs in the dependent parts of internal organs Table (7–2). When a body is in supine position, hypostasis is seen in the posterior portions of the cerebrum and cerebellum, the dorsal portions of the lungs, posterior wall of the stomach, dorsal portions of the liver, kidneys, spleen, larynx, heart, and the lowermost coils of intestine in the pelvic cavity. **Hypostasis in the heart can simulate myocardial infarction, and in the lungs it may suggest pneumonia; dependent coils of intestine appear strangulated.**

Colour changes: In asphyxia, the colour of the stains is deeply bluish-violet or purple. A brownish hypostasis may be seen in methaemoglobinaemia and rarely a bronze colour in Clostridium perfringens septicaemia usually associated with septic abortion. In septic abortion from Cl. welchii, greenish-brown colour is seen. A bright pink colour is seen in hypothermia and bodies taken from cold water, and in refrigerated bodies as the wet skin allows atmospheric oxygen to pass through, and also at low temperatures haemoglobin has a greater affinity for oxygen. This may be most marked over large joints and dependent areas. **Refrigerated bodies may also assume a pink colour.** In mummification, lividity may turn from brown to black with drying of the body.

Infection combined with disseminated intravascular coagulation sometimes causes blotchy purplish, red or pink rashes which may be mistaken for bruises or abrasions.

Colour Changes in Poisoning: The hypostatic areas have distinct colour in certain cases of poisoning, e.g. (1) In carbon monoxide poisoning, the colour is cherry-red Fig. (7–5). (2) In hydrocyanic acid poisoning and sometimes in burns the colour is bright-red. (3) In poisoning by nitrites, potassium chlorate, potassium bicarbonate, nitrobenzene, acetanilide, bromates, and aniline (causing methaemoglobinaemia) the colour is chocolate or brownish-red Fig. (7–6). (4) In poisoning by phosphorus the colour is dark-brown or yellow. (5) In poisoning by hydrogen sulphide, the colour is bluish-green. (6) In CO_2 poisoning, the colour is deep-blue.

Colour Changes in Decomposition: Changes in postmortem lividity occur when putrefaction sets in. In early stages, there is haemolysis of blood and diffusion of blood pigment into the surrounding tissues, where it may undergo secondary changes, e.g., sulphhaemoglobin formation. The capillary endothelium and the surrounding cells show lytic changes. Microscopically, the cellular outlines are obscured and the capillaries are not identifiable.

Fig. (7–5). Cherry-red PM hypostasis in CO poisoning.

Table (7–4). Difference between rigor mortis and cadaveric spasm

Trait	Rigor mortis	Cadaveric spasm
(1) Production:	Freezing and exposure to temperature above 65°C will produce rigor.	Cannot be produced by any method after death.
(2) Mechanism:	Known. Reduction of ATP.	Not clearly known.
(3) Predisposing factors:	Nil.	Sudden death, excitement, fear, exhaustion, nervous tension, etc.
(4) Time of onset:	1–2 hours after death.	Instantaneous.
(5) Muscles involved:	All the muscles of the body, both voluntary and involuntary.	Usually restricted to a single group of voluntary muscles.
(6) Muscle stiffening:	Not marked; moderate force can overcome it.	Marked; very great force is required to overcome it.
(7) Molecular death:	Occurs.	Does not occur.
(8) Body heat:	Cold.	Warm.
(9) Electrical stimuli:	Muscles do not respond.	Muscles respond.
(10) Muscular reaction:	Acidic.	Alkaline.
(11) Medicolegal importance:	Indicates time of death.	Indicates mode of death, i.e. suicide, homicide or accident.

system, firearm wound of the head, drowning, convulsant poisons, such as strychnine, etc. **No other condition simulates cadaveric spasm and it cannot be produced by any method, after death.** Very great force is required to overcome stiffness. It passes without interruption into normal rigor mortis and disappears when rigor disappears. **Coagulation of protein is seen in burns on microscopic examination but not in cadaveric spasm.**

Medicolegal importance. (1) Occasionally, in case of suicide, the weapon, e.g., pistol or knife is seen firmly grasped in the victim's hand which is a strong presumptive evidence of suicide. Attempts may be made to simulate this condition in order to conceal murder. But, ordinary rigor does not produce the same firm grip of a weapon, and the weapon may be placed in the hand in a way which could not have been used by a suicide. (2) If the deceased dies due to assault, some part of clothing, e.g., button of his assailant or some hair may be firmly grasped in the hands. (3) In case of drowning, material such as grass, weeds or leaves may be found firmly grasped in the hands, which indicates that the victim was alive on entering the water.

CASE: (1) Tidy (1882) mentioned the soldier at Balaclava whose body remained in position on his horse for some time after he had been killed by a shell.

(2) Tidy also cited the incident at Sedan, described by Rossbach (1870), when six soldiers were killed by a shell. The head of one preserved his laughing expression, present at the moment of death. The body of another, who also had his head blown off, remained in a sitting posture, with a cup still in his hand.

(3) Tidy (1882) described two lovers who, after taking cyanide were found folded in each other's arms, their bodies stiffened in this position.

(4) Spilsbury (1944) recorded the case of a woman found dead, seated upright in her bath; she held a sponge in her hand which was raised half way to her face. Death was due to cerebral haemorrhage.

Secondary Relaxation: Flaccidity following rigor mortis is caused by the action of the alkaline liquids produced by putrefaction. Another view is that rigidity disappears due to solution of myosin by excess of acid produced during rigor mortis. A third view is that enzymes are developed in dead muscle which dissolve myosin by a process of autodigestion.

DECOMPOSITION

It involves two processes: (1) Autolysis and (2) Putrefaction.

AUTOLYSIS: Autolysis is self-digestion of tissues. Soon after death, cell membranes become permeable and breakdown, with release of cytoplasm containing enzymes. Lysosomes and their digestive enzymes (mainly hydrolases) are released from the cells. The proteolytic, glycolytic and lipolytic action of ferments cause autodigestion and disintegration of organs, which occurs without bacterial influence. This chemical process is increased by heat and is stopped by freezing or inactivation of enzymes by the heat. The earliest autolytic changes occur in parenchymatous and glandular tissues and in the brain. The lining of intestines, adrenal medulla and pancreas autolyse within hours of death. Autolytic fermentation results in maceration of the dead foetus in utero, early softening and liquefaction of the brain of the newborn and infants, and softening of the internal organs. Autodigestion by acid gastric juice is a common finding in the newborn and infants, and is seen as softening and rupture of the stomach and lower oesophagus. In adults, such digestion may start before death in cases of intracranial lesions. The earliest external sign is a whitish, cloudy appearance in the cornea.

PUTREFACTION: It is the final stage following death, in which destruction of the soft tissues of the body occurs. The terms decomposition and putrefaction are used as synonyms. Putrefaction usually follows the disappearance of rigor mortis. During hot season, it may commence before rigor mortis has completely disappeared from the lower extremities.

Mechanism: Organisms enter the tissues shortly after death, mainly from the alimentary canal, and less often through the respiratory tract or through an external skin wound. Multiplication of bacteria begins within 4 hours and peak is reached within 24–30 hours. The intestines contain more than one thousand different species of bacteria. The fall in the oxygen concentration in the tissues and rise in hydrogen ion concentration after death favour bacterial growth and spread throughout the body. **Because the protective agencies of the body are absent, the bacteria spread through the blood vessels using the proteins and carbohydrates of the blood as culture media. Destruction is caused mainly by the action of bacterial enzymes, mostly anaerobic organisms derived from the intestines.** Other enzymes are derived from fungi, such as Penicillium and Aspergillus and sometimes from insects, which may be mature or in larval stage. **The chief destructive bacterial agent is Cl. welchii, which causes marked haemolysis, liquefaction of postmortem clots and of fresh thrombi and emboli, disintegration of tissue and gas formation in blood vessels and tissue spaces.** Bacteria produce a large variety of enzymes and these breakdown the various tissues of the body. **Lecithinase produced by Cl. welchii is most important.** This hydrolyses the lecithin which is present in all cell membranes including blood cells, and is responsible for the postmortem haemolysis of blood. The other organisms include Streptococci, Staphylococci, bacteroids, anaerobic lactobacilli, diphtheroids, B. proteus, B. coli., B. aerogenes capsulatus, etc. Streptococci and Staphylococci multiply 10 to 100 times and even more in blood and tissues of corpses kept at room temperatures. As body temperature falls, multiplication of bacteria is slowed and below 20°C multiplication is almost completely stopped, though most enzymes produced by bacteria will continue to act at much lower temperatures. Any factor which delays cooling of body will hasten putrefaction process. It begins immediately after death at the cellular level, which is not evident grossly. **Lipolytic enzymes are less active, but hydrolytic breakdown starts early and goes on steadily until little neutral fat remains.** There is progressive breakdown of soft tissues and the alteration of their proteins, carbohydrates and fats.

Features: The characteristic features of putrefaction are: (1) changes in the colour of the tissues, (2) the evolution of gases in the tissues, and (3) the liquefaction of tissues. The same changes seen on surface of the body occur simultaneously in internal organs.

The exact chronological order of the appearance of putrefactive changes is highly variable and depends on a broad variety of individual as well as environmental conditions.

(1) **Colour Changes: External: Bacteria spread directly from the bowel into the tissues of the abdominal wall. At an early stage of putrefaction, haemoglobin diffuses through the vessels and stains the surrounding tissues a red or reddish-brown colour Figs. (7–8 to 7–13).** In tissues, various derivatives of haemoglobin are formed including sulphur-containing compounds, and the colour of the tissues gradually changes to a greenish-black. **The first external sign of putrefaction in a body lying in air is usually a greenish discolouration of the skin over the region of the caecum Fig. (7–8)** which lies fairly superficially, and where the contents of the bowel are more fluid and full of bacteria**. Internally, this is seen on the undersurface of the liver, anterior peritoneal surface of right lobe of liver and adipose tissue around gallbladder,** where that organ is in contact with the hepatic flexure and transverse colon. **The colour results from the conversion of haemoglobin of blood into sulphmethaemoglobin by the hydrogen sulphide** formed in the large intestine and escaping into the surrounding tissues. **The colour appears in 12 to 18 hours in summer and in one to two days in winter.** Green colouration is more clearly seen on a fair skin than on a dark one. The green colouration then spreads over the entire abdomen, external genitals and then **patches appear successively on the chest, neck, face, arms and legs.** The patches become dark-green and later purple and dark-blue. They are at first scattered, but later on join together and the whole skin of the body appears discoloured. The putrefactive bacteria spread most easily in fluid and tend to colonise the venous system. Wrinkling of the fingertips occurs early which become leathery, and the nails become prominent.

Marbling of Skin: The superficial veins especially over the roots of the limb, thighs, sides of the abdomen, shoulders, chest and neck are stained greenish-brown or purplish-red depending on the total amount of sulphhaemoglobin formation within the affected vessels (linear branching pattern) due to the haemolysis of red cells, which stains the wall of the vessel and infiltrates into the tissue, giving a **marbled appearance (red, then greenish pattern in skin resembling the branches of a tree). This starts in 24 hours, but is prominent in 36 to 48 hours Fig. (7–9).** The clotted **blood becomes** fluid, and as such, the position

Fig. (7–8). Greenish discoluoration of right iliac fossa.

Fig. (7–9). Putrefactive network (Marbling).

Fig. (7–10). **Advanced decomposition. Marbling, bloating.**

Fig. (7–11). **Blisters in puterfaction.**

Fig. (7–12). **Gaseous distension of body due to decomposition.**

Fig. (7–13). **Blackening of body and maggots in advanced.**

of the postmortem staining is altered, and the fluid blood collects in the serous cavities. **Putrefactive effusion of foul-smelling bloodstained fluid** into the pleural cavities usually starts at about the time when the skin becomes macerated Fig. (7–10). Such effusions usually do not exceed 60 to 100 ml. unless death resulted from drowning, when several hundred ml. of drowning medium which oozed out through the lungs and visceral pleura, may be present in the thoracic cavities. **The reddish-green colour of the skin may become dark-green or almost black in 3 to 4 days.**

Internal: **The earliest internal change is a reddish-brown discolouration of the inner surfaces of the vessels, especially of the aorta.** Internally, decomposition proceeds more slowly than the surface. **The same changes of colour are seen in the viscera, but the colour varies from dark-red to black, rather than green.** With this colour change, **the viscera become softer and greasy to touch. Finally, they breakdown into a soft disintegrating mass.**

(2) Development of Foul-smelling Gases: The chemical processes in this stage are those of reduction, the **complicated proteins and carbohydrates being split into simpler compounds** of aminoacids, ammonia, CO, CO_2, hydrogen sulphide, phosphorated hydrogen, methane and mercaptans. The gases are non-inflammable in the early stages, but as the decomposition progresses, enough of hydrogen sulphide is formed, which can be ignited to burn with a blue flame. **Gases collect in the intestines in 12 to 18 hours in summer, and 1 to 2 days in winter and the abdomen becomes tense and distended.** At about the same time, the eyeballs become soft, the cornea becomes white and flattened or compressed. Later, the eyes collapse. **The gas formation in the blood vessels may force bloodstained fluid, air or liquid fat between the epidermis and dermis forming small blisters in 18 to 24 hours** Fig. (7–11). Blisters are formed first on the lower surfaces of trunk and thighs, where tissues contain more fluid due to hypostatic oedema.

Gas bubbles accumulate in the tissues, causing crepitant, sponge-like feeling which soon begins to distend the body. **From 18 to 36 hours after death, the gases collect in the tissues, cavities and hollow viscera under considerable pressure, and the features become bloated and distorted** Fig. (7–7). On opening the abdomen, the gas escapes with a loud explosive noise. Discoloured natural fluids and liquefied tissues are made frothy by gas.

Pressure effects of putrefactive gases: **Due to the presence of gas in the abdomen, the diaphragm is forced upwards**

Fig. (7–14). Advanced decomposition showing bulging eyes and protruding rongue.

Fig. (7–16). Maggots over the face in advanced putrefaction.

Fig. (7–15). Prolapse of rectum in highly decomposed body.

Fig. (7–17). Liquefaction of eyeball in decomposition.

become greasy and softened. The softer the organ, the more blood it contains, and the nearer to the sources of bacteria, the more rapidly it putrefies. The lining of the intestine, adrenal medulla and pancreas autolyse within hours of death. The capsules of the liver, spleen and kidney resist putrefaction longer than the parenchymatous tissues, which are usually converted into bag-like structures filled with thick, turbid fluid material. The organs composed of muscular tissues and those containing large amount of fibrous tissue resist putrefaction longer than the parenchymatous organs, with the exception of the stomach and intestine, which because of the contents at the time of death, decompose rapidly.

The various organs putrefy at different rates, depending on their structure, vascularity and access of air and bacteria Figs. (7–14, 7–15 and 7–17). As a general rule, the organs show putrefactive changes in the following order. (1) Larynx and trachea. (2) Stomach, intestines, pancreas and spleen. (3) Liver, lungs. (4) Brain. (5) Heart. (6) Kidneys, bladder. (7) Prostate, uterus. (8) Skin, muscle, tendon. (9) Bones.

LARYNX AND TRACHEA: At first the mucous membrane becomes brownish-red and later greenish, and is softened in 12 to 24 hours in summer and 2 to 3 days in winter.

STOMACH AND INTESTINES: They putrefy in 24 to 36 hours in summer, and 3 to 5 days in winter. Dark-red irregular patches involving the whole thickness of the wall are first seen on the posterior wall and then on the anterior wall. Gas blebs are formed in the submucous layer which project as small multilocular cysts of varying size into the lumen. They become softened, dark-brown and change into dark, soft, pulpy mass. The mucosa appears macerated and can be easily peeled off. The mucosa becomes brown by diffusion of blood into tissues with subsequent alteration of the haemoglobin. Other breakdown products reacting with sulphur may stain the mucosa green or black. The stomach may show perforation.

In the intestines, when there is much blood or bile pigment in the lumen, it passes into the wall and stains it. Various breakdown products of protein with sulphur produce green or black mucosa.

SPLEEN: It becomes soft, pulpy and liquefies in two to three days.

OMENTUM AND MESENTERY: They putrefy early if loaded with fat. They become greyish-green and dry in one to three days in summer.

LIVER: It becomes softened and flabby in 12 to 24 hours in summer. Multiple blisters appear in 24 to 36 hours. Cl. welchii form

drained of the blood, and intestinal organisms do not gain entry. The trunk putrefies rapidly because of the action of intestinal bacteria, and the access of airborne organisms.

Because of the above variable factors, it is not of much use to attempt to construct a timetable for the stages of decomposition.

In advanced putrefaction, no opinion can be given as to the cause of death, except in cases of poisoning, fractures, firearm injuries, etc.

ODOUR MORTIS: About 50 volatile chemical compounds were identitied as being associated with human remains which are unique to the decomposition of human remains. Field portable analytical instruments can be used to locate human remains in shallow burial sites.

PUTREFACTION IN WATER: Casper dictum states that **a body decomposes in air twice as rapidly as in water, and eight times as rapidly as in earth. The variations are very real, and it is not of much practical value. The rate of putrefaction is slower in water than in air. Putrefaction is more rapid in warm, fresh water than in cold, salt water. It is more rapid in stagnant water than in running water. Putrefaction is delayed when a body is lying in deep water and is well protected by clothing, while it is rapid in a body lying in water contaminated with sewage. As the submerged cadavers float face down with the head lower than the trunk, gaseous distension and postmortem discolouration are first seen on the face and then spread to the neck, upper extremities, chest, abdomen and the lower extremities in that order. Fluid gravitation in the head favours marked decomposition. When the body is removed from the water, putrefaction is hastened as the tissues have absorbed much water. The epidermis of the hands and feet becomes swollen, bleached and wrinkled after immersion, and may be removed as a cast of the extremity, after 2 to 4 days. After several weeks in water, macerated flesh may be stripped off from the body by the action of currents or the contact with the floating objects. Fish, crustacea (crabs, lobsters, shrimps, etc.) and water-rats in a sewer may destroy the body. Moulds may be located anywhere on the body, but generally are found only on the exposed surfaces. Fungus may grow on body which varies in colour from white and yellow to green and black.**

SKELETONISATION: The time required for skeletonisation varies considerably and mainly depends on the ambient temperature, insect colonisation of the body and scavenger activity. In a hot humid environment with heavy insect activity a corpse can be skeletonised within a few days. In the case of an exposed body, flies, maggots, ants, cockroaches, rats, dogs, jackals, vultures, etc., may reduce the body to a skeleton within a few days. When the body is in the water, it may be attacked by fishes, crabs, etc., which reduce the body to a skeleton in a few days. In an uncoffined body buried in a shallow grave, putrefaction is delayed to a moderate extent. In a deeply buried body, the lower temperature, the exclusion of air, absence of animal life, etc., markedly delay decomposition. The important factors are seasonal, climatic variation, the amount of soil water, the access of air, and the acidity or otherwise of the soil water. In bodies placed in airtight coffins, decay process may not occur for several decades, but in a poor coffin which admits air and water, bodies will decompose quickly. In India, an uncoffined buried body is reduced to a skeleton within about 1 to 2 years. Disarticulation often occurs from the head downwards and from central to peripheral. Articulated bones are seen up to 3 weeks. In about 5 weeks some are articulated. In about one year complete disarticulation occurs and most of small bones are missing. Some bones are broken in 2 to 4 years. Bone decay occurs after 10 to 12 years. Buried bones may decay at different rates, e.g. neutral soil may not destroy the skeleton at all. Acidic soil may cause decay in about 25 to 100 years. In a hot climate, bones on the ground surface may decay in 5 to 10 years. In tropical countries, weathering of the skull may occur in 5 to 6 years. The outer table disappears irregularly, followed by inner table. The protein content of the bones decomposes. As the bones contain largely inorganic material, they will crumble, rather than decompose. Flat bones and the bones of the infants and old, breakdown faster.

ADIPOCERE (Saponification)

Adipocere (cire = wax) is a modification of putrefaction. It is the formation of soft, whitish, crumbly, waxy and greasy material, occurring in fatty or fat containing tissues of a dead body **Figs. (7–19 and 7–20)**. In this, **the fatty tissues of the body change into a substance known as adipocere.** It is seen most commonly in bodies immersed in water or in damp, warm environment.

Mechanism: The change is due to the gradual hydrolysis and hydrogenation of pre-existing fats, such as olein, into higher fatty acids, which combine with calcium and ammonium ions to form insoluble soaps, which being acidic, inhibit putrefactive bacteria. Ultimately, the whole of the fat is converted into palmitic, oleic, stearic and hydroxystearic acid, together with some glycerol, and a mixture of these substances forms adipocere. These form a matrix for remnants of tissue fibres, nerves and muscles. Crystals with radial markings can be found in adipocere. **At the time of death, body fat contains, about half percent of fatty acids, but in adipocere they rise to 20% within a month and over 70% in three months. The process starts under the influence of intrinsic lipases, and is continued by the bacterial enzymes of the clostridia group, mainly Cl. perfringens,** as the bacteria produce lecithinase,

Fig. (7–19). Early stage of adipocere formation.

Fig. (7–20). Adipocere.

Fig. (7–22). **Mummified foetus.**

dark-brown and black and become a single mass. Later, due to putrefaction and maggot activity, they may disappear. If a mummified body is not protected, it will break into fragments gradually, and reduced to skeleton, though tough, leathery shreds of skin tendons and ligaments may persist for many years, but if protected, it may be preserved for years. Mummified bodies may be attacked by insects especially moths and larvae of various flies which destroy the body. **A mummified body is practically odourless.**

Collagen, elastic tissues, cardiac and skeletal muscle, cartilage, and bone are usually demonstrable histologically in the mummification material.

Factors necessary for the production of mummification: (1) The absence of moisture in the air, and (2) the continuous action of dry or warmed air.

Mummification of newborn children may occur if they are left in a trunk, or a kitchen cupboard, where the atmosphere is warm and dry. **Marked dehydration before death favours the development of mummification. Mummification occurs in bodies buried in shallow graves in dry sandy soils, where evaporation of body fluids is very rapid** due to the hot dry winds in summer. Chronic arsenic or antimony poisoning is said to favour the process.

Differential decomposition: Occasionally, a **body which shows evidence of mummification in certain parts may show adipocere changes in others.** Thus, there may be found some adipocere in cheeks, abdomen and buttocks, and mummification of the arms and legs.

The time required for complete mummification of a body. It varies from three months to a year and is influenced by the size of the body, atmospheric conditions and the place of disposal.

Medicolegal Importance: It is the same as that of adipocere.

CONDITIONS PRESERVING THE BODY

(1) EMBALMING: Embalming is the treatment of the dead body with antiseptics and preservatives to prevent putrefaction and preserve the body. By this process proteins are coagulated, tissues are fixed, organs are bleached and hardened and blood is converted into a brownish mass. **Embalming produces a chemical stiffening similar to rigor mortis, and normal rigor does not develop. Embalming rigidity is permanent.** Decomposition is inhibited for many months, if the injection is made shortly after death, and if done several hours after death, the body will show mixture of bacterial decomposition and mummification, and will disintegrate in a few months.

Embalming alters the appearance of the body, tissues and organs, making it difficult to interpret any injury or disease. Embalming completely destroys cyanide, alcohol and many other substances. Determination of the presence of many of the alkaloids and organic poisons becomes very difficult. The fixation process makes it difficult to extract drugs. Blood grouping cannot be made out. Extraction of DNA becomes difficult. Thrombi and emboli will be dislocated and washed away.

A body weighing 70 kg will require a fluid equivalent of ten litres Table (7–5). About 10% of it will be lost through venous drainage, purging, etc. **To be very satisfactory, embalming should be done within six hours of death.**

Embalming is required when a body has to be transported to distant places either by road, rail or air. It assures that the body is not hazardous to public health. If ascites is present, the fluid should be removed. No objection certification should be taken from police for transportation of a dead body.

Vitreous humour, synovial fluid and bile are ideal samples for screening for toxicology. Muscle mass from psoas and gluteal region can also be used.

INJECTION METHODS: Arterial injection is forcing of fluid in an artery to reach the tissues through the arterioles and capillaries. Diffusion occurs into the cells and tissues for preservation at the capillary level. Femoral artery is located in the inguinal canal, midway between the anterior superior iliac spine and pubic tubercle. Femoral vein is located one cm. medial to the artery. Various instrumental procedures are used for injecting the embalming fluid.

(1) Hand/foot pump.

(2) Stirrup pump.

(3) BULB SYRINGE: This is a variety of manual pump, similar to Higginson's syringe. It consists of a bulb-type rubber syringe and rubber tubing at either end. Valves built into the bulb allow suction on one side and ejection on the other side, when the bulb is squeezed. The injection needle is attached to the delivery end, and the suction end dips into the fluid container.

Table (7–5). **A typical embalming fluid**

Ingredient	Proportion
Formalin (Preservative)	1.5 L
Methanol (Preservative)	500 mL
Phenol (Germicide)	50 mL
Thymol (Fungicide)	5 gm
Sodium borate (Buffer)	600 gm
Sodium citrate (Anticoagulant)	900 gm
Glycerin (Wetting agent)	600 mL
Sodium chloride (Controls pH)	800 gm
Eosin (1%, Cosmetic)	30 mL
Soluble wintergreen (Perfume)	90 mL
Water (Vehicle)	up to 10 L

***NB*: Sodium borate and sodium citrate should be dissolved in hot water and allowed to cool. Add rest of the components and dilute with water to make up 10 L. Allow to stand for a few hours and filter.**

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