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DEATH AND ITS CAUSES

Thanatology deals with death in all its aspects. Death is of two types: (1) somatic, systemic or clinical, and (2) molecular or cellular.

Somatic Death: It is the complete and irreversible stoppage of the circulation, respiration and brain functions, but there is no legal definition of death.

THE MOMENT OF DEATH: Historically (medically and legally), the concept of death was that of "heart and respiration death", i.e. stoppage of spontaneous heart and breathing functions. Heart-lung bypass machines, mechanical respirators, and other devices, however have changed this medically in favor of a new concept "brain death", that is, irreversible loss of Cerebral function.

Brain death is of three types: (1) Cortical or cerebral death with an intact brain stem. This produces a vegetative state in which respiration continues, but there is total loss of power of perception by the senses. This state of deep coma can be produced by cerebral hypoxia, toxic conditions or widespread brain injury. (2) Brain stem death, where the cerebrum may be intact, though cut off functionally by the stem lesion. The loss of the vital centers that control respiration, and of the ascending reticular activating system that sustains consciousness, cause the victim to be irreversibly comatose and incapable of spontaneous breathing. This can be produced by raised intracranial pressure, cerebral oedema, intracranial haemorrhage, etc.(3) Whole brain death (combination of 1 and 2).

Whole or part of the brain can be irreversibly damaged due to hypoxia, cardiac arrest, intracranial hemorrhage, poisoning and trauma to the brain. If the cortex alone is damaged, the patient passes into deep coma, but the brainstem will function to maintain spontaneous respiration. This is called "persistent vegetative state" and death may occur months or years later due to extension of cerebral damage or from intercurrent infection.

Molecular Death: It means the death of cells and tissues individually, which takes place usually one to two hours after the stoppage of the vital functions. Molecular death occurs piecemeal. Individual cells will live on their residual oxygen for a variable time
after the circulation has stopped, depending on the metabolic activity of the cell. The subsequent changes occur due to metabolic dysfunction and later from structural disintegration. Nervous tissues die rapidly, the vital centres of the brain in about five minutes, but the muscles live up to one to two hours.

Modes of Death: According to Bichat, there are three modes of death, depending on whether death begins in one or other of the three systems, irrespective of what the remote causes of death may be. These modes are: (1) Coma. (2) Syncope, and (3) Asphyxia.

**Asphyxia**

Asphyxia is a condition caused by interference with respiration, or due to lack of oxygen in respired air due to which the organs and tissues are deprived of oxygen (together with failure to eliminate CO2), causing unconsciousness or death. Nervous tissues are affected first by deficiency of oxygen and their functions are disturbed even by mild oxygen lack. The neurons of the cerebral cortex will die in 3 to 7 minutes of complete oxygen deprivation, and the other nerve cells remain alive for a little longer time. Irreversible cortical damage may occur if oxygenated blood fails to perfuse the brain up to 7 to 10 minutes. Subnormal oxygen in the blood supply to the brain causes rapid unconsciousness. In all forms of asphyxia, heart may continue to beat for several minutes after stoppage of respiration. The rule of thumb is: breathing stops within twenty seconds of cardiac arrest, and heart stops within twenty minutes of stopping of breathing.

Types and Causes: (1) Mechanical: In this the air-passages are blocked mechanically, (a) Closure of the external respiratory orifices, as by closing the nose and mouth with the hand or a cloth or by filling these openings with mud or other substance, as in smothering, (b) Closure of the air-passages by external pressure on the neck, as in hanging, strangulation, throttling, etc. (c) Closure of the air-passages by the impaction of foreign bodies in the larynx or pharynx as in choking, (d) Prevention of entry of air due to the air-passages being filled with fluid, as in drowning, (e) External compression of the chest and abdominal walls interfering with respiratory movements, as in traumatic asphyxia.
(2) Pathological: In this, the entry of oxygen to the lungs is prevented by disease of the upper respiratory tract or of the lungs, e.g., bronchitis. acute oedema of the glottis, laryngeal spasm, tumours and abscess.

(3) Toxic: Poisonous substances prevent the use of oxygen, (a) poisoning by CO. (b)

(4) Environmental

(5) Traumatic: (a) Pulmonary embolism from femoral vein thrombosis due to an injury to the lower limb, (b) Pulmonary fat embolism from fracture of long bones, (c) Pulmonary air embolism from an incised wound of internal jugular vein, (d) Bilateral pneumothorax from injuries to the chest wall or lungs.

(6) Postural asphyxia: This is seen when an unconscious or stuporous person, either from alcohol, drugs or disease, lies with the upper half of the body lower than the remainder. The inverted position allows the abdominal vise era to push up the diaphragm and this together with reduced respiratory effect can cause death. It may also result from forcible flexion of the neck on the chest. Positional asphyxia is always accidental and associated with alcohol or drug intoxication jatrogenic is associated with anaesthesia.

Pathology: When the neck is compressed, occlusion of jugular veins prevents venous drainage from the head, but the arterial supply continues through the carotid and vertebral arteries. When the air-passages are occluded, the impaired oxygenation in the lungs causes decrease in the oxygen content of arterial blood. Reduction in oxygen tension causes capillary dilation which is followed by stasis of blood in the dilated capillaries and in the venules, which produces capillo-venous engorgement. This blood stasis causes congestion of organs and venous return to the heart is diminished leading to anoxia, which causes capillary dilation and the vicious cycle goes on. Petechial haemorrhage rostral are caused due to raised venous pressure from impaired venous return and not to hypoxia of the vessel walls. Sometimes, large haemorrhages are seen on upper chest and back of the body. The haemorrhages may be found in the substance of the viscera, but they are readily seen in serous membranes, particularly in the visceral pleura and pericardium. A minimum of 15 to 30 seconds is required to produce congestion and petechiae. The increased
capillary and venous permeability causes transudation of fluid into tissues. If the hypoxia continues, oedema of the tissues develops. Oedema of the lungs is common, and is usually caused due to combination of hypoxia and raised pulmonary vessel pressure, and its presence indicates struggle for survival for a time. Oedema of the brain occurs due to back-pressure and hypoxia. Congestion and oedema are non-specific and result due to obstructed venous return. When the neck is compressed, the face, lips, and tongue become swollen and reddened; Cyanosis (bluish colour of the skin, mucous membranes and internal organs, especially lungs, liver, spleen, kidneys and meninges), follows congestion of the face as venous blood containing much reduced haemoglobin after perfusing the head and neck becomes more blue. If the air-way is blocked, the impaired oxygenation in the lungs causes decrease in the oxygen content of the arterial blood, which causes darkening of all organs and tissues and will increase the cyanosis of the face.

Signs and symptoms of asphyxia: The phases of asphyxiation are: (1) The respiratory rate and depth of excursions are increased, and cyanosis develops. The pulse is rapid and blood pressure rises. These signs occur due to decreasing oxygen saturation of the blood. (2) Respirations are labored with increased cyanosis and petechial haemorrhages. Lungs are oedematous and salivation occurs. The pulse becomes more rapid and bounding, and the blood pressure rises further. When the throat is constricted, the face, eyes and tongue become turgid and bulge. Consciousness is clouded. (3) Respiration becomes irregular and gasping. The person becomes unconscious and convulsions may occur. Capillaries rupture both in the skin and internally, and the saliva and bronchial fluid may be blood-stained. Vomit may regurgitate into the throat, and urine and faeces may be passed. The pulse becomes weak and irregular, blood pressure falls and death occurs. At any stage through this progression sudden death may occur due to cardiac arrest.

Asphyxial Stigmata: Asphyxia is not a pathological entity, and cannot be clearly recognised from morbid anatomical findings alone. The triad of (1) cyanosis, (2) facial, palpebral, bulbar, subpleural and subepicardial petechiae, (3) visceral congestion, are all due to raised venous pressure. They are merely consistent with, but not diagnostic of asphyxia from anoxic anoxia.
local indications of fatal obstructing trauma must be demonstrated to establish that death occurred from mechanical asphyxia. Parenchymatous degenerative changes develop in rapid hypoxic and rapid anoxic deaths, but they are non-specific.

**Symptoms:**

(1) Stage of Dyspnoea: The excess on carbon dioxide in the blood stimulates the respiratory centre! The respiratory movements become increased in rate and amplitude, blood pressure rises, puke rate increases and there is slight cyanosis.

(2) Stage of Convulsions: The effort to breathe is mostly expiratory, the face is deeply congested, blood pressure is increased, veins in the neck become swollen and pulse is fast. There are frequently convulsions which cease as the victim becomes insensible and the reflexes are abolished.

(3) Stage of Exhaustion: The respiratory centre is paralyzed, the muscles become flaccid. There is complete insensibility, reflexes are lost and the pupils are widely dilated.

*Fig (6-1).* Tardieu spots on foot and leg in a case of hanging.

*Fig. (6-2).* Subconjunctival petechial haemorrhages.
The breathing is gasping, mostly inspiratory with long intervals between the gasps. The blood pressure falls, muscles relax, respiration ceases, and death takes place. The pulse is imperceptible, but the heart may continue to beat for some minutes after respirations have ceased. The three stages last for 3 to 5 minutes before death takes place. Occasionally, death is instantaneous.

Post-mortem Appearances: External: Post-mortem lividity is well developed. The face is either pale in slow asphyxia, or distorted, congested, often cyanosed and purple, and sometimes swollen and oedematous. Ears and fingernails are bluish. The tongue is protruded in most cases, and frothy and bloody mucus escapes from the mouth and nostrils. The eyes are prominent, the conjunctivae are congested and the pupils are dilated. Petechial haemorrhages, known as Tardieu spots are most marked where for mechanical reasons, capillary congestion is most prominent. Their distribution lies above the level of obstruction. They appear commonly as a rash-like shower in the scalp, eyebrows and face in hanging and strangulation and in the zone above the level of compression in traumatic asphyxia. A hand lens is useful to identify petechial haemorrhages. They are produced by simple mechanical obstruction to the venous return of blood from the parts, resulting in acute rise in venous pressure and over-distension and rupture of thinwalled peripheral venules and capillaries, especially in lax unsupported tissues, such as the eyelids, forehead, skin behind the ears, circumoral skin, conjunctivae.

**Fig. (6-3).** Petechial haemorrhages in lungs and sclera.
Fig. (6-4). Petechial haemorrhages in the brain.

Internal: The blood is fluid and dark, because of increased amount of CO2. The large veins are full of blood. Vessels may burst in the eardrum and in the nose causing bleeding. The larynx and trachea are usually congested, and contain a varying amount of slightly frothy mucus. The lungs are dark and purple. Some of the marginal portions of the lungs may show emphysematous changes. The abdominal viscera show marked venous congestion. The brain is often congested. Tardieu spots are numerous where the capillaries are least firmly supported, as in subconjunctival tissues and under the pleural and pericardial membranes, but they can appear almost anywhere if the degree of congestion and cyanosis is sufficient. They may occur as isolated minute haemorrhages or present in large numbers, and at times fuse to form patches of red colour, especially at the back of the heart. They are numerous in the region of auriculoventricular junction and the lower lobes and the interlobar fissure of the lungs and thymus. Tardieu spots are usually round, dark, and well-defined, varying in size from a pin’s head to two mm. Petechiae are rarely seen in the parietal pleura or the peritoneum. Petechiae and ecchymoses are common non-specific autopsy findings and may be seen in many non-asphyxial deaths beneath the pericardium, pleura, interlobar fissures and around the hilum.

The natural diseases which produce haemorrhage in the skin include bacterial endocarditis, meningococcal septicaemia and blood dyscrasias, especially purpura and haemophilia, and also in
deaths from secondary shock, coronary thrombosis, acute heart failure, and rapid anoxia. These conditions produce relatively large haemorrhages which tend to combine. Their distribution is general, whereas Tardieu spots are present above the level of obstruction.

COMA: It is a state of unrousable unconsciousness determined by the absence of any psychologically understandable response to external stimuli or inner need. It involves the central portion of the brain stem. Coma is a clinical symptom and not a cause of death.

Causes: (1) Compression of the brain, e.g., effusion of blood on or in the brain, inflammation, abscess or neoplasm of brain. (2) Drugs: opium, cocaine, chloral hydrate, alcohol, anaesthetics, hypnotics, cyanide, atropine, phenol, oxalic acid, GO, etc. (3) Metabolic disorders and infections. uraemia, eclampsia, diabetes, cholaemia, pneumonia, infectious fevers, heat stroke, etc. (4) Other causes: embolism and thrombosis in the cerebral vessels, epilepsy, hysteria, etc.

Autopsy: Injuries or disease of the brain may be present as noted in the causes of coma. The lungs, brain and the meninges are congested.

Autopsy: Injuries or disease of the brain may be present as noted in the causes of coma. The lungs, brain and the meninges are congested. SYNCOPE

Syncope is sudden stoppage of action of the heart, which may prove fatal. This term is also not used as a cause of death. Syncope or fainting is due to vasovagal attacks resulting from reflex parasympathetic stimulation. Syncope is caused by reflex bradycardia or asystole, or by reflex splanchnic vasodilation. Due to the acute reflex circulatory changes, blood pressure falls suddenly causing cerebral anaemia and rapid unconsciousness. Recovery is common.

Causes: (1) Anaemia due to sudden and excessive haemorrhage. (2) Asthenia from deficient power of heart muscle as in fatty degeneration of the heart, myocardial infarction and certain poisons. (3) Vagal inhibition. (4) Exhausting diseases.

Autopsy: The heart is contracted and the chambers are empty when death has occurred from anaemia, but chambers contain blood when death occurs due to asthenia. The lungs, brain and
abdominal organs are usually pale and the capillaries are congested.

**CAUSE OF DEATH**

The cause of death is the disease or injury responsible for starting the sequence of events, which are brief or prolonged and which produce death. It may be divided into: (1) Immediate cause, i.e., at the time of terminal event, e.g., bronchopneumonia, peritonitis, trauma, etc. (2) Basic cause, i.e., pathological processes responsible for the death at the time of the terminal event or prior to or leading to the event, e.g., gunshot wound of abdomen complicated by generalized peritonitis. (3) Contributory cause, i.e, the pathological process involved in or complicating, but not causing the terminal event. In some cases, the basic and the immediate cause may be identical.

The manner of death is the way in which the cause of death was produced. If death occurs exclusively from disease, the manner of death is natural. If death occurs exclusively by injury or is hastened due to injury in a person suffering from natural disease, the manner of death is unnatural or violent. Violence may be suicidal, homicidal, accidental or of undetermined or unexplained origin. The manner of death is established mainly by the investigational information and also by the pathological findings.

Mechanism of death is the physiological or biochemical disturbance, produced by the cause of death which is incompatible with life, e.g., shock, sepsis, toxaemia, severe metabolic acidosis and alkalosis, ventricular fibrillation, respiratory paralysis, etc.

Agonal period is the time between a lethal occurrence and death. Determination of the cause of death following autopsy is an interpretive and intelligent procedure, and depends upon sound evaluation of all data, circumstances surrounding the death, morphological evidence of disease and injury and additional laboratory investigations. In fact, the more a forensic pathologist knows about the total investigation, the more he can contribute from his autopsy. The effectiveness of the doctor would be greatly diminished if he had to work alone, and receives bodies for
autopsy without clothing, or a knowledge of the circumstances surrounding death

Classification of the Cause of Death: According to the autopsy findings, the cause of death may be grouped as follows.

(I) Natural Causes: (a) Where a lesion is found at autopsy which is incompatible with life.
   (b) Where a lesion is found at autopsy which is known to cause death.

(II) Unnatural Causes: (a) Where a lesion is found at autopsy which is incompatible with life,
   (b) Where a lesion is found which may have caused death or which may have precipitated death, but which is also known to be compatible with continued life.

(III) Obscure Causes: Where no lesion is found at autopsy, or if a lesion is found it is of a minimal or indefinite nature.

NATURAL CAUSES: Where a lesion is found at autopsy which is not compatible with life: In this category, the structural abnormalities establish beyond any doubt the identity of the disease which caused death. It is apparent that the lesions observed are incompatible with life because of its nature, site or extent, and they are ante-mortem in origin. The examples are: massive pulmonary thromboembolism, spontaneous intracerebral haemorrhage, ruptured myocardial infarct, rupture of an aortic aneurysm.

(B) Where a lesion is found at autopsy which is known to cause death: This category includes deaths in which some lesion is found at autopsy which may have caused death, but which is also compatible with continued life, e.g., arteriosclerosis of the coronary arteries, advanced chronic heart diseases, lobar pneumonia, etc. The autopsy does not reveal any other reasonable explanation for death, and the location, nature, severity and extent of the anatomical changes are sufficient to cause death, but it is not a conclusive proof. In such cases, the clinical history is important. In the case of coronary arteriosclerosis, if the deceased had several attacks of angina pectoris before his death, it can be reasonably assumed to be the cause of death. If the clinical history is unusual, the possibilities suggested by the history should be excluded before the death is attributed to the lesion.
Stenosing coronary atherosclerosis can cause sudden death, in which the autopsy may reveal a few scattered foci or only a single site of significant luminal narrowing, and there may be no recent vascular occlusive lesion. In most cases of sudden coronary death, a fresh thrombus or a recent myocardial infarct is not found at autopsy. In these cases, correlation of the morbid anatomy with the suddenness of death must be based on hypotheses. Emotional stress, e.g., anger, fear, joy, apprehension, etc., can precipitate acute failure in persons with organic heart disease, especially of the coronary atherosclerotic type. Emotional excitement significantly increases the workload of the heart which can overtax the limits of tolerance of damaged, laboring heart. In a normal person sudden release of adrenaline due to extreme terror can initiate ventricular fibrillation and death. Sudden deaths following assaults or even threats may occur due to existing heart disease.

Such events may be encountered in criminal charges arising out of collapse during fights, in minor assaults upon old persons, in litigation related to death from work stress, etc.

Sufferers from asthma and epilepsy can die suddenly and unexpectedly for no obvious reasons.

Un-natural Causes: (A) Where a lesion is found at autopsy which is not compatible with life: In some deaths, injuries may be found at autopsy which are incompatible with life in any person, e.g., decapitation, crushing of the head, avulsion of the heart from the large blood vessels. If they are ante-mortem, they are the definite cause of death.

(B) A lesion is found at autopsy which may have caused or precipitated death, but is compatible with life: At autopsy certain injuries may be found which from their nature, site or extent may not appear to be sufficient to cause death in a healthy person. But such injury may be the cause of death due to some complication resulting directly from the injury, but which is not demonstrable at autopsy. The degree of shock or the extent of haemorrhage following an injury cannot be assessed at autopsy. In such cases, the absence of any other adequate cause of death, and a consideration of the circumstances of the injury and of the symptoms found, may enable the doctor to attribute death to the injury with reasonable certainty.
In some cases, an injury may not appear to be sufficient to cause death, but some natural disease may be present which is known to cause death, e.g., coronary arteriosclerosis. In such cases, the circumstances of death and the symptoms found at the time of collapse may suggest that the death was precipitated by the injury.

NEGATIVE AUTOPSY: When gross and microscopic examination, toxicological analyses and laboratory investigations fail to reveal a cause of death, the autopsy is considered to be negative. Two to 5% of all autopsies are negative. Majority of obscure autopsies are in young adults. A negative autopsy may be due to; (1) Inadequate history: Deaths from vagal inhibition, status epilepticus, hypersensitivity reaction, etc. may not show any anatomical findings. If death results from laryngeal spasm in drowning, no anatomical findings may be present. (2) Inadequate external examination: The presence of fresh and old needle marks may be missed on cursory examination in a drug addict. The burn may be missed in electrocution. Death from snake bites and insect bites cannot be explained unless the bite marks are identified. (3) Inadequate or improper internal examination: Air embolism and pneumothorax are often missed. (4) Insufficient laboratory examinations. (5) Lack of toxicological analysis. (6) Lack of training of the doctor.

(1) Natural Causes: Cardiac Lesions

Concealed Trauma: (a) Cerebral Concussion: This may cause death without any external or internal marks of injury.

(b) Neck Injury: Cervical spinal fracture-dislocation may occur in diving, fall on head, impact downstairs with a wall-facing, from oblique impact or by fall of some object on the head, in such a way as to cause the dislocation especially with the head thrown back. The dislocation may be associated with tears of the ligaments and with the displacement of the skull from the spine. Sudden movements of the head over the spine with displacement may cause contusion and laceration of the spinal cord and rapid death. If death is delayed, there may be oedema, softening and necrosis of the cord. Injury to the spinal cord causes spinal concussion and may cause death. Unconsciousness is not seen in all persons, but all get up with residual tingling, numbness, weakness of arms or
legs and gait defects. Routine autopsy and X-ray may not show any abnormality. The dislocation of the cervical segments is often self-reducing, and externally there may not be any injury, or there may be abrasions on the brow or chin. Complete dissection of spine is essential. The spinal cord, cut longitudinally, may show internal bruising. Death may be instantaneous.

(c) Blunt Injury to the Heart: Contusion of the chest as in steering-wheel impacts, head-on collisions, from blast or heavy punching, may temporarily or permanently derange the heart with out much evidence of trauma. Contusion of the heart may cause death. Trauma may cause arterial spasm and it is likely that a functional inhibition or coronary spasm may cause sudden death that sometimes follow upon blows to the chest.

(d) INHIBITION OF THE HEART: (vagal inhibition; vaso-vagal shock; reflex cardiac arrest; nervous apoplexy or instantaneous physiological death): Sudden death occurring within seconds or a minute or two due to minor trauma or relatively simple and harmless peripheral stimulation are caused by vagal inhibition. Pressure on the baroreceptors situated in the carotid sinuses, carotid sheaths, and the carotid body causes an increase in blood pressure in these sinuses with resultant slowing of the heart rate, dilatation of blood vessels and a fall in blood pressure. In normal persons, pressure on the carotid sinus causes minimal effects with a decrease in heart rate of less than six beats per minute, and only a slight reduction (less than 10 mm. Hg) in blood pressure. Some individuals show marked hypersensitivity to stimulation of the carotid sinuses, characterized by bradycardia and cardiac arrhythmias ranging from ventricular arrhythmias to cardiac arrest.
The commonest cause of such inhibition is pressure on the neck particularly on the carotid sinuses as in hanging or strangulation. Unexpected blows to the larynx, chest, abdomen and genital organs and the impaction of food in larynx or unexpected inhalation of fluid into the upper respiratory tract, or the insertion of an instrument into the bronchus, uterus, bladder or rectum, puncture of a pleural cavity usually for producing a pneumothorax, may cause sudden death.

Autopsy: When death results from inhibition, there are no characteristic post-mortem appearances. The cause of death can be inferred only by exclusion of other pathological conditions, and from the accurate observations by reliable witnesses, concerning the circumstance of death.

**SUDDEN DEATH**

Death is said to be sudden or unexpected when a person not known to have been suffering from any dangerous disease, injury or poisoning is found dead or dies within 24 hours after the onset of terminal illness. Some authors limit sudden deaths as those occurring instantaneously or within one hour of onset of symptoms.
The incidence is approximately 10 percent of all deaths. No period in life is exempt. Natural death means that the death was caused entirely by the disease, and the trauma or poison did not play any part in bringing it about.


**Central Nervous System (10 to 18%):**
MECHANICAL INJURIES

(An injury is any harm, whatever illegally caused to any person in body, mind, reputation or property (Sec. 44, I.P.C). Mechanical injuries (wounds) are injuries produced by physical violence. A wound or injury is a break- of the natural continuity of any of the tissues of the living body. The question of wounds may be raised in a Court of law in both civil and criminal cases.


(II) Thermal Injuries: (1) Due to cold: (a) Frostbite, (b) Trench foot, (c) Immersion foot. (2) Due to heat: (a) Burns, (b) Scalds.

(III) Chemical Injuries: (1) Corrosive acids, (b) Corrosive alkalis,

(IV) Injuries due to electricity, lightning,

Legally, injuries are classified into: (1) Simple, and (2) Grievous General Principles:A wound is caused by a mechanical force which may be either a moving weapon or object, or the movement of the body itself.

ABRASIONS

An abrasion (gravel rash) is a destruction of the skin, which usually involves the superficial layers of the epidermis only. They are caused by a lateral rubbing action by a blow, a fall on a rough surface, by being dragged in a vehicular accident, fingernails, thorns or teethbite. Some pressure and movement by agent on the surface of the skin is essential. Sometimes, full thickness of the skin may be damaged in places, but usually in an interrupted, irregular manner, and intact epidermis remains within the area of the abrasion. The rougher the surface, and the more rapid the movement of the skin over it, the deeper is the injury. The exposed raw surface is covered by exudation of lymph and blood which produces a protective covering known as a scab or crust. They are simple injuries, bleed slightly, heal rapidly and scar is not formed. Large abrasions can cause severe pain and bleeding. The size,
situation, pattern and number of abrasions should be noted. Types: Abrasions are of four types.

(1) Scratches: They are caused by a sharp or pointed object passing across the skin, such as fingernails, pin or thorn. The surface layers of the skin are collected in front of the object, which leaves a clean area at the start and tags at the end. Sharp fingernails produce a scratch which is usually curved, one to 2 mm. in breadth, wide at the start and narrow at the end.

(2) Grazes (sliding, scraping or grinding abrasion): They are the most common type. They occur when there is movement between the skin and some rough surface in contact with it. They show uneven, longitudinal parallel lines with the epithelium heaped up at the ends of these lines, which indicate the direction in which the force was applied (fig.8-1). The presence of clothing will modify the appearance of abrasions. These abrasions are commonly seen in a road accident.

(3) Pressure Abrasions (crushing or friction abrasions): They are caused by crushing of the superficial layers of the epidermis and are associated with a bruise of the surrounding area. If the movement of instrument is around 90° to the skin, a pressure type of abrasion occurs. The ligature mark in cases of hanging and strangulation and the teethbite marks are the examples.

(4) Impact Abrasions (contact or imprint abrasions): They are caused by impact with a rough object, when the force is applied at or near a right angle to the skin surface.

**Fig (8-1).** Abrasion indicating direction of force.

**Fig. (8-2).** Abrasion of the skull due to
When a person is knocked down by a motor car, the pattern of the radiator grille, a headlamp rim or the tread of the tyre may be seen on the skin, which may contain road dirt, paint flakes, grease, etc. Impact abrasions and pressure abrasions reproduce the pattern of the object causing it and are called patterned abrasions. Patterned abrasions are produced when the force is applied at right angle to the surface of skin. Other examples of patterned abrasion are: imprint of bicycle chain, weave of coarse fabrics, the spiral of electric wires, ropes, serrated knife, etc.

**Age of the Abrasions:** The exact age cannot be determined.

- Fresh: Bright red. 12 to 24 hours: Lymph and blood dries up leaving a bright scab.
- 2 to 3 days: Reddish-brown scab, 4 to 7 days: Epithelium grows and covers defect under the scab. After 7 days: Scab dries, shrinks and falls off.

Histologically perivascular, cellular infiltration is seen at four to six hours. At 12 hours three layers are seen: a surface zone of fibrin and red cells; a deeper zone of infiltrating polymorphs; and a deepest layer of abnormally staining collagen. At 48 hours, scab is well-formed and epithelial regeneration is seen at the margins of the scab. By 4 to 5 days, small abrasions are completely covered by epithelium. By 5 to 8 days, subepithelial formation of granulation.
tissue is prominent. Reticulum fibres are seen at 8 days, and collagen fibres at 9 to 12 days.

Circumstances of Injuries: Abrasions are usually seen in accidents and assaults. Suicidal abrasions are rare.

**Table (8-1).** Difference between ante-mortem and post-mortem abrasions.

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<th>Trait</th>
<th>Ante-mortem abrasions</th>
<th>Post-mortem abrasions</th>
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<tr>
<td>1) Site:</td>
<td>Anywhere on the body.</td>
<td>Usually over bony prominences. Yellowish, translucent, and parchment-like. Less; scab often lies slightly below the level of skin. No intravital reaction and no congestion.</td>
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<tr>
<td>2) Colour:</td>
<td>Bright reddish-brown.</td>
<td></td>
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<tr>
<td>3) Exudation:</td>
<td>More; scab slightly raised.</td>
<td></td>
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<tr>
<td>4) Microscopic:</td>
<td>Intravital reaction and congestion seen.</td>
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Sometimes, hysterical women produce abrasions over accessible areas like the front of forearms or over the face, to fabricate a false charge of assault. Abrasions on the face or body of the assailant indicate a struggle. Persons collapsing due to heart attack tend to fall forward and receive abrasions to the supraorbital ridge, nose and cheek, but there will be no injuries on the upper limbs. A conscious person when falling puts out his hands to save himself, and abrasions may be produced on the palmar surfaces of the hands. The alcoholic tends to fall backwards and strikes his occiput on the ground.

Medico-legal Importance: (1) They give an idea about the site of impact and direction of the force. (2) They may be the only external signs of a serious internal injury. (3) Patterned abrasions are helpful in connecting the wound with the object which produced them. (4) The age of the injury can be determined, which helps to corroborate with the alleged time of assault. (5) In open wounds, dirt, dust, grease or sand are usually present, which may connect the injuries to the scene of crime. (6) Character and manner of injury may be known from its distribution, (a) In throttling,
crescentic abrasions due to fingernails are found on the neck, (b) In smothering, abrasions may be seen around the mouth and nose, (c) In sexual assaults, abrasions may be found on the breasts, genitals, inside of the thighs and around the anus, (d) Abrasions on the face of the assailant indicate a struggle, (e) Abrasions on the victim may show whether the fingernails of the assailant were long, irregular or even broken.

CONTUSIONS (BRUISES)

CA contusion is an effusion of blood into the tissues, due to the rupture of blood vessels, caused by blunt trauma. J Contusions may be present not only in skin, but also in internal organs, such as the lung, heart, brain, muscles and any tissue. They are caused by blunt force, such as fist, stone, stick, bar, whip, boot, etc.) The bruise is usually situated in the corium and subcutaneous tissues, often in the fat layer. In contusion, there is a painful swelling, and crushing or tearing of the subcutaneous tissues usually without destruction of the skin. The extravasated blood is diffusely distributed through the tissue spaces, and the margins are blurred. Bruises may be seen in association with abrasions (abraded-contusion) or lacerations.) When a large blood vessel is injured, a tumour-like mass called haematoma is formed.

Size: The size of a bruise is slightly larger than the surface of the agent which caused it, as blood continues to escape into the area.) As a general rule, the greater the force of violence used, the more extensive will be the bruises, but size and shape are modified by the following factors.

Condition and Type of Tissue If the part is vascular and loose, such as face, vulva,) scrotum, a slight degree of violence may cause a large bruise, as there is sufficient space for blood to accumulate the tissues are strongly supported, and contain firm fibrous tissues and covered by thick . dermis, such as abdomen, back, scalp, palms and soles, a blow of moderate violence may produce a comparatively small bruise) In boxers and athletes, bruising is much less because of good muscle tone. covered, or if the weapon used is a yielding one, such as sand bag. Bruising of the scalp is better felt than seen. Bruising of the scalp with
fluctuant centres can simulate depressed fracture. Bruising may be absent if the pressure is continued until death occurs. Even a severe injury may produce little haemorrhage, if it was preceded by an injury which produced deep shock. Bruising is relatively more marked on tissues overlying bone. Chronic alcoholics bruise easily. Resilient areas, such as the abdominal wall, and buttock bruise less.

**V(2) Age:** Children bruise more easily because of softer tissues and delicate skin, and old persons bruise easily because of loss of flesh and cardiovascular changes.

**^3** **Sex:** Women bruise more easily than men, because the tissues are more delicate and subcutaneous fat is more). Fat people bruise easily, because of greater volume of subcutaneous tissue.) A slight pressure with the fingers on the arm of a woman, and especially if she is obese and not accustomed to work or exercise, may produce a definite bruise.

**V*/"**(4) **Colour of Skin:** Bruising is more clearly seen in fair-skinned persons than those with dark skin, in whom they may be better felt than seen. The areas of extravasated blood appear darker even on heavily pigmented negroid skin. If the body is embalmed, skin bruises become more prominent probably (1) by forcing of additional blood into the damaged area, (2) increased transparency of overlying skin, and (3) formation of a dark pigment complex. Contusions appear much more clear in black and white photographs than by direct observation. Colour photographs more truly reproduce contusions.

**V(5) **Natural Disease:** When the vessels are diseased as in arteriosclerosis, bruising occurs very easily and may even result from coughing or slight exertion. In children, small bruises may be caused by the violent coughing as in whooping cough. Prominent bruising following minor trauma is seen in a person suffering from purpura haemorrhagica, leukaemia, haemophilia, scurvy, vitamin K and prothrombin deficiency and in phosphorus poisoning. Purpuric areas are clearly delineated from the surrounding tissue and never show swelling.

**V(6) **Gravity Shifting of the Blood:** Bruises do not always appear at the site of impact. A deep bruise, especially that due to the crushing of tissue against the bone may take a long time to become visible and also may not appear below the actual point of
impact. Blood will track along the fascial planes (or between muscle layers) which form the least resistance and may appear where the tissue layers become superficial (ectopic bruising). Haemorrhages in the soft tissues around the eyes and in the eyelids (spectacle haematoma; black eye), may be caused by (1) direct trauma, such as a punch in the eye, (2) blunt impact to the forehead, the blood gravitating downwards over the supraorbital bridge, (3) fracture of the floor of the anterior fossa of the skull. A bruise behind the ear may indicate a basal fracture, rather than a direct blow behind the ear. In fracture of the jaw, a bruise may appear in the neck. In fracture of the pelvis, a bruise may appear in the thigh. In fracture of the femur, a bruise may appear on the outer side of the lower part of the thigh. A blow to upper thigh may appear as a bruise above the knee. A kick on the calf of the leg may appear as a bruise around the ankle. The site of bruise does not always indicate the site of the violence.

**Patterned Bruising:** A bruise is usually round, but it may indicate the nature of the weapon especially when death occurs soon after infliction of injury.) If the person is living, this pattern may become obscure as the area of bruising tends to extend and merge with adjacent structures. A blow from a solid body, such as a hammer, or the closed fist usually produce a rounded bruise. Bruise made by the end of a thick stick may be round, but if any length of the stick hits the body, they are elongated and irregular. A blow with a rod, a stick or a whip produces two parallel, linear haemorrhages (railway line or tram line type). The intervening skin appears unchanged, because the rod forcibly dents the tissues inwards and momentarily stretches each side of the dent. This causes rupture of vessels in the marginal zones with a line of bruising, whereas the base of the dent becomes compressed and the vessels are not injured. When the rod is removed and the skin comes back to its normal position, the two sides of the depression remain as contused lines. In a bruise produced by a long rigid weapon, e.g.,

**Deep Tissue and Organ Contusions:** All organs can be contused. Contusions in vital centres, e.g., those control respiration and blood pressure can be fatal, even when very small. A small contusion of the heart can cause serious disturbance of normal rhythm or stoppage of cardiac action and death. Large
contusions often prevent adequate cardiac emptying and lead to heart failure. Contusions of other organs may cause rupture of that organ with slow or rapid bleeding into the body cavity, and may cause death.

The Age of Bruise: A bruise heals by destruction and removal of the extravasated blood. The red cells disintegrate by haemolysis, and the haemoglobin is broken down into haemosiderin, haematoidin and bilirubin by the action of enzymes. The colour change is very variable, starts at the periphery and extends inwards to the centre. At first. Red. Few hours to 3 days: Blue. 4th day: Bluish-black to brown (haemosiderin). 5 to 6 days: Greenish (haematoidin), 7 to 12 days: Yellow (bilirubin). 2 weeks: Normal.

Proof of Bruising: At autopsy, bruises may not be readily detected or they may be obscured by patches of post-mortem lividity, or by the dark colour of the skin. Contusions of the scalp can be demonstrated by reflecting the scalp and making incisions into the scalp from the aponeurotic surface. Contusions in the subcutaneous tissues may be detected by parallel incisions through the skin. Deep bruises are detected by deep incisions made into the muscles. When in doubt, a portion must be taken for microscopy.

(Medico-legal Importance: (1) Patterned bruises may connect the victim and the object or weapon, e.g., whip, chain, cane, ligature, vehicle, etc. (2) The age of the injury can be determined by colour changes. (3) The degree of violence may be determined from their size. (4) Character and manner of injury may be known from its distribution.

LACERATIONS

Lacerations are tears or splits of skin, mucous membranes, muscle or internal organs produced by application of blunt force to broad area of the body, which crushed or stretched tissues beyond the limits of their elasticity. They are also called tears or ruptures.

They are caused by blows from blunt objects, by falls on hard surfaces, by machinery, traffic accidents, etc. If the force produces bleeding into adjacent tissues, the injury is a/contused-laceration' or 'bruised-tear'. If the blunt force produces extensive bruising and
laceration of deeper tissues, it is called "crushing" injury. The force may be produced by some moving weapon or object or by a fall.

Types: (1) Split Lacerations: Splitting occurs by crushing of the skin between two hard objects.

Incised-like or Incised-looking Wounds: Lacerations produced without excessive skin crushing may have relatively sharp margins. Blunt force on areas where the skin is close to bone, and the subcutaneous tissues are scanty, may produce a wound which by linear splitting of the tissues, may look like incised wound. The sites are the scalp, eyebrows, cheek bones, lower jaw, iliac crest, perineum, and shin. (2) Stretch Lacerations: Overstretching of the skin, if it is fixed, will cause laceration.

This is seen in the running over by a motor vehicle, and the flap may indicate the direction of the vehicle. (3) Avulsion: An avulsion is a laceration produced by sufficient force (shearing force) delivered at an acute angle to detach (tear off) a portion of a traumatized surface or viscus from its attachments.

Fig. (8-4). Laceration of scalp (star-shaped).
Fig. (8-5). Crush injury leg caused by run over accident

Fig. (8-6). Avulsion caused by running over of the wheel of automobile.

In lacerations produced by shearing forces, the skin may not show signs of injury, but the underlying soft tissue is avulsed from the underlying fascia or connective tissue, producing a pocket which may be filled with blood. This is seen usually on the back of the thighs of pedestrians struck by motor vehicles.

(4) Tears: Tearing of the skin and tissues can occur from impact by or against irregular or semi-sharp objects, such as door handle of a car. This is another form of overstretched.

(5) Cut Lacerations: Cut lacerations may be produced by a heavy sharp-edged instrument.

The object causing a lacerated wound crushes and stretches a broad area of skin, which then splits in the centre. The edges are irregular and rough, because of the crushing and tearing nature of the blunt trauma. The margins are contused due to the bleeding into the tissues caused by trauma.

Characters: (1) Margins are irregular, ragged and uneven, and their ends are pointed or blunt, and they too show minute tears in the margins. Bruising is seen either in the skin or the subcutaneous tissues around the wound. Deeper tissues are
unevenly divided with tags of tissues at the bottom of the wound bridging across the margin. Tissue bridges consist of nerves, blood vessels and elastic and connective tissue fibres as they are stronger. (4) Hair bulbs are crushed. (5) Hair and epidermal tags may be driven deeply into the wound. (6) Haemorrhage is less because the arteries are crushed and torn across irregularly, and thus retract and the blood clots readily, except in wounds of the scalp, where the temporal arteries bleed freely as they are firmly bound and unable to contract. (7) Foreign matter may be found in the wound. (8) Depth varies according to the thickness of the soft parts at the site of the injury-and degree of force applied. (9) The shape and size may not correspond with the weapon or object which produced it. Long, thin objects, such as pipes, tend to produce linear lacerations, while objects with flat surfaces produce irregular, ragged, or Y-shaped lacerations.

Ante-mortem lacerations show bruising of margins, vital reaction, eversion, and gaping of the margins.

Medico-legal Importance: (1) The type of laceration may indicate the cause of the injury and the shape of the blunt weapon. (2) Foreign bodies found in the wound may indicate the circumstances in which the crime has been committed. (3) The age of the injury can be determined.

INCISED WOUNDS: An incised wound (cut, slash, slice) is a clean cut through the tissues, (usually the skin and subcutaneous tissues, including blood vessels), caused by sharp-edged instrument, which is longer than it is deep. It is produced by the pressure and friction against the tissue, by an object having a sharp-cutting edge, such as knife, razor, scalpel, sword, etc. In this, the force is delivered over a very narrow area, corresponding with the cutting edge of the blades.

Characters: (1) Margins: The edges are clean-cut, well-defined and usually everted. The edges are free from contusions and abrasions.

(2) Width: The width is greater than the edge of the weapon causing it, due to retraction of the divided tissues.

(3) Length: The length is greater than its width and depth, and has no relation to the cutting edge of the weapon, for it may be drawn to any distance.
Haemorrhage: As the vessels are cut cleanly, the haemorrhage is more: If the artery is completely cut, the bleeding will be more.

CX6) Direction: Incised wounds are deeper at the beginning, because more pressure is exerted on the knife at this point. This is known as the head of the wound. Towards the end of the cut the wound becomes increasingly shallow, till finally as the knife leaves the tissues the skin alone is cut. This is known as the tailing of the wound, and indicates the direction in which the cut was made.

Age of Incised Wound: In an uncomplicated wound, healing occurs as follows: Fresh: Haematoma formation. 12 hours: The edges are red, swollen and adherent with blood and lymph; leucocytic infiltration. 24 hours: A continuous layer of, endothelial cells covers the surface; overlying this a crust or scab of dried clot is seen.

Medico-legal Importance: (1) They indicate the nature of weapon (sharp-edged). (2) They give an idea about the direction of the force. (3) The age of the injury can be determined: (4) Position and the character of wounds may indicate mode of production, i.e., suicide, accident, homicide.

CHOP WOUNDS: They are deep gaping wounds caused by a blow with the sharp-cutting edge of a fairly heavy weapon, like a hatchet, an axe, machete sabre, or meat cleaver. The dimensions of the wound correspond to cross-section of penetrating blade. The margins are sharp and may show slight abrasion and bruising with marked destruction of underlying organs. If the edge is blunt, the margins are ragged and bruised. When the whole blade strikes the body at the same time, the depth may be same throughout the wound. Usually the lower end (heel) of the axe strikes the surface first, which produces a deeper wound than the upper (toe) end. The deeper end indicates the position of the assailant.
A stab wound is produced from penetration by long narrow instruments with blunt or pointed ends, such as knife, dagger, nail, needle, spear, arrow, screw driver, etc. into the depths of the body, penetrating the skin and the underlying tissues, that is deeper than its length and width on skin.*i This can occur by driving the object into the body, or from the body's pressing or falling against the object. The most common stabbing instruments are kitchen knives,
sheath knives or pen-knives. They are called penetrating wounds, when they enter a cavity of body. When the weapon enters the body on one side and comes out from the other side, perforating wounds or through-and-through puncture wounds are produced. The victim of a fatal penetrating injury may not show signs and symptoms of injury until many hours have passed. Characters: (1) Margins: The edges of the wound are clean-cut. There is usually no abrasion or bruising of the margins, but in full penetration of the blade, abrasion and bruising may be produced by the hilt-guard (metal piece between the blade and handle) striking the skin?)The margins may be abraded, and ragged if the cutting edge is blunt.

(2) Length: The length of the wound is slightly less than the width of the weapon up 10 which it has been driven in, because of stretching of the skin. For measuring the length of stab wound, the edges of the wound should be brought together. Deliberate lateral, forward, or backward movement of the weapon during its withdrawal from the body lends to widen the wound, and the length will be more than the maximum width of the blade.

(4) Depth: The depth (length of track) is greater than the width and length of the external injury. It is not safe to find out the depth of a stab wound by introducing a probe, because it may disturb a loose clot and may lead to fatal haemorrhage, or cause serious damage and may produce multiple erroneous wound tracks. The depth of a stab wound is usually equal to, or less than the length of the blade that was used in producing it, but on yielding surfaces like the anterior abdominal wall, the depth of the wound may be greater, because the force of the thrust may press the tissues underneath. If a double-edged weapon is used, the wound will be elliptical or slit-like and both angles will be sharp, or pointed. (3) A round object like the spear may produce a circular wound. (4) A round blunt-pointed object, such as a pointed stick, or metal rod may produce a circular surface wound with inverted ragged and bruised edges. Foreign material, such as dirt, rust or splinters may be found in the wound. (5)

The external and internal appearances of a stab wound help to give an opinion upon: (1) dimensions of the weapon. (2) the type of weapon. (3) the taper of the blade, (4) movement of the knife in the wound, (5) the depth of the wound. (6) the direction of the stab, and (7) the amount of force used.
Medico-legal Importance: (1) The shape of the wound may indicate the class and type of the weapon which may have caused the injury. (2) The depth of the wound will indicate the force of penetration. (3) Direction and dimensions of the wound indicate the relative positions of the assailant and the victim. (4) The age of the injury can be determined. (5) Position, number and direction of wounds may indicate manner of production, i.e., suicide, accident, or homicide. (6) If a broken fragment of weapon is found, it will identify the weapon or will connect an accused person with the crime.

**MEDICO-LEGAL AUTOPSY**

Autopsy means, post-mortem examination of a body. In every case the autopsy must be complete, all the body cavities should be opened, and every organ must be examined, because evidence contributory to the cause of death may be found in more than one organ. Partial autopsies have no place in forensic pathologic practice. A poor autopsy is worse than no autopsy at all, as it is more likely to lead to a miscarriage of justice.

The autopsy should be carried out by the doctor, and not left to a mortuary attendant. The doctor should remove the organs himself. The attendant should prepare the body and help the doctor where required, such as sawing the skull cap, reconstruct the body, etc.

The approach of the forensic pathologist to the investigation of death is different from that of the hospital pathologist. The hospital pathologist has easy access to relevant information about the history, physical condition and course of the disease leading to death. His main aim is to find morphologic changes explaining signs or symptoms of the disease. In medico-legal autopsies, often the clinical history is absent, sketchy or doubtful. In some cases, identity may not be known. He has to determine time of death and age of injuries. If there are any inconsistencies between the apparent death scene and his actual findings, he has to visit the scene of crime. He has to carry out careful external examination including clothing, in the determination of the pattern of injuries and their relationship to the object or weapon causing them. He has also to determine the manner and mechanism of death.
To find out the cause of death, whether natural or unnatural. To find out how the injuries occurred. To find out the manner of death, whether accidental, suicidal or homicidal. To find out the time since death. To establish identity when not known. To collect evidence in order to identify the object causing death and to identify the criminal. To retain relevant organs and tissues as evidence. In newborn infants to determine the question of livebirth and viability. If autopsy is not done, the exact cause of death, the presence and extent of disease or injury, the incapacitation produced by them, and whether there was any pain or suffering becomes only speculation. Rules for Medico-legal Autopsies: The body should be labelled as soon as it arrives in the mortuary. The autopsy should be conducted in a mortuary and never in a private room. However, it may become necessary to do an autopsy at the site, when the body is in an advanced state of putrefaction, and its transportation will be difficult, and materials of evidential value may be lost in transport. It should be conducted only when there is an official order authorizing the autopsy from the police, Magistrate or Coroner. It should be performed as soon as possible after receiving requisition, without undue delay. The medical officer should first read the inquest report carefully and find out the apparent cause of death, and obtain all the available details of the case from case sheet, accident register, etc., so that attention may be directed to the significant points, while doing the post-mortem examination and to carry out appropriate investigations, e.g. toxicologic, microbiologic, virologic, radiologic, etc. Lack of such information may result in loss of vital evidence. The examination should be conducted in daylight as far as possible, because colour changes, such as jaundice, changes in bruises, changes in post-mortem staining, etc. cannot be appreciated in the artificial light. If the body is received late in the evening, a preliminary examination is done to note the external appearances, the body temperature, and the appearance of the superficial injuries, extent of post-mortem lividity and rigor mortis, etc. The body must be identified by the police constable who accompanies it. The names of those who identify the body must be recorded. In unidentified bodies, the marks of identification, photographs, and fingerprints should be taken. No unauthorized person should be present at the autopsy. The investigating police officer may be present. As the autopsy is
conducted, details of examination should be noted verbatim by an assistant, and sketches made of all the important injuries. Nothing should be erased and all alterations should be initialled in the report. Even if the body is decomposed, autopsy should be performed as certain important lesions may still be found.

The autopsy report consists of:

**The preamble:** This should mention the authority ordering the examination, time of arrival of the body at the mortuary, the date and place of examination, the name, age and sex of the deceased and the means by which the body was identified.

The body of the report: This consists of a complete description of the external and internal examination of the body. It should contain a description of the nature, direction, exact situation and dimensions of the wounds. All negative findings should also be recorded.

Conclusions: The conclusion as to the cause of death must be given, based on the post-mortem findings. Conciseness and clear language are of high value in the expression of the opinion. The report should be honest, objective and scientific. This is followed by the signature and qualifications of the doctor.

**EXTERNAL EXAMINATION**

The external examination will provide most of the substance of the report, where death occurred due to trauma. The following should be noted: The clothing should be listed and examined, and described with regard to type of garment, its colour and consistence, tears, loss of buttons, or disarrangement indicating a struggle. The clothes should be removed carefully without tearing them to avoid confusion of signs of struggle. If they cannot be removed intact, they should be cut in an area away from any bullet hole or objects, along a seam in the garment. Cuts, holes, burns or blackening from firearm discharges should be noted and compared with the injuries on the body. Do not put objects through defects in clothing or wound. Blood stains, seminal stains, grease stains, etc., should be described. Stains due to poison, vomit should be kept for analysis. Stained and unstained areas of clothes should not be allowed to come in contact to avoid additional soiling, and as such clothes should not be folded while stains are wet. The clothes should be placed into clean plastic bags or other suitable clean
containers. Separate bags or containers should be used for each article. Nail scraping should be taken. Any visible fibres or other matter in the hand or adherent to it should be removed and placed in envelopes. Ten small envelopes are labelled, one for each finger. Height and weight of the body, and general state, body build, development, and nourishment.

Scalpel¹ Cartilage Dissecting knife

General condition of the skin (rash, petechiae, colour, looseness, turgor), asymmetry of any part of the body or muscular wasting. General description: This includes sex, age, colour, race, build, stature, deformities, nutrition, hair, scars, tattoo marks, moles, pupils, skin disease, circumcision, amputations, deformities, etc. Vaginal and anal swabs are taken and also swabs from areas of suspected seminal staining, in all cases of sexual assault. Note the presence of stains on the skin from blood, mud, vomit, faeces, corrosive or other poisons, or gunpowder. They should be described precisely and in detail. The presence of signs of disease, e.g., oedema of legs, dropsy, surgical emphysema about the chest, skin disease, eruptions, etc., are to be noted. The time since death should be noted from rectal temperature, rigor mortis, post-mortem hypostasis, putrefaction, etc. The head hair should be examined. Any foreign matter should be removed with forceps, and the hair combed through for trace evidence. Samples of both cut and pulled hair from at least six different areas of the scalp should be taken and labeled as to their origin. The face should be examined for frothy fluid at the mouth and nose, cyanosis, petechial haemorrhages, pallor, etc. The eyes should be examined
for the condition of the eyelids, conjunctivae, softening of the eyeball, color of sclerae, opacity of the cornea and lens, state and colour of pupils, artificial eyes, contact lenses, petechiae, and periorbital tissues for extravasation of blood. (13) The ears should be examined for leakage of blood, or CSF. (14) The neck must be examined for bruises, fingernail abrasions, ligature marks or other abnormalities. Observe degree of distention of neck vessels. (15) Thyroid: size, nodularity. (16) Lymph nodes: cervical, axillary, inguinal. (17) Thorax: symmetry, general outline. (18) Breasts: size, masses. (19) Abdomen: presence or absence of distension or retraction, striae gravidarum. (20) Back: bedsores, spinal deformity. (21) External genitalia: general development, oedema, local infection, position of testes. (22) The natural orifices, i.e., mouth, nostrils, ears, vagina, etc. should be examined for injuries, foreign matter, blood, etc. If the mouth cannot be opened, the masseter and temporalis muscles are divided above their insertion into the mandible, to allow the jaw to become mobile. The state of the lips, gums and teeth, marks of corrosion, and injuries to inside of the lips and cheeks should be noted. The state of the tongue, position with relation to the teeth, and the presence or absence of bruising or bite marks should be noted. The presence of froth about the mouth and nostrils and smell of alcohol, phenol, etc., should be noted. (23) Note the position of all the limbs- and particularly of the arms, hands and fingers. The hands should be examined for injuries, defense wounds, electric marks, etc., and if clenched to find out if anything is grasped in them. To open the hand completely, the flexor tendons of the fingers are cut at the wrist. The fingernails must also be carefully examined for the presence of any blood, dust or other foreign matter, indicative of struggle. Note for edema, needle marks, ulcers, gangrene, tumours, digital clubbing, etc. (24) External wounds should be systematically examined taking up each part of the body in turn. The description of wounds should include nature, site, length, breadth, depth, direction, position, margins, base and extremities. The condition of their edges, presence of foreign matter, coagulated blood and evidence of bleeding into nearby tissues noted. Determine whether they were caused before or after death, and their time of infliction. Collect foreign materials, e.g. hair, grass, fibres, etc., that may be in the wound If the injuries are
obscured by hair, as on the scalp, jthc area should be shaved. Deep or penetrating wounds should not be probed until the body is L-shaped opened. In burns, their character, position, extent and degree should be mentioned. The use of printed body sketches is very useful. Each injury can be drawn in, and measurement noted alongside each and distances from anatomical landmarks recorded. Photographs are useful, there is no substitute for a good colour photograph to preserve the appearance of a wound or injury. If the blood spots or smears on the skin are important, the area should be photographed before and after the skin is cleaned. Excluding stab and firearm wounds, all the injuries should be divided into two broad areas: external and internal. The position of the injuries should be filled in on the skeleton diagrams provided for the purpose. (25) the limbs and other parts should be examined for fractures and dislocations by suitable movements and by palpation and confirmed by dissection. (26) A list should be made of all items removed from the body, e.g., clothes jewellery, bullets, etc. The should be labelled, mentioned in the report and handed over to the police constable in a sealed cover after obtaining receipt. (27) The report should include all of the surgical procedures, applied dressings and other diagnostic and therapeutic measures found on external examination.

In case of discrepancy between the injuries noted in the inquest form and the findings of the doctor during postmortem examination who has conducted the inquest, the doctor should bring these facts to the notice of the officer who has conducted the inquest so that necessary corrections may be done in the inquest report.

**INTERNAL EXAMINATION** It is convenient to start the examination with the cavity chiefly affected. In a case of suspected cranial injury, the skull should not be opened until the blood has been drained out by opening the heart. Skin Incisions: Primary incisions are of three types (Fig. 5-2).
(1) The "I" shaped incision, extending from the chin straight down to the symphysis pubis, passing either to the left or right of the umbilicus. The umbilicus is avoided because the dense fibrous tissue is difficult to penetrate with a needle, when the body is stitched after autopsy.

(2) "Y" shaped incision begins at a point close to the acromial process. It extends down below the breast and across to the xiphoid process. A similar incision is then made on the opposite side of the body. From the xiphoid process, the incision is carried downwards to the symphysis pubis.

(3) An incision is made in midline from suprasternal notch to symphysis pubis. The incision extends from suprasternal notch over the clavicle to its centre on both sides and then passes upwards over the neck behind the ear.

CHEST: The muscles of the chest are dissected away, keeping the edge of the knife directed inwards towards the ribs, carried back to the midaxillary line, down to the costal margin and up over the clavicles. Cases of pneumothorax are demonstrated before the chest wall is opened. A pocket is dissected on the affected side between the chest wall and the skin, and is filled with water. and the wall is punctured with the knife under the water. The scalpel should be twisted a few times to make sure that the wound is open. If air under pressure is present, it will bubble out of the opening through the water. In case of tension pneumothorax, the gas will escape with a definite hiss when the intercostal spaces are punctured, and also the lung will be collapsed.

The ribs, sternum and spine should be examined for fractures, and the chest is opened by cutting the costal cartilages near the costal ends, with a cartilage knife. Begin at the upper border of the second cartilage, keeping very close to the
costochondral junctions. The knife should be inclined about 30° to the vertical. In old persons where the rib cartilages are calcified, a pair of rib shears are used. Then, disarticulate the sternoclavicular joint on each side by holding the knife vertically and inserting the point into the semicircular joint. The position of this joint can be made out by moving the shoulder tip with the left hand, which causes the joint capsule to move. To divide the joint capsule, the knife is put in vertically and turned in a circular manner. The diaphragm is divided at its attachment to the lower ribs and sternum up to the spine.

The pleural cavity should be examined before complete removal of the sternum, to prevent leakage of blood from subclavian and jugular veins into the pleural cavity before inspection. Before removal of the thoracic organs, in situ inspection should include: (1) observation of the lumen of the main pulmonary vessels, (2) observation of the right atrium and ventricle for air embolism, (3) the state of distension or collapse of the lungs, (4) pleural cavities for the presence of fluid, blood or pus and pleural adhesions, (5) pericardium for cardiac tamponade, and (6) collection of blood sample from the heart for toxicological examination.

The pericardial sac normally contains 20 to 50 ml. of straw-coloured fluid and the pericardium is smooth and glistening. White spots (milk spots) on the surface of the heart indicate healed pericarditis. In acute pericarditis, the sac contains large collections of serous or purulent fluid and fibrin deposits (bread-and-butter pericardium). Haemorrhagic fluid in the sac is seen in malignancy and rarely in tuberculosis, uraemia, bleeding diseases and secondary to myocardial infarction.

**AIR EMBOLISM:** If air embolism is suspected, the head should be opened first and the surface vessels of the brain examined for gas bubbles, which must be prominent and definite, but not segmental breakup of the blood in the vessels with collapsed segments between. Care should be taken to avoid pulling the sternum and ribs to avoid creating negative pressure in the tissues which may result in aspiration of air into vessels. Before handling the thoracic organs, the pericardium is opened, heart is lifted upwards and the apex is cut with a knife. The left ventricle is filled with frothy blood, if air is present in sufficient quantity to
cause death. If the right ventricle contains air, the heart will float in water. Another method of demonstrating air embolism is by cutting the pericardium anteriorly and grasping the edges with haemostat on each side. The pericardial sac is filled with water and the heart is punctured with a scalpel and twisted a few times. Bubbles of air will escape if air is present. The amount of air can be measured by placing an inverted water-filled graduated glass cylinder, with the mouth of the cylinder in the pericardial sac. Air in inferior vena cava can be demonstrated by puncturing it under water, and looking for escape of bubbles of gas.

There are two distinct methods of removing the viscera from the abdominal and thoracic cavities:

**Fig. (5-3).** Changes in positions and interrelationships of the organs in erect and supine positions.

**EXAMINATION OF ORGANS:** The en masse chest and abdominal organs are kept on a wooden board with posterior surface upwards and the tongue facing the operator.
DESCRIPTION OF AN ORGAN: A description of the organ systems should be limited to a clear, concise, objective description of shape, colour, and consistency and the presence or absence of any lesions other than those systematically described under trauma. The microscopic description may be limited to the positive findings. The pathologist should indicate those tissues he had examined and the number of sections he has taken in any one tissue.

(1) Size: Measure by tape. In the liver, blunting of the inferior border points to enlargement, and sharpness to atrophy. A usually tense capsule is in favour of enlargement, and loose capsule with laxness. A straight course of superficial vessels as on heart shows increased size, while undue tortuosity means decrease.

(2) Shape: Note any departure from normal.

(3) Surface: Most organs have a delicate, smooth, glistening, transparent capsule of serosa. Look for any thickening, roughening, dullness or opacity.

(4) Consistency: The softness or firmness as measured by pressure of the finger.

(5) Cohesion: It is the strength within the tissue that holds it together. It is judged by the resistance of the cut surface to tearing, pressure or pulling. An organ with reduced cohesion is friable, while when it is increased, the tissue seems to be tough or leathery. If a small toothed forceps bites into a testes it should pull away threads composed of tubules.

(6) Cut surface: (A) Colour: Every organ (except brain) is basically some shade of grey, but this is altered by the red contributed by its blood supply. Other colours can be added by jaundice or fatty infiltration (yellow), lipofuscin or haemosiderin (brown), malarial pigment (grey-brown). Anaemia causes pallor, while congestion adds a blue tinge. (B) Structure: This is a factor of the particular organ, e.g., cortex and medulla in the kidney. In disease these may become indistinct or greatly exaggerated.

HEART: It is held at the apex and lifted upwards and pulmonary vessels, superior and inferior vena cavae, and the ascending aorta are cut as far away as possible from the base of the heart. The heart is examined externally for adhesions, pericarditis, discoloration of an underlying infarct and for aneurysms. The pulmonary arteries should be palpated before
they are cut and looked for an embolus when they are incised. The pericardium is examined and incised with the tip of the scissors and the heart is exposed. Any blood or fluid in the pericardium is noted. The isolated heart is studied as follows (fig.5-4). It is opened in the direction of the flow of blood with the enterotome. The right atrium is cut between the openings of superior and inferior vena cavae. A small secondary incision is made to open the auricular appendage to detect thrombi. In opening the right ventricle, the lateral margin of right ventricle faces the dissector, the atria being directed towards him. The enterotome introduced into the right atrium, cuts through the tricuspid orifice, and opens the right ventricle along the lateral margin. The circumference of an intact valve of heart can be measured by inserting specially made graduated cones, marked at various levels with the circumference. The heart is held in the palm of the hand so that the pulmonary valve is horizontal and neither collapsed nor stretched. To demonstrate the competence of the pulmonary valve, a gentle stream of water is directed on to the valve. After the blood is washed away, it can be observed how well the cusps come into apposition, and whether water leaks into the already opened ventricle. The competence of tricuspid and mitral valve cannot be
satisfactorily tested post-mortem. In opening the pulmonary valve, the heart is placed so that the apex is directed towards the examiner. The enterotome is introduced into the right ventricle close to the apex, and the conus pulmonalis and pulmonary valve are cut about 15 mm. to the right of, and parallel to the interventricular septum in the anterior wall of the right ventricle. The interventricular septum is identified by the anterior descending branches of the coronary vessels crossing down the epicardium. The incision should extend into the pulmonary trunks and the left pulmonary artery. Note whether the contents of the right ventricle and auricle are fluid blood, currant-jelly clot or chicken-fat clot. The left atrium is cut between the openings of the pulmonary veins. Then, the left atrium is cut along its lateral wall. This incision extends through the mitral orifice, and passes along lateral margin of the left ventricle up to the apex. The next incision extends from the apex along the interventricular septum into the aorta, opening the aortic valve. The water competency of the aortic valve can be tested after cutting the aorta transversely. Both auricular appendages should be examined for the presence of thrombi. The heart should be weighed, after the blood clots in the cavities are removed, and measurements of the circumference of valves and of thickness of right and left ventricle should be taken.

The anatomy of coronary arteries varies considerably. Usually, there is a short main trunk of the left coronary artery, which soon bifurcates into the circumflex branch, and the anterior descending branch. The coronary arteries are examined by making serial cross-sections along the entire course of the major vessels about 2 to 3 mm.apart, using a scalpel. This method demonstrates narrowing of the vessel, and any ante-mortem thrombus in its lumen, without danger of dislodging it. The coronaries should not be opened by passing a scissors through them from the ostia, as they have a crushing and cutting action and produce so much distortion that any thrombus is obscured, and also the thrombus may be pushed along with the point of the scissors. The anterior descending branch of the left coronary artery is cut downwards along the front of the septum, then the circumflex branch on the opposite side of the mitral valve. The right coronary artery is followed from the aorta to the cut near the pulmonary valve, and then above the tricuspid valve. The presence of acute coronary
lesions, e.g. plaque rupture, plaque haemorrhage, or thrombus should be noted. The muscle of the right and left ventricles is incised in a plane parallel to epicardial and endocardial surfaces, which reveals infarction and fibrosis most clearly.

Subendocardial Haemorrhage: The haemorrhages are seen in the left ventricle, on the left side of the interventricular septum and on the opposing papillary muscles and adjacent columnae carnae. The haemorrhages are flame-shaped, confluent and tend to occur in one continuous sheet rather than patches. When the bleeding is severe, it may raise the endocardium into a flat blister. They are seen: (I) after sudden severe hypotension due to severe loss of blood or from shock, (2) after intracranial damage, such as head injury, cerebral oedema, surgical craniotomy, or tumours, (3) death from ectopic pregnancy, ruptured uterus, ante-partum or post-partum haemorrhage, abortion, (4) various types of poisoning, especially arsenic.

**HEAD:** A wooden block is placed under the shoulders so that the neck is extended and the head is fixed by a head rest, which should have a semicircular groove to hold the back of the neck.

A coronal incision is made in the scalp, which starts from the mastoid process just behind one ear, and is carried over the vertex of the scalp to the back of the opposite ear (fig. 5-5). The incision should penetrate to the periosteum. The scalp is reflected forwards to the superciliary ridges, and backwards to a point just below to the occipital protuberance. Any bruising of the deeper tissues

![Fig. (5-5). Primary scalp](image)

![Fig. (5-6).](image)

Common method incision of removing the skull cap. of the scalp or injury to the bone should be noted. The temporal and masseter muscles are cut on either side, for sawing the skull. The saw-line is made in slightly V-shaped direction, so that the skull
cap will fit exactly back into the correct position. The saw-line, should go through the bones along a line extending horizontally on both sides, from about the centre of the forehead to the base of the mastoid process, and from these latter points backwards, and upwards to a point a little above the external protuberance (fig.5-6). Thickness of the skull varies in different parts, being thinner where protected by thick muscles. The average thickness is 3 to 5 mm. Care should be taken to avoid sawing through the meninges and brain. To avoid this, stop when saw meets little resistance. A chisel and hammer should not be used to loosen the skull completely. Heavy hammering may cause false fractures, or extend any existing fractures. An elevator is inserted into the frontal saw-line, and the cap of the skull is raised, and with the hands the whole skull cap is pulled backwards, and broken across at the lambdoid suture and removed. Fixation of dura to bone is much firmer in children, in whom it tends to dip into the sutures. The meninges are examined for congestion, disease, etc. In old persons, the meninges over the vertex are often white and thickened, with little calcified patches (arachnoid granulations). A note should be made of extradural or subdural haemorrhage and also of intracranial tension. If possible, measure accumulations of extradural or subdural haemorrhage. If they are solid, express in terms of grams of weight, or area covered over the superior portion of the brain. Describe variation in thickness if the material is semi-liquid and cannot be easily collected. The superior longitudinal sinus is opened along its length with a scalpel, and carefully examined for an ante-mortem thrombus. This is of medico-legal importance, as ante-mortem thrombus in this situation can lead to back-pressure in the bridging veins crossing the subdural space. and cause subdural haemorrhage.

The dura mater is grasped anteriorly with a forceps, and with a scissors or scalpel, the dura mater is divided from before backwards at the level of the skull division on both sides. The scalpel is now passed vertically downwards alongside the falx cerebri at its anterior end, and the knife turned medially to divide the falx. With the forceps, the dura and the falx are now pulled backwards, and the surface of the brain examined.
Fig. (5-7). Technique for opening the skull of a newborn infant.

Fig. (5-8). Circle of Willis.

**PRESERVATION OF VISCERA IN CASES OF SUSPECTED POISONING:** The following must be preserved in all fatal cases of suspected poisoning.

1. Stomach and its contents. If the stomach is empty, the wall should be preserved.
(2) The upper part of small intestine (about 30 cm. length) and its contents. / 
(3) Liver not less than half kilogram. 
(4) One kidney or half of each kidney. 
(5) Blood 100 ml. Minimum 10 ml. 
(6) Urine 100 ml.

As most poisons are taken orally, the poison is most likely to be present in the stomach and intestinal contents and in their wall. After absorption all poisons pass through the liver, which is the major detoxicating organ and has the power of concentrating many poisons making them identifiable when the blood and urine concentrations may have declined to very low levels. The kidney being the organ of excretion contains large amounts of poison, which is excreted into the urine.

**CAUSE OF DEATH:** After completing the post-mortem examination, a complete but concise report should be written in duplicate using carbon papers. One copy is sent to the investigating officer and another copy is retained for future reference. Autopsy report should contain a list of specimens and samples retained for further examination. The report should be given on the same day, as the details cannot be accurately recorded from memory, if there is much delay. If laboratory tests have to be carried out an interim report should be written, and later after obtaining the reports, a supplementary report written. It has been said with a considerable measure of truth, that autopsy reveals the diseases and lesions that the person lived with, and not necessarily those which killed him. A definite opinion should be given whenever possible, but if the cause of death cannot be found out, it should be mentioned, in the report. In such cases, viscera should be preserved and histological and bacteriological examinations carried out. While giving cause of death, the word 'probably' should be avoided. In suspected cases of poisoning, the opinion should be kept reserved until the Chemical Examiner's report is received. The conclusion that death was caused by poison depends on evaluation of clinical, autopsy, toxicologic and circumstantial evidence. If opinions are given to police before evaluation of data are complete, they should be clearly and unmistakably labelled as preliminary impressions, subject to change if and when the facts so warrant. It must be recognised
that the determination of cause and manner of death are opinions, not facts. The opinion of one medico-legal officer can differ from another. If the cause of death is not found on autopsy, the opinion as to the cause of death should be given as "undetermined," and the manner of death as "unknown."

Autopsy of Decomposed Bodies: It is a fundamental rule of forensic pathology that all human remains should be examined, even when they are not likely to provide information. Even when the body shows advanced decomposition, a thorough examination may show a gross traumatic or pathological lesion. The skin though discoloured, may show the presence of a gross external injury, e.g., a bullet wound, lacerated wound or incised wound. Fractures are easily detected. Gross pathological lesions may be found, e.g., valvular lesions of the heart. Ante-mortem thrombi may persist.

EFFACEMENT OR OBLITERATION OF IDENTITY: The identity of a dead body may be carried out the following methods.

1) Purposive removal of the identifying features, e.g., fingerprints, tattoo marks, scars, moles, teeth, hair, etc. and articles of clothing.
2) Animals, e.g., rats, dogs, jackals and hyenas and birds, such as vultures may attack a dead body and mutilate it in a very short time, when the body is exposed in an open place.
3) Burning or incineration.
4) Advanced putrefaction.
5) Dismemberment and burying or throwing different parts in different places.
6) Chemical destruction of the body in corrosive acids or alkalis.
7) Dismemberment by moving vehicles, like trains or by machinery.
8) Bomb explosions, which may disintegrate the body.

EXAMINATION OF MUTILATED BODIES OR FRAGMENTS: Mutilated bodies are those which are extensively disfigured, or in which a limb or a part is lost but the soft tissues, muscles and skin are attached to the bones. Sometimes, only a part of the body, such as head, trunk or a limb may be sent.

1) Human or Animal: This is easy if the head, trunk or limbs are available, but when pieces of muscle only are available without
attached skin or viscera, it is very difficult. In such cases, definite opinion can be given by performing precipitin test or anti-globulin inhibition test using blood, or any other soft tissue, if the tissue is not severely decomposed.

(2) One or more Bodies: This is determined by fitting together all separate parts. If there is no disparity or reduplication, and if the colour of the skin is same in all parts, they belong to one body.

(3) Sex: It can be determined if the head or trunk is available, from the presence and distribution or absence of hair, characters of the pelvis, skull, etc. It can also be determined from the recognition of prostatic or uterine tissue under a microscope which resist putrefaction, and are found even in advanced state of putrefaction.

(4) Age: Age can be determined from general development, skull, teeth and ossification of bones.

(5) Stature: It can be determined from the measurement of long bones.

(6) Identity: It can be determined from fingerprints, tattoo marks, scars, moles, hair, teeth, deformities, etc., articles of clothing and superimposition technique.

(7) Manner of Separation of Parts: This is determined by examining the margins of the parts, whether they had been cleanly cut, sawn, hacked, lacerated, disarticulated at the joints or gnawed through by animals.

(8) Time since Death: The probable time since death may be determined from the condition of parts.

(9) Cause of Death: The cause of death can be made out if there is evidence of fatal injury to some vital organ or large blood vessel, or marks of burning or deep cuts or fractures of bones, especially of the skull or the cervical vertebrae, hyoid bone or of several ribs.

(10) Ante-mortem or Post-mortem: This may be determined by examining the margins of parts for evidence of vital reaction.

**EXHUMATION**

Exhumation is the digging out of an already buried body from the grave. There is no time limit for exhumation in India. Autopsies
are performed on exhumed bodies: (1) In criminal cases, such as homicide, suspected homicide disguised as suicide or other types of death, suspicious poisoning, death as a result of criminal abortion and criminal negligence. (2) In civil cases, such as accidental death claim, insurance, workmen's compensation claim, liability for professional negligence, survivorship and inheritance claims or disputed identity.

Authorisation: The body is exhumed only when there is a written order from the Executive Magistrate.

Procedure: The body is exhumed under the supervision of a medical officer and Magistrate in the presence of a police officer. Wherever practicable, the Magistrate should inform the relatives of the deceased, and allow them to remain present at the enquiry (176(4)Cr.P.C.)- The grave site should be positively identified with identifying features, such as location of burial plot, the headstone, and gravemarker. The distance of the grave from some of the permanent objects like trees, rocks, road or fence should be noted. It should be conducted in natural light. If the grave is in an open place with lot of spectators, the area should be screened off. The burial should be uncovered 10 to 15 cm. at a lime, and notes should be made about the condition of the soil, water content, and vegetable growth. After the dirt has been removed from above and around the corpse, it should be photographed in the position in which it was found. A drawing of the grave and body or skeleton should be made noting all the details, e.g., if the face is up, or to the right, arms are extended, or the lower limbs are flexed. If decomposition is not advanced, a plank or a plastic sheet should then be lowered to the level of the earth on which the body rests, the body gently shifted on to planks or sheet and then removed from the grave. If skeletonisation is advanced, then it may become necessary to dig down beside and then beneath the body so that some firm material, such as a sheet of hardboard may gradually be inserted under the body, which can then be lifted and transported on it. If the body is skeletonised, after removing the remains, the soil must be sifted in a finely-meshed screen to recover smaller objects, e.g., teeth, epiphyses, bullets, etc.

The condition of the burial clothes and the surface of the body should be noted. In cases of suspected mineral poisoning, samples of the earth (about half kg.) in actual contact with the body
and also from above, below and from each side should be collected. Any fluid or debris in the coffin should also be collected. A portion of the coffin and burial clothes must be removed in order to exclude any possibility of contamination from external sources. The body should be identified by close relatives and friends. All personal effects, clothing, hair, nails, etc., should be picked up for examination.

Autopsy: Disinfectants should not be sprinkled on the body. If the body has been buried recently, the post-mortem examination is conducted in the usual manner. Various artifacts have to be interpreted correctly. In much putrefied bodies, an attempt should be made to establish the identity. All the viscera should be preserved for chemical analysis. If the body is reduced to skeleton, the bones should be examined.

Second Autopsy: Before performing the second autopsy, the doctor should obtain all the available documents relating to the case especially the first autopsy report, photographs of the scene of death, of the body taken during first autopsy, hospital records, police investigative reports, etc. and if possible the first autopsy pathologist should be called to correlate all the findings. Contusions become visible when the blood is drained from the tissues following the first autopsy. Decomposition causes merger of the contusions with blurring of their patterns. The interpretation of the findings of a second autopsy performed on a previously autopsied exhumed body is difficult due to the various artifacts of burial and exhumation, and serious alterations resulting from the first autopsy. The findings should be documented in great detail, whether the findings are confirmatory or contradictory from the result of first autopsy. It is possible that valuable results may be obtained. Even if no new information is obtained from the second autopsy, it will help in putting an end to rumours or suspicions.

**POST-MORTEM CHANGES**

**SIGNS OF DEATH AND CHANGES 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. The signs of
death appear in the following order.

(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) Post-mortem lividity.
   (9) Rigor mortis.
(III) Late (decomposition and decay).
   (10) Putrefaction.
   (11) Adipocere formation.
   (12) Mummification.

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.
Suspended animation may be produced voluntarily.

CHANGES IN THE SKIN: Skin becomes pale and ashy-white
and loses elasticity within a few minutes of death.

CHANGES IN THE EYE: Loss of Corneal Reflex:
   Opacity of the Cornea: The opacity is due to drying and is
delayed
   if the lids are closed after death.

COOLING OF THE BODY: The cooling of the body (algor
mortis; 'chill of death') after death is a complex process, which
does not occur at the same rate throughout the body. Cessation of
energy production and of heat occur after somatic death, which
results in fall in body temperature. The body cools more rapidly on
the surface and more slowly in the interior. The body heat is lost by conduction, convection and radiation. Only a small fraction of heat is lost by evaporation of fluid from the skin. The rectum is the ideal place to record temperature except in cases of sodomy. The temperature can also be recorded by making a small midline opening into the peritoneal cavity and inserting the thermometer in contact with the inferior surface of the liver. The time of this reading is recorded and temperature of environment is recorded at the same time. Reading should be made at intervals, 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:

Normal body temperature — rectal temperature Rate of temperature fall per hour

In cases of fat or air embolism, certain infections, septicaemia, heatstroke and in pontine haemorrhage, thyrotoxicosis, drug reactions, etc. a sharp rise in temperature occurs. 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 0.5 to 0.7° 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. A body immersed in cold water cools rapidly; the rate of fall being almost twice as fast as by air cooling. Bodies cool more rapidly in running water than in stagnant water. Bodies buried in earth cool rapidly than those in air, but more slowly than those in 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.
Determination of temperature of the body is important only in cold and temperate climates. In tropical zones, the post-mortem fall in temperature may be minimal. A body in zero weather may undergo freezing and become stony-hard from formation of ice in cavities and blood vessels. The ice inside the skull may expand and cause separation of sutures.

Medico-legal Importance: It helps in the estimation of the time of death.

Post-mortem 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.

**POST-MORTEM HYPOSTASIS**

This is the bluish-purple or purplish-red discoloration 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 capillary-venous distention. It is also called post-mortem staining, subcutaneous hypostasis, livor mortis, cadaveric lividity. 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 small vessels of the dependent parts of the body. Filling of these vessels produces a bluish-purple colour to the adjacent skin.

*Fig. (7-1).* Post-mortem hypostasis, and areas of contact flattening.
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. When the pressure is released the lividity will reappear. Post-mortem 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. It is usually well developed within four hours and reaches a maximum between 6 and 12 hours. It is present in all bodies. The extent and the time of appearance of lividity mainly depend 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 poisoning, congestive cardiac failure, and asphyxia. In such cases, hypostasis will be marked shortly after death. The distribution of P.M. hypostasis: The distribution of the stain depends on the position of the body. In a body lying on its back, it first appears in the neck, and then spreads over the entire back with the exception of the parts directly pressed on, i.e. occipital scalp, shoulder-blades, 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. 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. 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. If the body has been suspended in the vertical position as in hanging, hypostasis will be most marked in the legs, external genitalia, lower parts of the forearms and hands, and if suspension be prolonged for a few hours, petechial haemorrhages are seen in the skin. If the body is moved before the blood coagulates, these patches will disappear and new ones will form on dependent parts, but lividity to a lighter degree remains in the original area, due to staining of the tissues.
by haemolysis. When coagulation in capillaries takes place, the stains become permanent and this is known as fixation of post-mortem staining. Hypostases may resemble bruises. In doubtful cases, a portion should be removed for microscopic examination.

In a dead body lying on its back, blood accumulates in the posterior part of the scalp due to gravity. In advanced decomposition, due to lysis of red cells and breakdown of the vessels, blood seeps into the soft tissues of the scalp. This appears as a confluent bruising and cannot always be differentiated from true ante-mortem bruising.

The hypostatic areas have distinct colour in certain cases of poisoning, e.g. (1) In carbon monoxide poisoning, the colour is cherry-red. (2) In hydrocyanic acid poisoning and sometimes in burns the colour is bright-red. (3) In poisoning by nitrites, potassium chlorate, potassium bicarbonate, nitrobenzene and aniline (causing methaemoglobinaemia) the colour is red-brown, or brown. (4) In poisoning by phosphorus, the colour is dark-brown.

In asphyxia, the colour of the stains is deeply bluish-violet or purple. A brownish hypostasis may be seen in methaemoglobinaemia. A bright pink colour is seen in hypothermia, and bodies taken from cold water as the wet skin allows atmospheric oxygen to pass through and also at low temperatures haemoglobin has a greater affinity for oxygen.

**Internal Hypostasis:** 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. Hypostasis in the heart can simulate myocardial infarction, and in the lungs it may suggest pneumonia.

**Medico-legal Importance:** (1) It is a sign of death. (2) Its extent helps in estimating the time of death which is unreliable. (3) It indicates the posture of the body, at the time of death. (4) It may indicate the moving of the body to another position some time after death. (5) Sometimes, the colour may indicate the cause of death.
MUSCULAR CHANGES

After death, the muscles of the body pass through three stages: (1) Primary relaxation or flaccidity. (2) Rigor mortis or cadaveric rigidity. (3) Secondary flaccidity.

Primary Flaccidity: During this stage, death is only somatic and it lasts for one to two hours. All the muscles of the body begin to relax soon after death. The lower jaw falls.

RIGOR MORTIS

This is a state of stiffening of muscles.
Mechanism: A voluntary muscle consists of bundles of long fibres. These myofibrils are the contractile elements, and are made up of protein filaments of two types, actin filaments and myosin filaments which form a loose physico-chemical combination called actomyosin, which is physically shorter than the two substances uncombined.

During life, the separation of the actin and myosin filaments, and the energy needed for contraction are dependent on adenosine triphosphate (ATP). ATP is responsible for elasticity and plasticity of the muscle. The dephosphorylation of ATP by the action of ATPase produces ADP and phosphate, and a large amount of energy which is used for muscle contraction. The lost ATP is replaced during life by resynthesis. At the time of somatic death, enough ATP is present in the muscle to maintain relaxation. After death the ATP is progressively and irreversibly destroyed leading to increased accumulation of lactates and phosphates in the muscles. There is no resynthesis of ATP. The post-mortem alteration of ATP is due to dephosphorylation and deamination. When the ATP is reduced to a critical level (85% of the normal), the overlapping portions of myosin and actin filaments combine as rigid link of actomyosin, which is viscous and inextensible, and causes hardness and rigidity of muscle rigor. The rigidity of the muscle is at its maximum, when the level of ATP is reduced to 15%. Simultaneously, there will be a rise in lactic acid and a fall in hydrogen ion concentration due to glycolysis. When lactic acid concentration reaches a level of 0.3%, muscles go into an
irreversible state of contraction known as rigor mortis. Rigor persists until decomposition of the proteins of the muscle fibres makes them incapable of any further contraction. The muscles then soften and relax.

![Graph showing temperature, rigor, lividity, and decomposition over time.](image)

6 12 15 24 36 42 48
Time After Death (Hours)

**Fig. (7-2).** Chart showing the major changes to estimate time since death.

The Order of Appearance of Rigor: All muscles of the body, both voluntary and involuntary are affected. It first appears in involuntary muscles; the myocardium becomes rigid in an hour. It begins in the eyelids, lower jaw and neck and passes upwards to the muscles of the face, and downwards to the muscles of the chest, upper limbs, abdomen, lower limbs and lastly in the fingers and toes. Such a sequence is not constant, symmetrical or regular. In individual limbs, it disappears in the same order in which it has appeared.

Rigor is tested by trying to lift the eyelids, depressing the jaw, and gently bending the neck and various joints of the body. Note the degree (complete, partial or absent) and distribution. Before rigor mortis develops, the body can be moved to any posture, and the rigor will fix in that posture. When rigor is developing, the extremities can be moved and the rigor, temporarily overcome, develops later and fixes the extremities in their new position, although the rigidity will be less than other symmetrical groups, which have not been disturbed.

Time of Onset: In India, it begins one to two hours after death and takes further one to two hours to develop. In temperate
countries, it begins in three to six hours and takes further two to three hours to develop.

Duration of Rigor Mortis: In India, usually it lasts 24 to 48 hours in winter and 18 to 36 hours in summer. It lasts for 2 to 3 days in temperate regions. These times are variable.

Conditions Altering the Onset and Duration:

1. Age: Rigor does not occur in a foetus of less than seven months, but is commonly found in stillborn infants at full term.

2. Nature of Death: In deaths from diseases causing great exhaustion and wasting, e.g., cholera, typhoid, tuberculosis, cancer, etc. and in violent death as by cut-throat, firearms electrocution, lightning, the onset of rigor is early and duration is short. In strychnine and other spinal poisons, the onset is rapid and the duration longer.

3. Muscular State: The onset is slow and the duration long in case where muscles are healthy

4. Atmospheric Conditions: The onset is slow and duration long in cold weather. The onset is rapid due to heat, because of the increased breakdown of ATP but the duration is short.

Medico-legal Importance: (1) It is a sign of death. (2) Its extent helps in estimating the time of death. (3) It indicates the position of the body at the time of death

Conditions Simulating Rigor Mortis:

1. Heat Stiffening: When a body is exposed to temperatures above 65°C. a rigidity is produced, which is much more marked than that found in rigor mortis. It is seen in deaths from burning, high voltage electric shocks and from falling into hot liquid. Heat causes stiffening of the muscles, because the tissue proteins are denatured and coagulated as in cooking.

2. Cold Stiffening: When a body is exposed to freezing temperatures, the tissues become frozen and stiff, due to freezing of the body fluids and solidification of subcutaneous fat simulating rigor. The body is extremely cold and markedly rigid.

3. Cadaveric Spasm or Instantaneous Rigor: Cadaveric spasm (cataleptic rigidity) is a rare condition. In this, the muscles that were contracted during life become stiff and rigid immediately after death without passing into the stage of primary relaxation. It occurs especially in cases of sudden death, excitement, fear, severe pain, exhaustion, cerebral haemorrhage, injury to the
nervous system, firearm wound of the head, convulsant poisons, such as strychnine, etc. Occasionally, the whole body is affected as seen in soldiers shot in battle, when the body may retain the posture which it assumed at the moment of

**Table (7-2). Difference between rigor mortis and cadaveric spasm**

<table>
<thead>
<tr>
<th>Trait</th>
<th>Rigor mortis</th>
<th>Cadaveric spasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Production:</td>
<td>Freezing and exposure to temperature above 65°C will produce rigor. Known. Nil.</td>
<td>Cannot be produced by any method after death. Not clearly known. Sudden death, excitement, fear, exhaustion, nervous tension, etc. Instantaneous. Usually restricted to a single group of voluntary muscles. Marked: very great force is required to overcome it. Poes not occur. Warm. Muscles respond. Indicates mode of death, i.e., suicide, homicide, or accident.</td>
</tr>
<tr>
<td>(2) Mechanism:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(3) Predisposing factors:</td>
<td></td>
<td></td>
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<tr>
<td>(4) Time of onset:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(5) Muscles involved:</td>
<td></td>
<td></td>
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<tr>
<td>(6) Muscle stiffening:</td>
<td></td>
<td></td>
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<tr>
<td>(7) Molecular death:</td>
<td></td>
<td></td>
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<td>(8) Body heat:</td>
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<td></td>
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<tr>
<td>(9) Electrical stimuli:</td>
<td></td>
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<tr>
<td>(10) Medico-legal importance</td>
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</tbody>
</table>

**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.

*Fig. (7-3).* A case of suicidal cut-throat. The knife is firmly held in cadaveric spasm.death.
**PUTREFACTION**

Putrefaction or decomposition is the final stage following death, produced mainly by the action of bacterial enzymes, mostly anaerobic organisms derived from the bowel. 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 post-mortem clots and of fresh thrombi and emboli, disintegration of tissue and gas formation in blood vessels and tissue spaces. The other organisms include Streptococci, Staphylococci, B. Proteus, B. Coli., B. aerogenes capsulatus, diphtheroids, etc. 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 hydrolysages the lecithin which is present in all cell membranes including blood cells, and is responsible for the post-mortem haemolysis of blood. It begins immediately after death at the cellular level, which is not evident grossly. There is progressive breakdown of soft tissues and the alteration of their proteins, carbohydrates and fats. 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.

**Autolysis:** Soon after death, cell membranes become permeable and breakdown, with release of cytoplasm containing enzymes. The proteolytic, glycolytic and lipolytic action of ferments leads to autodigestion and disintegration of organs, and occurs without bacterial influence. Viscera which contain more enzymes undergo autolysis faster than organs with lesser amounts of enzymes, e.g. pancreas autolyses before the heart. The earliest autolytic changes occur in parenchymatous and glandular tissues and in the brain.

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.

(1) **Colour Changes:** Bacteria spread directly. At an early stage of putrefaction, haemoglobin diffuses through the vessels and stains the surrounding tissues a red or reddish-brown colour. 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, which lies fairly superficially, and where the contents of the bowel are more fluid and full of bacteria. The colour results from the conversion of haemoglobin of blood into sulphmethaemoglobin by the hydrogen sulphide formed in the large intestines and escaping into the surrounding tissues. The colour appears in 12 to 18 hours in summer and in one to two days in winter.

*Fig. (7-4).* Putrefactive network (marbling).

*Fig. (7-5).* Blisters of putrefaction.

The green colouration then spreads over the entire abdomen, external genitals and then patches appear successively on the
chest, neck, face, anus and legs. The putrefactive bacteria spread most easily in fluid and tend to colonise the venous system. The superficial veins especially over the roots of the limb, thighs, sides of the abdomen, shoulders, chest and neck are stained greenish-brown or reddish-brown due to the haemolysis of red cells, which stains the wall of the vessel and infiltrates into the tissue, giving a marbled appearance. This occurs soon, but is prominent in 36 to 48 hours. Marbling is often first seen in the vessels close to the caecum and sigmoid colon.

![Image](image.png)

**Fig. (7-6).** Advanced decomposition showing marked gaseous swelling of face, eye balls and tongue.

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 and produces the appearance of congestion. 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: death complicated proteins and carbohydrates being split into simpler compounds of aminoacids, ammonia, CO₂, CO₂r hydrogen sulphide, phosphorated hydrogen, methane and mercaptans. Gases collect in the intestines in six to twelve hours in summer, and the abdomen becomes tense and distended. On opening the abdomen, the gas escapes with a loud explosive noise. At about the same time, the eyeballs become soft, the cornea becomes white and flattened or
compressed. Due to the presence of gas in the abdomen, the diaphragm is forced upwards compressing the lungs and heart, and bloodstained froth exudes from the mouth and nostrils (post-mortem purge), which can be mistaken for the bleeding following antemortem injury. Pressure of the gases may force food from the stomach into the fauces, and this may fall into the larynx. The maggots have proteolytic enzymes and penetrate the skin fairly rapidly. This allows air to enter under the skin and more maggots into the body cavity. The activities of maggots may raise the temperature to something near or even above that of normal body heat.

Gas bubbles accumulate in the tissues, causing crepitant, sponge-like feeling which soon begins to distend the body. From 18 to 36 or 48 hours after death, the gases collect in the tissues, cavities and hollow viscera under considerable pressure, and the features become bloated and distorted. Swelling due to gases is most marked in the face, genitalia and abdomen. Sometimes limbs are relatively free of putrefaction when changes are marked in the face and trunk. The subcutaneous tissues become emphysematous, due to which even a thin body appears obese. The breasts, scrotum and penis are greatly distended. The tongue is forced out between the swollen and discoloured lips. The sphincters relax and urine and faeces may escape. The gas formation in the blood vessels may force blood stained fluid, air or liquid fat between the epidermis and dermis forming small blisters. Blisters are formed first on the lower surfaces of trunk and thighs, where tissues contain more fluid due to hypostatic oedema. Pink-red serous fluid. The anus and uterus may prolapse after two to three days, and post-mortem delivery of a foetus may take place.

3) Liquefaction of Tissues: Colliquative putrefaction begins from five to ten days or more after death. The tissues become soft, loose and are converted into a thick, semi-fluid, black mass and are separated from the bones. Decomposition may differ from body to body, from environment to environment and from one part of the same body to another. Sometimes, one part of the body may be mummified, while the rest may show liquefying putrefaction.

Skeletonisation: The time required for skeletonisation varies considerably. 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 a deeply buried body, the lower temperature, the exclusion of air, absence of animal life, etc., markedly delay decomposition. In India, an uncoffined buried body is reduced to a skeleton within about a year. Acidic soil may cause decay in about 25 to 100 years. 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 diffusent 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.

As a general rule, the organs show putrefactive changes in the following order. (1) Larynx and trachea. (2) Stomach, intestines and spleen. (3) Liver, lungs. (4) Brain. (5) Heart. (6) Kidney, bladder, uterus. (7) Skin, muscle, tendon. (8) Bones.

UTERUS: The virgin uterus is the last organ to putrefy.

Conditions Affecting the Rate of Putrefaction: (A) External: (1) Temperature: Putrefaction begins above 10°C and is optimum between 21°C and 38°C. A temperature increase of 10°C usually doubles the rate of most chemical processes and reactions. It is arrested below 0°C and above 48°C. Advanced putrefaction may be seen within 12 to 18 hours in summer. A frozen body will not undergo decomposition.

(2) Moisture: For putrefaction moisture is necessary.

(3) Air: Free access of air hastens putrefaction, partly because the air conveys organisms to the body.

(4) Clothing: Initially clothing hastens putrefaction by maintaining body temperature above that at which putrefactive organisms multiply for a longer period. Clothes prevent the access of airborne organisms, flies, insects, etc., which destroy the tissues.

(5) Manner of Burial: If the body is buried soon after death, putrefaction is very much delayed. Putrefaction is rapid in a body buried in a damp, marshy or shallow grave without clothes or coffin, because the body is exposed to constant changes of temperature.
(B) Internal: (1) Age: The bodies of newborn children who have not been fed, decompose very slowly because the bodies are normally sterile.
   (2) Sex: Sex has no effect.
   (3) Condition of the Body: Fat and flabby bodies putrefy quickly than lean bodies.
   (4) Cause of Death: Bodies of persons dying from septicaemia, peritonitis, inflammatory and septic conditions, general anasarca, asphyxia, etc., decompose rapidly. Putrefaction is delayed after death due to wasting disease, anaemia, debility, poisoning by carbolic acid, zinc chloride, strychnine and chronic heavy metal poisoning, due to the preservative action of such substances on the tissues or their destructive or inhibitive action on organisms, which influence decomposition.
   (5) Mutilation: Bodies in which there are wounds, or which have suffered from other forms of violence before death, putrefy rapidly owing to the ease with which organisms gain access to the damaged tissues.

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

Putrefaction in Water: A body decomposes in air twice as rapidly as in water, and eight times as rapidly as in earth. The rate of putrefaction is slower in water than in air. Putrefaction is more rapid in warm, fresh water than in cold, salt water.

ADIPOCERE (Saponification) Adipocere (cire=wax) is a modification of putrefaction. In this, the fatty tissues of the body change into a substance similar to soaps, known as adipocere. It is seen most commonly in bodies immersed in water or in damp, warm environment. 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 hydroxy-stearic acid, and a mixture of these substances forms 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, which facilitates hydrolysis and hydrogenation. The water required for the hydrolysis is obtained mainly from the body tissues, which therefore become more and more dehydrated. Water helps to remove glycerine which is formed during hydrolysis of the fats. Adipocere is delayed by cold and hastened by heat. Fresh adipocere is soft, moist, whitish, translucent, and greasy, resembling pale, rancid butter. After some years it becomes dry, hard, cracked, yellowish and brittle.

Distribution: It forms in any site where fatty tissue is present. It is formed first in the subcutaneous tissues. Occasionally, the whole body may be affected. The face, buttocks, breasts and abdomen are the usual sites. The limbs, chest wall, or other parts of the body may be affected, but sometimes the entire body is converted into adipocere. Fatty tissue between the fibres of skeletal muscle and in the myocardium and in the substance of the liver, kidney, etc., is also converted into adipocere.

Time Required for Adipocere Formation: In temperate countries, the shortest time for its formation is about three weeks in summer, when it occurs to a certain extent. Complete conversion in an adult limb requires at least three to six months. Fat bodies and the bodies of mature newborn children form adipocere readily, but foetuses under seven months do not show this change. In India, it has been observed within three days. Adipocere may persist for years or decades.

Medico-legal Importance: (1) When the process involves the face, the features are well preserved, which help to establish the identity. (2) The cause of death can be determined, because injuries are recognised. (3) The time since death can be estimated.
MUMMIFICATION

Fig. (7-8). Mummified forearm.

It is a modification of putrefaction. Dehydration or drying and shrivelling of the cadaver occurs from the evaporation of water, but the natural appearances and features of the body are preserved. It begins in the exposed parts of the body like face, hands and feet and then extends to the entire body including the internal organs. The skin may be shrunken and contracted, dry, brittle, leathery and rusty-brown in colour. Mummified tissues are dry, leathery and brown in colour. Mummification may be partial in some cases, with only limbs or head or trunk being affected. The internal organs become shrunken, hard, dark-brown and black and become a single mass or may disappear. The entire body loses weight, becomes thin, stiff and brittle. If a mummified body is not protected, it will break into fragments gradually, become powdery and disintegrate, but if protected, it may be preserved for years. A mummified body is practically odourless. The time required for complete mummification of a body varies from three months to a year and is influenced by the size of the body, atmospheric conditions and the place of disposal.

Two factors are 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 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.

Medico-legal 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. By this process proteins are coagulated, tissues are fixed, organs are bleached and hardened and blood is converted into a brownish mass. 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 produces a chemical stiffening similar to rigor mortis, and normal rigor does not develop. Embalming rigidity is permanent. 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. Thrombi and emboli will be dislocated and washed away.

ESTIMATION OF POST-MORTEM INTERVAL

The interval between death, and the time of examination of a body is known as post-mortem interval.

The exact time of death cannot be fixed by any method, but only an approximate range of time of death can be given, because there are considerable biological variations in individual cases. The longer the post-mortem interval, the wider is the range of estimate, i.e. the less accurate the estimate of the interval.

The points to be noted are: (1) Cooling of the body. (2) Post-mortem lividity. (3) Rigor mortis. (4) Progress of decomposition, adipocere and mummification.

(5) Entomology of the Cadaver: Flies may deposit their eggs on the fresh corpse, between the lips or the eyelids, in the nostrils, genitalia, or in the margins of a fresh wound, within a few minutes after death, and in some cases even before death during the agonal period. When skin decomposition begins, the eggs can be deposited anywhere. In eight to twelve hours in summer, larvae or maggots are produced from the eggs, which crawl into the interior
of the body and produce powerful proteolytic enzymes, and destroy the soft tissues. The maggots burrow under the skin and make tunnels and sinuses which hasten putrefaction by allowing air and bacteria. The maggots become pupae in four to five days, and the pupae become adult flies in three to five days. Body lice usually remain alive for three to six days, after death of a person.

(7) Cerebrospinal Fluid: All the chemical methods for estimating the time since death are temperature-dependent. Examination of cerebrospinal fluid obtained by cisternal puncture, which should be free from blood, is useful in adults above 15 years of age. Lactic acid concentration rises from the normal 15 mg. % to over 200 mg. % in 15 hours following death. Non-protein nitrogen shows a steady increase from 15 to 40 mg. % in 15 hours.

Aminoacids rise evenly from one to 12 mg. % in 15 hours. Using these values, the time of death can be estimated during the first 15 hours up to ± 3 hours.

(8) Blood: Potassium, phosphorus and magnesium levels rise after death, and sodium and chloride levels decrease. Non-protein nitrogen, aminoacid nitrogen, ammonia, lactic acid and bilirubin levels rise after death. The enzymes acid phosphatase, alkaline phosphatase, amylase, serum glutamic oxaloacetic transaminase, and lactic dehydrogenase increase after death.

(12) Hair: Hair does not grow after death; the contraction of the skin towards the hair roots gives the illusion of growth.

**FORENSIC BALLISTICS**

A firearm is any instrument which discharges a missile by the expansive force of the gases produced by burning of an explosive substance.

**Forensic Ballistics** is the science dealing with the investigation of firearms, ammunition and the problems arising from their use.

**Classification:** (I) Rifled weapons: (1) Rifles: (a) Air and gas-operated rifles. (c) Military and sporting rifles. (2) Single-shot target-practice pistols. (3) Revolver. (4) Automatic pistols. (II) **Smooth-bored weapons** (shotgun). Cylinder bore. (2)

**Rifled Arms:** The bore is scored internally with a number of shallow spiral “grooves”, varying from two to more than 20, the
most common being six, which run parallel to each other but twisted spirally, from breech to muzzle. These grooves are called “rifling” and the projecting ridges between these grooves are called “lands” (fig. 8-18). Riflings vary in number, direction, depth and width. When the bullet passes through the bore, its surface comes into contact with the projecting spirals which give the bullet a spinning or spiralling motion. Rifling gives the bullet a spin, greater power of penetration, a straight course and prevents it from unsteady movement as it travels in the air. Rifled firearms are divided into: (1) Low velocity (up to 360 metres per second). (2) Medium velocity (360 to 750 m/s). (3) High velocity (900 m/s and above).

**Cartridge**: It consists of a metal cylinder with a flat. The primer cup (percussion cap) is fitted in a circular hole, usually in the centre of the base and has a flash hole in the centre which communicates with the powder space inside. The metal cylinder or cartridge case is elongated, and its distal end tightly grips the base of the bullet (projectile or missile). The gunpowder lies between the detonator and the bullet.

**Primers**: Centrefire rifle and pistol primers are small metal cups usually held in place in the cartridge head primer pocket by friction. The primer cup contains the priming mixture and an anvil so placed that the blow of firing pin on the primer cup crushes the priming mixture against the anvil centre and burns it, which then flashes through the flash hole, and ignites the powder charge. The priming mixture contains lead peroxide, lead styphnate, tetrazene, barium nitrate, antimony sulphide pentaerythritol tetranitrate, etc.

**Powders**: (1) **Black Powder**: It consists of potassium nitrate 75%; sulphur 10%; and charcoal 15%. It is designated as FG, FFG, FFFG, etc., depending on the size of the grains. The powder grains are black, coarse or fine, without any particular shape. It burns with production of much heat, flame and smoke. Fine grains travel 60 to 90 cm. or more. One gram of powder produces 3,000 to 4,500 c.c. of gas. The gas consists of CO, CO₂, nitrogen, hydrogen sulphide, hydrogen, methane, etc., all at a very high temperature.

(2) **Smokeless Powders**: It consists of nitrocellulose (gun cotton), or nitroglycerine and nitrocellulose (double-base). They produce much less flame and smoke and are more completely
burnt than black powder. One gram produces 12,000 to 13,000 c.c. of gases. The colour varies from bright orange to bluish-black, and in shape from minute globules, flakes, square, rectangular, irregular discs, cylinders to longer threads. Semi-smokeless powder is a mixture of 80% of black and 20% of the smokeless type.

"Mechanics of Bullet Wound Production: (1) Bullet Velocity: A bullet's ability to wound is directly related to its kinetic energy \( E=mv^2/2 \) at the moment of impact.

(2) Tissue Density: The greater the tissue density, the greater is the amount of energy discharged by the bullet's passing through it. A bullet may cause slight damage to the soft tissues, but the same bullet, at the same speed can produce extensive comminution of the bone.

(3) Hydrostatic Forces: Hydrostatic forces cause excessive degree of destruction. When a bullet passes through a fluid-distended hollow organ, e.g., food-filled stomach, urine-filled bladder, CSF-filled ventricles of the brain, or a heart chamber distended with blood in diastole, produces extensive lacerations due to the explosive displacement of the liquid in all directions.

WOUNDS FROM REVOLVERS AND AUTOMATIC PISTOLS

Entrance Wound (in-shot wounds): They may be classified on the distance of the muzzle of a firearm from the body; contact shot, close shot, near shot and distant shot. The flame extends up to 8 cm; smoke up to 30 cm. and unburnt and partially burnt powder grains and small metallic particles up to 60 to 90 cm. The amounts of smoke, flame and powder grains and the distances to which they will be carried will vary depending upon the type of gun powder used, the amount of powder load, the size and weight of the projectile, the tightness of fit between the projectile and gun barrel and the type of firearm.

(1) Contact Shot: The wound is large, shows cavitation, and triangular, stellate, cruciate or elliptic due to the expansion of the liberated gases in the skin and tissues, which show laceration. The margins are contused and everted due to gases coming out of the
entering wound under pressure. There is no burning, blackening and tattooing around the wound of entrance. The area immediately around the perforation is abraded, and this thin rim of abrasion is usually covered with powder residue. In firm contact the resulting wound is similar to that from a shotgun. In some contact wounds, the imprint of the muzzle of the gun is found as patterned abrasion on the skin around the wound. This results from the great distension of the subcutaneous tissues from the entry of gases which forces the surface against the muzzle. The mark may be an incomplete, indistinct bruise and occasionally a perfect imprint of the muzzle. Many muzzle impressions are not recorded due to the rapid removal of the weapon by recoil. In firm contact with the skin where the bone is not shallowly situated, the ever expanding gas continues to penetrate deeper, to be scattered in the soft tissues of the body. In contact shot, the muzzle blast and the negative pressure in the barrel following discharge may suck blood, hair, fragments of tissues and cloth fibres several cm. back inside the barrel called "back spatter". Sometimes, blood may soil the hand of the person firing the gun. In loose contact or near contact shot, some of the gases escape with the resulting scattering of the muzzle blast and an unusual arrangement of soot is seen on the skin known as corona. The corona consists of a circular zone of soot deposit surrounding the bullet defect, but separated from it by a band of skin without a deposit of soot. This is due to the gas expanding about the muzzle, first at a velocity too high to allow for the settling out of soot, with a subsequent loss in velocity at a short distance from the muzzle, allowing the soot to finally deposit on the skin. The blast effect is not as marked as in tight contact, and splitting of the wound edges does not occur Evidence of burning is noted on microscopic examination in the edges of the contact and near-contact bullet wounds due to the flame of muzzle blast. Singeing of the hair may also be seen. The discharge from the muzzle, i.e., gases, flame, powder, smoke, and metallic particles are blown into the track taken by the bullet through the body. The powder residue is usually grossly visible in the subcutaneous and deeper areas. The entrance track is blackened by powder and smoke and seared and charred by flame.

Head wounds appear, as very large explosive type of injury with bursting fractures. The skin wound is large and irregular
because of the expansion of gases between the scalp and the skull which causes eversion and splitting of the skin at the margins of the entrance wound. Fissured fractures often radiate from the circular defect due to the considerable sudden expansion resulting from the muzzle blast. The margins of the wound of entry, subcutaneous tissues and muscles around the track of the bullet may be bright pink due to the presence of CO. Abdominal wounds show cavitation because of the blast effect.

(2) Close Shot: This term is applied when the victim is within the range of the flame, i.e., 5 to 8 cm. The term "point blank" is used when the range is very close to or in contact with the surface of the skin. The entrance wound is circular with inverted edges, but the rebounding gases may level up or even evert the margins. The skin is burnt with singeing of the hair. The skin surrounding the wound is hyperaemic and shows some bruising, burning, blackening and tattooing. The blackening can be wiped off the skin by a wet cloth, but the tattooing cannot be wiped off. Carboxyhaemoglobin will be present in the wound track in diminishing concentrations. Usually, as the distance between the muzzle and the target increases, the pattern of soot or powder on the target increase in diameter and the density of particle deposition decreases. In handguns, up to 15 cm. from the muzzle, abundant gunpowder and diminishing amount of soot are deposited on the target. Hair in the surrounding area may be clubbed, swollen at intervals by heat, or burnt. Abraded collar and grease or dirt collar are present. The internal injuries are almost same as in the case of contact shot.

(3) Near Shot: This term is applied when the victim is within the range of powder deposition, and outside the range of flame, i.e., up to 90 cm. If the discharge occurs at a distance of about 15 cm., the lacerating and burning effects of gases are usually lost due to the dispersion cooling of the gases before they reach the skin. The entrance wound is seen as a round hole about the size of the bullet with a bruised and inverted margin and a zone of blackening and tattooing. As the distance increases, the intensity decreases and blackening and tattooing is spread out over a large area, and there is no singeing of the skin. Abrasion and grease collar are present.
(4) Distant Shot: The entrance wound is smaller than the missile due to the elasticity of the skin, circular, and margins are inverted. Burning, blackening and tattooing are not seen. The skin adjacent to the hole shows two zones, the inner of grease collar and the outer of abraded collar. The calibre of a bullet cannot be determined, if it strikes the skin surface obliquely.

The Abrasion Collar (marginal abrasion): As the bullet strikes the skin, it first indents and then stretches the skin surface, so that perforation takes place through a tense area. After the bullet has perforated the skin, the elasticity of the skin causes the skin defect to contract. The skin is abraded (abrasion collar) around the hole due to rubbing of the gyrating body of the bullet against the inverted epidermis and heat of the bullet (Fig.8-32). A black coloured ring "grease or dirt collar" (bullet wipe) is seen as a narrow ring of skin, lining the defect, and is sharply outlined. This is caused from the removal of substances from the bullet as it passes through the skin, i.e., bullet lubrication, gun oil from the interior of the barrel, lead from the surface of the bullet, barrel debris, etc. Dirt collar is less common if the bullet is jacketed. By contrast, soot is dark in the centre and fades towards the periphery. The abrasion collar surrounds the dirt collar. The abraded collar becomes reddish brown as it dries. Some contusion is present in abraded collar, and as such, it is also called "contusion collar". The abrasion and contusion collar is one to three mm. wide. These two features are proof of an entrance wound. Irregular and occasionally patterned abrasion collar is sometimes produced by coarse article of clothing scraping on the skin.

Skull: In the skull, the wound of entrance shows a punched-in (clean) hole in the outer table. The inner table is unsupported and a cone-shaped piece of bone is detached forming a crater that is larger than the hole on the outer table, and shows bevelling (sloping surface) (Fig.33&34). Fissured fractures often radiate from the defect. Pieces of bone from wound of entrance are often driven into the cranial cavity and may establish the bullet track. At the point of exit, a punched-out opening is produced in the inner table and bevelled opening on the outer table. There are often fissured, sometimes comminuted fractures radiating from the central hole.

The same appearance is seen in sternum, peivis, ribs, dentures and thumbnails.
**Puppe's Rule**: It can determine the sequence of shots, when several bullets have struck the cranium. This rule is applicable to any multiple blunt force, causing skull fractures. This rule has been developed by Madea in relation to bullet injuries. The test depends on the observations of the fracture lines either when they intersect each other or when they intersect a cratered lesion, so that one can determine which crack or defect must have been formed first.

**The Wounding Power of Bullets**: The size and velocity of the bullet are the two most important factors. The wounding power of a bullet is proportional to its mass, multiplied by the cube of its velocity. Another factor is the density of the tissue, e.g., destruction is greater in dense tissue such as bone than in soft tissues. A bullet travelling at high velocity produces a clean, circular punched-out aperture or slit and usually perforates the body. It is not deviated from its path by striking a bone, but may cause its comminution or splintering. A bullet of low velocity causes contusion and laceration of the margins. Large bullets cause greater damage than small ones. Round bullets produce larger wounds than conical ones.

**Clothes**: If the shot is through a clothed surface, examination of clothing only can indicate its range. In a contact shot, the clothing usually shows a cross-shaped perforation, and the skin around the bullet hole and the deeper layers of the cloth are blackened. In close shots, the clothing may absorb or filter out all of the products of discharge except the bullet. An entrance hole in clothing, if made by a lead or full metal-jacketed bullet, may produce a grey to black rim known as "bullet wipe" (grease, soot or debris from the barrel of the gun). Sometimes, pieces of cloth are carried into the wound of entrance.

**Exit Wounds**: If the bullet fragments on impact, an exit wound may not occur. The bullet may be reduced to granules, and there may be difficulty to remove them from the body, even when identified by X-ray. Exit wounds may vary considerably in size and shape. They may be round, stellate, cruciate, elliptical, crescent shaped, or appear as linear lacerations or even incised wounds. In some cases, the entrance and exit wounds may look alike. The edges of the exit wound may be puckered or torn and everted and pieces of contused, haemorrhagic subcutaneous fat may protrude through the defect. The edges are free from signs of burning,
blackening or tattooing and there is no contusion or abrasion collar. If the skin at the exit wound is supported, the exit wound appears as a circular or nearly circular defect surrounded by a margin of abrasion resembling a wound of entrance (shored or supported exit wound). Many shored exit wounds are caused if a firm object, e.g., a belt, the waist band of trousers, etc., brassiere, collar and tie, is pressed against the body at the site of exit wound, or if the body is leaning against a hard surface such as a wall, back of a chair or the floor, mattress, bedding, or if the person was lying down. In such cases the skin, crushed by the exiting bullet, produces an irregular, lopsided and large abrasion around or adjacent to the wound ("shored" exit wound).

Alteration of Gunshot Wounds: The appearance of a gunshot wound can be altered by the following conditions: (1) Drying of margins of the wound opening. (2) Decomposition of the body. (3) Healing of the wound itself. (4) Interference by emergency care personnel. (5) Surgical operation. (6) Interference by non-professional personnel at scene of death. (7) Washing or cleaning of the wound after death.

X-ray Examination of Gunshot Wound Victims: It helps to (1) locate the bullet or pellets, (2) locate bullet fragments or jackets, (3) show the track of the bullet. Internal ricochet within the skull may be demonstrated, which helps to determine the direction of the fire, (4) determine the break up pattern of the bullet. This may also indicate the type of ammunition used, (5) determine defects in bone, (6) locate air embolism accompanying large vessel damage by the missile.

SEXUAL OFFENCES

RAPE

S.375, I.P.C.: A man is said to commit rape, if he has sexual intercourse with a woman (1) against her will, (2) without her consent, (3) with her consent when her consent has been obtained by putting her or any person in whom she is interested in fear of death, or of hurt, (4) with her consent, when the man knows that he is not her husband and that she has given consent because she believes that he is another man to whom she is lawfully married, (5) with her consent, when at the time of giving such consent, by
reason of unsoundness of mind or intoxication or the administration of any stupefying substance, she is unable to understand the nature and consequences of that to which she gives consent, (6) with or without her consent, when she is under 16 years of age.

**Exception:** Sexual intercourse by a man with his own wife (even against her will) is not rape, if she is above fifteen years of age.

**EXAMINATION OF THE VICTIM**

The objects of medical examination are: (l) To search for physical signs (injuries) that will corroborate the history given by the victim. (2) To search for, collect and preserve all physical (trace) evidence for laboratory examination. (3) To treat the victim for any injuries and against venereal disease or pregnancy, and prevention or lessening of permanent psychological damage. The scene of alleged offence may be examined if it appears desirable.

The police should advise the victim not to change clothes, bathe, or douche prior to the medical examination.

**General Procedure:** The following is an outline of a planned procedure. (1) The victim should not be examined without requisition from investigating police officer or the Magistrate. The Court or the police has no power of forcing a woman for medical examination against her will. (2) The written, witnessed consent of the woman, for examination, collection of specimens, taking of photographs, treatment, and for the release of information to the police, and if she is under twelve years, or of unsound mind, the consent of her parents must be taken in writing. (3) The victim should be identified by the escorting police constable, whose name and number should be recorded. Identification marks should also be noted. (4) The name of the victim and her parent, marital status, residence, occupation, time, date, year, place of examination and by whom requisition is given should be recorded. Date and time is important because the interval between the alleged incident and the examination is material. (5) The examination should be carried out without delay. Minor degrees of injury may fade rapidly, and swelling and tenderness of vulva may disappear in few hours. Detection of spermatozoa from the genital tract also diminishes with delay. (6) Statements of the victim and of others with her are recorded separately of: (a) preliminary affairs, (b) date, time and
place of alleged offence, (c) location: inside or outside, wet or dry weather, (d) number of alleged assailants, (e) alcohol or drugs involved with details, (f) restraints, or weapons and their use, (g) details or struggle or resistance: injuries sustained by victim (when, how and where on the body), (h) injuries sustained by the assailant, due to scratching, bites, etc., (i) exact relative positions of the parties, (j) type and number of sexual acts, (k) use of condoms or lubricants, (l) use and disposal of sanitary pads or tampons, (m) was any pain experienced either at the time of the incident or subsequently, (n) did ejaculation take place during the act, either within the vagina or outside, (o) the appearance of any discharge, (p) was there bleeding from the vagina, (q) calls for help, and (r) recent consenting intercourse if the alleged victim is a married woman, (s) whether consciousness was lost at any time during the attack, (t) details of events after the alleged assault: changed or washed clothing; bathed, doused, defaecated or urinated prior to the examination, washed, brushed or combed hair; alcohol or drugs taken, treatment taken, (u) the time of the first complaint, and if there was any undue delay, the reason for such a delay. The victim may complain of severe abdominal pain after a gang rape due to unaccustomed weight of many men atop her in a short time, or forceful sexual intercourse. This must be taken down verbatim. The degree of agreement of various statements will be strong proof of their truth or the contrary. It also indicates the position of any injury or bruising, which although present, may not be obvious externally on examination. (7) Previous history with regard to sexual experience, menses, vaginal discharge, venereal disease, pregnancies, pelvic operations, etc. should be recorded. (8) The victim is examined in the presence of a third person, preferably a female nurse or a female relative of the woman, whose name should be recorded. This is necessary to avoid himself being accused. (9) The age should be determined and the height and weight recorded. (10) The physical development should be noted in order to determine her capacity for struggle and resistance. In the case of children, bodily development, especially of the breasts and genitalia should be noted. (11) An attempt at undressing the woman should not be made, but she should be requested to undress herself. (12) If the victim is in menstrual period, a second examination should be
done after stoppage of menstruation. (13) Her general demeanour (distressed or calm, dishevelled, dazed or shocked, intoxicated, excited, agitated, withdrawn, tearful, cooperative or aggressive, etc.); emotional and mental state should be observed while she tells her story. Elevated pulse may reflect emotional stress or major trauma. (14) If it appears that she is under the influence of alcohol or drugs, it must be noted and sample of blood, and urine should be collected. (.15) The gait should be observed; whether she complains of pain on walking, on micturition or defaecation. The pain leads to guarded gait, the victim walking with legs apart and slow paces.

**Examination Proper:**

(1) **Clothes:** Find out whether the clothes are those worn at the time of assault, or changed. Ideally, each item of clothing should be removed by the patient in the presence of the doctor. The patient should be standing on a clean sheet of paper and anything that falls, e.g., earth, buttons, hair, fibres, gravel, leaves, etc. should be preserved. Each item of clothing should be examined for stains (blood, seminal, mud, earth, grease, grass, etc.), soiling, tears and loss of buttons, and the site and type of damage. Semen is often found on clothing, bedding, carpets, car seats, etc. It can also be found on almost any other article, depending on the movements of the victim and the suspect. Seminal stains will be found or underclothing due to drainage from the vagina, especially if an upright position is adopted soon after ejaculation has occurred. If the offence has been committed in an open place, corroboration can sometimes be obtained by finding grass, leaves, mud, etc. on the buttocks or on the back. The clothes should be dried, stored in a clean paper bag and sent to the laboratory. In certain cases, stains may be present on pieces of material or handkercheif used by the victim after assault for cleaning purposes. Suspicious stains should be preserved for chemical analysis. Vulval pads and vaginal tampons should be preserved, whether worn at or after the time of the incident. Clothes are very important in corroborating or contradicting her story. Foreign hair, fibres, etc., found on clothes or on the skin surface must be preserved and compared with those found on the accused.

(2) **General Examination:** The whole body must be examined for marks of violence, especially scratches or bruises
resulting from struggle as regards their appearance, extent, situation and probable age. Petechiae on the face or conjunctivae indicate partial asphyxia caused during forcible restraint or with intent to make the victim unconscious or silence her. Marks of violence, especially bruises and scratches may be found: (1) about the mouth and throat, produced while preventing her from calling for help. Bruising of the lips and even tearing of the inner aspect may be found, due to blows or rough handling, (2) about wrists and arms where the man seized her, (3) about the inner sides of thighs and knees caused by forcing her legs apart, (4) on the back from pressure on gravel or hard ground, (5) on breasts by rough handling, (6) true bite marks and love-bites may be usually found on the breasts, which are often manually squeezed and manipulated, causing discoid bruises of one to two cm. on any part, especially around the nipples, neck, shoulders, chest wall, and also on the lower abdomen and upper parts of thighs. The nipples may be bitten off. Suction lesions may appear as circular or oval areas of bruising, in which there are many intradermal petechial haemorrhages produced by sucking the skin into the mouth due to rupture of small vessels from reduced air pressure. The lips may produce semilunar marks at the periphery, and teeth may produce indentation or abrasions. Marks of general violence are likely to be found in one-third cases. Absence of general injuries may be due to: (1) Submission of the victim due to fear of injury or death, etc. (2) The force used or the resistance offered is insufficient to produce an injury. (3) Bruises may not be noticed for 48 hours following the assault. (4) A delay in reporting the incident during which minor injuries will fade or heal. Genital injuries are present in one-fifth cases because: (1) The alleged sexual act may consist of only rubbing or touching the genitalia. (2) The victim is sexually experienced. (3) The elasticity of genitalia and hymen in a post-pubertal female. (4) The use of lubricants.

The extent and nature of the general injuries should correspond to the victim’s description of the assault. If the throat has been gripped, or if a severe blow is struck on the head, the victim’s capacity for resistance becomes greatly reduced. The age of injuries should be noted to see whether they correspond to the alleged time of offense. The woman usually scratches the assailant during the struggle, and any damaged fingernails should
be noted and the debris under the nails should be removed and examined for epidermal cells, blood, fibres, etc.

(3) Hair: The pubic hair should be combed out, as non-matching male pubic hair and foreign material may be present. In the case of the deceased victim, fifteen to twenty pubic hairs are pulled with forceps and placed in a separate envelope, so that root characteristics are available for comparison. In the living victim, fifteen to twenty hairs are cut, not pulled. (4) Seminal Stains: If the pubic hair is matted, the entire matted hair should be cut away as close to the skin as possible. Swabs must be taken from the area of the introitus and perineum before hymen is examined.

(4) Blood Stains: The presence or absence of blood stains about the vagina and legs should be noted. Determine whether such stains could be due to menstruation. Profuse bleeding from the vagina will wash out all the seminal contents and spermatozoa are not found.

(5) Venereal Disease: The presence of any discharge due to gonorrhoea, or inflammation of the parts, or signs of syphilis, should be noted. The degree of normal cleanliness of woman should be noted. In unclean woman, there may be superficial areas of erythema, irritation and sometimes abrasions. Redness from chronic inflammation or due to irritation by a chronic discharge must be differentiated from the effect of recent injury. Other sexually transmitted diseases are: Chlamydia, trichomoniasis, herpes simplex, HIV and human papilloma virus.

(6) Special Local Examination: The Genitals: The woman is placed on a table in good light, with her legs drawn up and widely open. If the separation of thighs is painful, cocaine solution should be applied to the parts. The examination of genitalia should be thorough, for much depends upon it.

Rape on Virgin: The labia are separated by gentle traction to examine the hymen. Rupture of hymen occurs with the first intercourse which is the main evidence of rape in the virgin. Usually tears due to digital penetration or insertion of tampons do not extend to the margin of the hymen, while tears due to penile penetration extend to the margin, but tears caused by full finger penetration can extend to the hymenal margin. Several hymenal lacerations indicate first sexual intercourse. Tearing usually occurs posteriorly at the sides, in the 4 or 8 'O clock position, or in the
middle line of the hymen. More than one tear may occur. One deep tear may be seen at 6 o'clock position or a number of tears usually in the posterior half of the membrane. Soon after the act, the margins of the torn hymen are sharp and red which bleed on touch, the tissues round about them are tender. After three to four days, the edges of tear are congested and swollen, which completely heal in a week, but they do not unite. Rupture of the hymen due to sudden stretching can be caused by agents other than the penis, such as fingers, and therefore evidence of local injury is not proof of penetration. Frequently, in the absence of frank hymenal tearing, there is abrasion and bruising of the hymen and the vaginal orifice.

The labia may be red and inflamed with slight oedema of the vaginal introitus, if it is the first sexual act, or if there is disproportion between the male and female genitalia. Injury to labia is not common, but fingernail scratches may be present on the labia, particularly the labia minora and upper parts of thighs. Swelling and tenderness of the labia minora may indicate sexual activity. Redness of the labia minora may be due to uncleanliness. Swelling, and congestion of the mucosa at the introitus, the clitoris and the labia minora are caused by genital stimulation, but they may also be caused by digital stimulation or masturbation. These signs usually fade in one to two hours. The posterior commissure may be ruptured, especially if there is disparity in size between the male and female organs. Abrasion and bruising of the hymen and vaginal office may be seen in the absence of hymenal tear, due to digital or penile penetration. Bruising of this nature is more consistent with penetration of the penis, than with digital penetration. Abrasion of the vaginal mucosa is more frequent in digital penetration. In most young women, a finger may be passed into vagina although the hymen is intact, which is felt as a constricting ring round the tip of the finger. Vaginal examination helps the examiner to assess elasticity of the hymen and to determine the degree of penetration which would be possible without its rupture. If the vaginal opening is enough to admit two fingers easily, sexual intercourse is possible without rupture of hymen.
A virgin \textit{(virgo intacta)} is a female who has not experienced sexual intercourse. \textbf{Defloration} means loss of virginity. The question of virginity arises in case of (1) nullity of marriage, (2) divorce, (3) defamation, and (4) rape.

Legally, marriage is a contract between man and woman, which implies physical union by coitus. Divorce means, dissolution of previously valid marriage.

\textbf{Divorce} means dissolution of previously valid marriage.

\textbf{Hymen:} The hymen is a fold of mucous membrane about one mm. thick, situated at the vaginal outlet. The average adult hymen consists of folds of membrane having annular or crescentic shape, the broadest part lying posteriorly. The different types of hymen are: (1) \textbf{Semilunar} or crescentic \textit{(commonest type)}. (2) \textbf{Annular}. (3) \textbf{Infantile}: several openings. (5) \textbf{Vertical}: the opening is vertical. (6) \textbf{Septate}: two lateral openings occur side by side, separated partially or completely by thin strip of tissue. (7) \textbf{Imperforate}: no opening.

The margin of the hymen is sometimes fimbriated (wavy or undulating) and shows multiple notches which may be mistaken for artificial tears. Natural notches are usually symmetrical, occur anteriorly, do not extend to the vaginal wall and are covered with mucous membrane. Tears caused by sexual intercourse or by foreign body are usually situated posteriorly at one or both sides, or in the midline, and usually extend to the vaginal wall and are not covered with mucous membrane.

\textbf{Causes of Rupture of Hymen:} (1) An accident, e.g., a fall on a projecting substance or by slipping on the furniture or fence or while playing at seesaw. In these cases tearing of the perineum occurs and usually injuries on other parts of the body will be seen. Such hymenal tears are never associated with abrasion and bruising of the margins. Hymen does not rupture by riding, jumping, dancing, etc. (2) \textbf{Masturbation}. (3) \textbf{Surgical operation} and gynaecological examination. (4) Foreign body, e.g., sola pith
introduced into vagina for rendering very young girls fit for sexual intercourse \textit{(aptae viris)}. (5) \textbf{Ulceration} from diphtheria, fungus or other diseases. (6) \textbf{Scratching} due to irritation of the parts from lack of cleanliness. (7) Sanitary \textbf{tampon} may sometimes rupture the hymen.

**Breasts:** In a virgin the breasts are firm, elastic and hemispherical with a small undeveloped nipple surrounded by an areola which is pink in fair complexioned women and dark-brown in dark women. The breasts become large and flabby by frequent handling and sexual intercourse, but are not affected by single act of coitus. Occasionally, milk may be found in the breasts of virgins.

**Medico-legal Aspects:** The presence of unruptured hymen is a presumption, but is not an absolute proof of virginity. The diagnosis of virginity is difficult and in many cases a physical examination of the genital organs may not be helpful. With an intact hymen, there are true virgins and false virgins. The hymen is present always in a virgin in some form or other, but very rarely may be absent congenitally. The principal signs of virginity are: (1) An intact hymen. (2) A normal condition of the fourchette and posterior commissure. (3) A narrow vagina with rugose walls. These signs taken together, may be regarded as evidence of virginity but taken singly they cannot be so regarded.

The hymen is usually ruptured at the time of the first coitus, and at first only presents a torn appearance. Hymen may not be ruptured even after repeated acts of coitus if it is loose, folded and elastic, or thick, tough and fleshy, which permit displacement, distortion and stretching without rupture. Cases have been recorded of women having sexual relations, of pregnant women and even prostitutes in whom the hymen was intact. In women who are used to coitus, and in those who have borne children, the hymen is destroyed and small, round, fleshy projections or tags, known as carunculae hymenales or myrtiformes are formid round the hymenal ring.

**PREGNANCY**

Pregnancy is the condition of having a developing embryo or foetus in the female, when an ovum is fertilized by a spermatozoon. It is most likely to occur between the ages of 14 and 45 years, but has been reported much earlier and later. The
question of pregnancy has to be determined in the following conditions. (1) When a woman pleads pregnancy to avoid attendance in Court as a witness. Pregnancy itself is not an excuse, unless it is so far advanced that delivery is likely to occur soon, or when she or the child is likely to suffer risk by such attendance. (2) When a woman sentenced to death, pleads that she is pregnant, to avoid execution. The High Court has the power to postpone the execution of death sentence until 6 months after delivery or to commute it (S.416. Cr.P.C). (3) When a woman feigns pregnancy soon after death of her husband to claim succession to estate. (4) To assess damages in a seduction or breach of promise of marriage case. When a woman blackmails a man and accuses that she is pregnant by him, to compel marriage. In allegations that an unmarried woman, widow or a wife living apart from her husband is pregnant. When a woman alleges that she is pregnant in order to get greater compensation when her husband dies through the negligence of some person. (8) When pregnancy is alleged to be motive for suicide or murder of unmarried woman or widow. (9) In cases of divorce, the woman may claim to be pregnant to receive more alimony. (10) In cases of alleged concealment of birth or pregnancy and infanticide.

**Diagnosis of Pregnancy:** The signs and symptoms are usually classified into three groups: (1) The presumptive signs. (2) The probable signs. (3) The positive signs.

(1) **Presumptive Signs:** (1) **Amenorrhoea:** This is the earliest and one of the most important symptom of pregnancy.

(2) **Changes in Breasts:** Breast changes are quite characteristic in primigravidas but are of less value in multiparas. A sense of tenseness and tingling in the breasts is frequent in early weeks. After the second month, breasts begin to increase in size and become nodular due to hypertrophy of the mammary alveoli. As they become still larger, the superficial veins are seen more distinct and enlarged, the nipples more deeply pigmented and more erectile and the areola which is pink in the virgin, gradually becomes dark-brown. Around the nipple, the sebaceous glands become enlarged by the end of second month to form small rounded dark coloured tubercles. **Colostrum** is secreted usually in the third month, which can be expressed from the breasts by gentle massage.
(3) **Morning Sickness** : It usually appears about the end of the first month and disappears 6 to 8 weeks later. Nausea and vomiting are usually present in the morning, and pass off in a few hours. **Quickening** : From about the 16th to 20th week, the pregnant woman feels slight fluttering movements in her abdomen, which gradually increase in intensity. These are due to movements of the foetus, and their first appearance is known as "quickening".

(5) **Pigmentation of the Skin** : The vulva, abdomen and axillae become darker due to the deposit of pigment, and a dark line extends from the pubis to beyond the umbilicus, the so-called **linea nigra**.

(6) **Changes in the Vagina** : The mucous membrane of the vagina changes from pink to violet, deepening to blue as a result of venous obstruction, after the fourth week. This is known as **Jackquemier's sign or Chadwick's sign**. The tissues become softer, the secretion of the mucus is increased, and pulsation can be felt at an early period.

(7) **Urinary Disturbances** : During the early weeks of pregnancy, the enlarging uterus exerts pressure on the bladder and produces frequent micturition.

(8) **Fatigue** : Easy fatigue is very frequent.

(9) **Sympathetic Disturbances** : Salivation, perverted appetite and irritable temper are common.

(II) **Probable Signs of Pregnancy** :

(1) **Enlargement of the Abdomen** : During pregnancy, abdomen gradually enlarges in size after the twelfth week.

(2) **Uterus** : **Hegar's sign** is positive at about the sixth week. If one hand is placed on the abdomen and two fingers of other hand in the vagina, the firm hard cervix is felt and above it the elastic body of the uterus, while between the two the isthmus is felt as a soft compressible area. This is the most valuable physical sign of early pregnancy.

(3) **Cervix**: From the second month, the cervix progressively softens from below upward, which is well marked by fourth month. This is known as **Goodell's sign**. There is shortening of the cervix towards the last months of pregnancy. The orifice becomes circular instead of being transverse, and admits the point of finger to greater depth.
(4) **Intermittent Uterine Contractions (Braxton-Hick’s sign):** Intermittent, painless uterine contractions are difficult to be observed before the third month, but are easily felt after the fourth month. Each contraction lasts about a minute and relaxation for about two to three minutes. They are present even when the foetus is dead.

(5) **Ballottement:** It means to toss up like a ball. This is positive during the fourth and fifth months of pregnancy as the foetus is small in relation to the amount of amniotic fluid present. To obtain vaginal ballottement, two fingers are inserted into the anterior fornix and a sudden upward motion given. This causes the foetus to move up in the liquor amnii and after a moment, the foetus drops down on the fingers like a ball bouncing back. External ballottement can be obtained by imparting a sudden motion to the abdominal wall covering the uterus; in a few seconds the rebound of the foetus can be felt. This can be negative if the amniotic fluid is scanty.

(6) **Uterine Souffle:** This is a soft blowing murmur, which is synchronous with the mother's pulse. It is heard by auscultation on either side of the uterus just above inguinal ligament, towards the end of fourth month. It is due to passage of blood through the uterine vessels.

(7) **Biological Tests:** They are based on the reaction of test animals to chorionic gonadotropins contained in the pregnant woman's blood or urine. They are: (1) The rapid rat test. (2) The Aschheim-Zondek test. (3) Freidman test. (4) Hogben or female toad test. (5) Male frog test. (6) Galli-Mainini test.

(8) **Immunological Tests:** The hormone chorionic gonadotropin (HCG) and human chorionic somatomamnotropin (HCS) are secreted by the syncytiotrophoblastic cells into the fluids of the mother. It can be detected in maternal blood on about the eighth day after ovulation. The rate of secretion rises rapidly to reach a maximum about seven weeks after ovulation, and decreases to a relatively low volume by 16 weeks after ovulation. These tests utilise antibodies to react with another substance for the detection of HCG. Because they are convenient and very reliable (accuracy 98%), they have replaced bioassays for routine screening. An early morning urine specimen will contain the highest level of HCG and is preferable for testing.
(1) Inhibition (indirect) latex slide test:
(2) Direct latex slide test
(3) Haemagglutination inhibition tube test.

(III) Positive Signs of Pregnancy:
(1) Foetal Parts and Movements:
(2) Foetal Heart Sounds:
X-ray Diagnosis:
Radiological signs of foetal death are: (1) Spalding's sign.
(2) Collapse of the spinal column due to absence of muscle tone.
(3) Presence of gas in the heart and great vessels.
(4) Sonography:

DELiVeRy

Delivery means the expulsion or extraction of the child at birth. The question of delivery arises in (1) abortion, (2) infanticide, (3) concealment of birth, (4) feigned delivery, (5) legitimacy, (6) nullity of marriage, (7) divorce, (8) chastity, and (9) blackmail.

Signs of Recent Delivery in the Living:
(1) General Indisposition: For the first two or three days the woman is pale, exhausted and ill-looking with increase in pulse and slight fever.
(2) Breast Changes: These are full, enlarged and tender with a knotty feeling, and colostrum or milk may be expressed. The areolae are dark, nipples enlarged, and superficial veins prominent, and Montgomery's tubercles are present. The presence of colostrum corpuscles in milk strongly indicates that delivery has taken place within a few days.
(3) The Abdomen: The abdominal walls are pendulous, wrinkled and show striae gravidarum especially in flanks which appear as irregular, white or silvery subcutaneous scars. They are simply evidence of previous prolonged distension of the abdomen. They are not formed always and if formed they may disappear.
(4) The Perineum: It is sometimes lacerated and the age of tear may be of value in fixing the date of the delivery.
(5) The Labia: They are tender, swollen and bruised or lacerated.
(6) The Vagina: It is smooth-walled, relaxed, capacious and may show recent tears which usually heal by the seventh day. The rugae begin to reappear about the third week.

(7) The Uterus: Immediately after delivery, the contracted and retracted body of uterus feels like hard muscular tumour, the upper border of which lies about three cm. below the umbilicus. It then diminishes in size by about one-and-half cm. a day. On the sixth day, it is midway between the umbilicus and pubis, and returns to normal condition in nine weeks.

(8) The Cervix: It is soft and dilated and its edges torn and lacerated transversely. The internal os begins to close in the first 24 hours. The external os is soft and patent and admits two fingers. At the end of a week one finger is admitted with difficulty and is closed in two weeks.

(9) The Lochia: It is a discharge from the uterus which lasts for two or three weeks. It has a peculiar sour, disagreeable odour. During the first 4 to 5 days, the discharge is bright-red and contains large clots (lochia rubra). During the next four days, it becomes serous and paler in colour (lochia sersa). After the ninth day colour becomes yellowish-grey or turbid (lochia alba) until its final disappearance.

(10) Intermittent Uterine Contractions: They are usually present for the first 4 to 5 days.

(11) If blood or urine gives a positive pregnancy test, it is strong corroborative evidence that pregnancy has recently terminated. They are likely to disappear in a week or ten days.

The above signs are more characteristic of a full term delivery than of a premature one.

In self-delivery scratches may be found on the neck of a newborn infant due to fingernails of the mother trying to assist birth by pulling on the neck.

Signs of Recent Delivery in the Dead: All the local signs mentioned above may be present. The size of the uterus will vary with the period of gestation and the time after delivery at which death occurred. It is flabby for a day or two, and then gradually shrinks and resumes its firmness. Immediately after delivery the uterine wall is 4 to 5 cm. thick. The uterine cavity is almost obliterated by apposition of the anterior and posterior walls; its
total length is 20 cm. and the length of its cavity 15 cm. In the first 2 or 3 days after delivery, the length is 17.5 cm. and breadth 10 cm. At the end of a week 13 to 14 cm. long and 5 cm. in thickness, and at the end of a fortnight it is less than 12 cm., and returns to normal size, i.e., 7 to 8 cm. length, cm. in breadth, and 2 cm. in thickness in about weeks. After delivery at term, the uterus weighs about 1000 g.; at the end of first week 500 g.; at the end of second week about 350 g.; at the end of 6 weeks 100 to 120 g. Shortly after delivery, the placental site appears as an irregular, nodular elevated area 15 cm. in diameter and is covered with clotted blood, lymph and portions of decidua. The placental site measures 3 to 4 cm. in diameter at the end of the second week, and only one to two cm. at the end of six weeks. The ovaries and Fallopian tubes are usually congested, but may become normal within a few days. A large corpus luteum is usually found in one of the ovaries. For the first few days after delivery, the peritoneum covering the lower part of the uterus is arranged in folds which soon disappears. Soon after delivery, the bladder shows oedema and hyperaemia and frequently submucous extravasation of blood.

**Signs of Remote Delivery in the Living:** A previous pregnancy usually leaves permanent marks on a woman, especially when it has gone to full term. If however, there has been one pregnancy only and if this has not been of full duration, little or no indication of the previous pregnancy state may be left.

1. **Abdomen:** The abdominal walls tend to be lax and show multiple white scars on the lateral aspects. Linea nigra is commonly present.
2. **Breasts:** The breasts are lax, soft and pendulous, frequently wrinkled if the woman has nursed her baby, and occasionally show linea albicantes. The nipples are enlarged, the areolae dark, and Montgomery's tubercles are usually present.
3. **Vulva:** The vagina is partially open as the labia do not completely close the orifice. The perineum may show the scar of an old tear. The vaginal rugae are absent and walls are relaxed.
4. **Cervix:** The external os shows an increase in the opening and depressions at the site of the lacerations. The os appears like a transverse slit, whereas in virgin it is a small dimple in the middle of the cervix.
5. **Signs of Remote Delivery in the Dead:** The uterus is
larger, thicker and heavier. The walls are concave from inside forming a wider and rounded cavity. The body of the uterus is twice the length of the cervix. The cervix is irregular in form and shortened, its edges show cicatrices. The external os is not well defined.

TOXICOLOGY

Toxicology is the science dealing with properties, actions, toxicity, fatal dose, detection estimation of, interpretation of the result of toxicological analysis and treatment of poisons. Forensic toxicology deals with the medical and legal aspects of the harmful effects of chemicals on human beings. Poison is a substance (solid, liquid or gaseous), which if introduced in the living body, or brought into contact with any part thereof, will produce ill-health or death, by its constitutional or local effects or both. The definition of poison is vague and unsatisfactory for (1) a substance which is harmless in small quantities may act as poison, and cause death when taken in large amount, and (2) bacterial toxins are not regarded as poisons in ordinary sense of the term. Clinical toxicology deals with human diseases caused by, or associated with abnormal exposure to chemical substances. Toxinology refers to toxins produced by living organisms which are dangerous to man, e.g. poisonous plants, the venom of snakes, spiders, bees, etc. and bacterial and fungal toxins.

Chemical Analysis: If during autopsy any organ is removed from the body, it should never be placed on any surface, or in any container which is not clean. If this is not done, a doubt may arise, whether the poison found might have been accidentally introduced in the vessel used. If a refrigerator is available, all organic substances, should be kept in it as soon as possible after removal from the body. Chemical compounds should not be added, as they may confuse the issue. Decomposition may produce substances not in the original stomach, but allowances can almost always be made for these without confusion.

Blood is the specimen of choice for detection of poisons, as it gives the best indication of the quantity of drug exerting an effect on the person at the time of death. The urine concentrates the drug or poison in many cases. It is suitable for single direct
spot test, because there is no protein-binding to prevent extraction. The concentration of poisons found in urine is not important in evaluating the quantity ingested or the toxicity. In delayed deaths, the poison may be found in urine, when none is found in viscera.

The muscle, especially of thigh is well preserved in advanced decomposition. Levels of drugs in the muscle more accurately reflect blood levels than the liver or kidney.

Postmortem diffusion of the drugs occurs from the stomach into the liver, mainly the left lobe. Diffusion also occurs in the base of left lung, spleen and pericardial fluid and to a lesser degree into heart, aorta and inferior vena cava.

In a living person, the concentration of a poison is lower in the venous blood as compared to arterial, because tissues may take up the compound from the arterial supply. Portal blood has higher concentration of a poison that is being absorbed from the intestine.

After death, variation in concentration is caused by uneven destruction by enzymatic and microbiological activity and from diffusion from sites of higher concentration. Postmortem levels of many poisons are unreliable because the barriers formed by living cell membranes breakdown after death, and molecules can easily move through the tissues into blood vessels.

It is essential to prevent contamination of the solid viscera with the contents of the gastrointestinal tract, because an idea of the length of time since ingestion may be had from the relative amounts of poison in the stomach, intestines and the solid organs. If the poison is only found in the contents of the stomach, and none in the solid viscera and is not an irritant, doubts may occur about the actual cause of death. Poison found in liver or kidney is proof of absorption. Therefore, it is important to keep the contents of the alimentary canal in separate bottles. Poison found in urine, unless added with evil intention is a proof of absorption and excretion. If the poison is also found in the food or medicine preserved, this would be very strong additional evidence. The stomach contents are of primary value for estimating the quantity ingested in acute overdoses, and qualitatively in identifying substances which have been recently ingested.
Information supplied to the laboratory: The pathologist should provide all available information to the toxicologist, i.e. (1) brief details of symptoms if any, and length of illness, (2) if poison taken is not known, drugs or poisons to which the deceased was known to have access, including medication being taken, (3) history obtained from family members and friends, (4) empty containers or medications found at the scene, (5) autopsy findings, and (6) any special risk with the samples, e.g. hepatitis B virus, AIDS, etc. It is very important to recover and send the container in which the toxic substance had been kept, which narrows the toxicologist's search to one or more specific compounds. The pathologist should not ask the toxicologist to look for a "general unknown poison" in the viscera preserved.

THE ANALYTIC PROCEDURE

For toxicologic analysis poisons can be divided into five groups.

(6) Analysis: Toxicological analysis of biological tissues involves:

Classification: Poisons may be classified according to the chief symptoms which they produce. (I) Corrosives: (1) Strong acids: (a) Mineral or inorganic acids: Sulphuric, nitric, hydrochloric, (b) Organic acids: Carbolic, oxalic, acetic, salicylic. (2) Strong alkalis: Hydrates and carbonates of sodium, potassium and ammonia. (3) Metallic salts: Zinc chloride, ferric chloride, copper sulphate, silver nitrate, potassium cyanide, chromates and bichromates.

(II) Irritants: (1) Agricultural. (2) Inorganic:

(a) Non-metallic: Phosphorus, iodine, chlorine, bromine, carbontetrachloride. (b) Metallic: Arsenic, antimony, copper, lead, mercury, silver, zinc, (c) Mechanical: Powdered glass, diamond dust, hair, etc. (3) Organic: (a) Vegetable: Abrus precatorius, castor, croton, calotropis, aloes, (b) Animal: Snake and insect venom, cantharides, ptomaine.

(III) Systemic: (1) Cerebral: (a) CNS depressants: Alcohols, general anaesthetics, opioid analgesics, hypnotics, sedatives, (b) CNS stimulants: Cyclic antidepressants, amphetamine, caffeine, methylphenidate. (c) Deliriant: Datura, belladonna, hyocyamus, cannabis, cocaine, etc. (2) Spinal: Nux vomica, gelsemium. (3)

(IV) Miscellaneous: Food poisoning, botulism. Poisons may also be classified according to their morbid anatomic manifestations. (1) No morphologic changes are present which can be attributed to direct chemical action by the toxic agent, e.g. acute CNS depressants (alcohols, sedatives, hypnotics, tranquillisers, salicylates); chemical asphyxiants (.carbon monoxide, hydrogen cyanide); organophosphates and most alkaloids. The abnormalities seen at autopsy result from shock and terminal anoxia. (2) Systemic lesions are produced without injury at the portal of entry, e.g. acute haemolytic poisons (arsine, nitrobenzene). (3) Injury is present at portal of entry without systemic injuries, e.g., corrosives, chlorine, sulphur dioxide. Death may be caused by local tissue changes (pulmonary oedema) or acute vasomotor collapse. (4) Local and systemic injuries are present, e.g., heavy metals.

Poisoning may result from: (1) The administration of a poison for criminal purposes. (2) The swallowing of poison by mistake for harmless substance. (3) The inhalation through ignorance or accident, of the vapours of a poison. (4) The incorrect preparation of medicines containing a poison. (5) The accidental taking of a large dose of medicine containing a poison. (6) Excessive self-medication. (7) Addiction to drugs. (8) Bite by a poisonous animal. (9) Food infected with bacteria or their toxins.

Routes of Administration: In order of rapidity of action: (1) Inhaled in gaseous or vapourous form. It usually involves a volatile substance, gas, dust, smoke or aerosol. Volatile solvents, such as benzene, toluene, xylene, acetone, methylene chloride, methyl chloroform, and carbon tetrachloride poisoning in industrial exposures; solvent sniffing among adolescents, or accidents in the home; gases such as CO, hydrogen sulphide and methane in industries; smokes and dusts of industrial origin may involve lead, mercury, silicon, asbestos and beryllium. (2) Injection into blood vessels. (3) Intramuscular, subcutaneous and intradermal injection. (4) Application to a wound. (5) Application to a serous surface. (6) Application to a bronchotracheal mucous membrane. (7) Introduction into stomach. (8) Introduction into the natural
orifices, e.g. rectum, vagina, urethra, etc. Some drugs can be given by rectal route to produce a systemic effect, e.g., aspirin, barbiturates, chloral hydrate, chlorpromazine, etc. (9) Application to unbroken skin. Organic phosphates, nicotine, some organic solvents and lewisite gas can penetrate the skin and produce intoxication and death. Other substances which are absorbed through the skin are: phenol and its derivatives, endrin, methyl salicylate, mercury, tetraethyl lead and alkylated compounds, cantharidin, hydrocyanic acid, hormones, such as oestrogen, progesterone, testosterone and desoxycorticosterone, vitamin D and K.

Fate of Poisons in the Body: The greater part of a poison is thrown out of the body as a result of vomiting and purging. The portion absorbed is mainly deposited in a less soluble form in the liver, which either partially metabolises or completely destroys it. The unaltered portion enters into the general circulation and acts on the body as a whole, or on the particular organs with which it has special affinity, provided the poison is not destroyed or made harmless by the kidneys and muscles. Some inorganic poisons like arsenic and antimony are retained in certain tissues, such as nails, hair, bones, etc., for a considerable time. Certain poisons like chloroform, phosphorus, nitrates and acetic acid disappear by evaporation or oxidised or destroyed in the body and no trace of them can be detected in the viscera or tissues if post-mortem is delayed.

Drugs Secreted into the Stomach:


Routes of Elimination: The absorbed portion of poison is mainly excreted by the kidneys and to some extent by the skin. Other routes are bile, milk, saliva, mucous and serous secretions. The unabsorbed portion is excreted in the vomit and faeces.

Action of Poisons: (1) Local: The local action by coming in direct contact with the part. (1) Chemical destruction by corrosives. (2) Congestion and inflammation by irritants. (3) Effects on motor and sensory nerves, e.g., tingling of skin and tongue by aconite, dilation of pupils by belladonna or datura.
(2) **Remote:** Remote action produced either by shock acting reflexly through severe pain caused by corrosives, or by poison being first absorbed into the system through the blood, and then exerting a specific action on certain organs and tissues, e.g., cantharides acting on kidneys produces nephritis, nux vomica acting on the spinal cord causes tetanic convulsions.

(3) **Combined:** Drugs like carbolic acid, oxalic acid, phosphorus, etc., have local and remote actions.

**Causes Modifying Action of Poisons**

(1) **Quantity:** More the quantity, more severe are the toxic effects. A large quantity of poison taken orally may cause excessive vomiting, causing its rapid elimination and decreased toxicity, e.g. alcohol, copper sulphate, etc. The action of a poison varies with the dose, e.g., a very large dose of arsenic may produce death by shock without causing irritant symptoms, while moderate doses produce irritant symptoms and small doses produce therapeutic action.

(2) **Form:** *(a) Physical State:* Poisons act most rapidly when gaseous and less when liquid. In case of solids, the action depends on their solubility.

*(b) Chemical Combination:* The action of a poison depends upon the solubility or insolubility resulting from a chemical combination, e.g., silver nitrate and hydrochloric acid are both strong poisons, but when combined, form an insoluble salt of silver chloride which is harmless. A substance may be harmless in metallic state but its salt may be toxic, e.g., arsenic is not poisonous but its salts are poisonous. Certain poisons which are not soluble in water may become dissolved in the acid secretion of the stomach and absorbed into the blood, e.g., lead carbonate and copper arsenite.

*(c) Mechanical Combination:* The action of a poison may be altered if combined mechanically with inert substances, e.g., small dose of concentrated mineral acid produces corrosive action, but the same dose largely diluted with water is harmless.

(3) **Mode of Administration:** The rapidity of the action is in the order described under routes of administration. As a rough guide if the active dose by the mouth is considered as unit, the rectal dose is about one-and-half to two, and the hypodermic dose
about one-fourth. A lethal dose is usually ten or more times the maximum medicinal dose.

The rate of absorption from the alimentary canal is variable. Absorption by the stomach occurs more rapidly when the stomach is empty than when it is full. Absorption may be hastened if nature of stomach contents is such as will dissolve the poison, e.g., action of phosphorus will be hastened if oil is taken immediately after it is swallowed. Gastroenterostomy hastens the entry of poisons into the small bowel. Sleep, narcosis and trauma causing gastrointestinal stasis will retard it. Retardation during gastrointestinal absorption, dilution and alteration during digestion, or metabolism by the action of the liver render some poisons almost inactive and greatly reduce the potency of others. The skin is on the whole a bad absorptive organ.

(4) Condition of the Body (a) Age: Age has a considerable effect upon the dosage of drugs. Poisons have greater effect at the two extremes of age. A child under two years of age has not yet fully developed the drug-metabolising enzymes of the liver, and does not have an effective blood-brain barrier, and as such is much more susceptible to the effect of most drugs. There are some drugs of which children can take more than their proportionate dose, e.g., mercury and belladonna. There are some of which they cannot take even a proportionate dose e.g., morphine.

(b) Idiosyncracy: It may be defined as the inherent personal hypersensitivity to the agent in question. Certain people are sensitive for certain drugs and even articles of diet, e.g., shellfish, eggs and fruit. The symptoms usually occur in the skin as an urticaria, but may be of a more general nature with dyspnoea, rigors, fever, diarrhoea, haemorrhage from the bowel and albuminuria. Fatal cases are comparatively rare, but symptoms may be alarming or dangerous. Iodine, bromine, opium, belladonna, aspirin and mercury are common examples of drugs to which many people are allergic.

(c) Habit: The effect of certain poisons decreases with habituation. Tolerance is the ability of an organism to show less response to a specific dose of a chemical than it showed on a previous occasion from the same dose. It results from a decreased reaction between the chemical and the biologic effector substance.
Opium preparations frequently taken, lose much of their effect after a time, and require to be administered in increased doses. Addicts can tolerate quantities of the drug which would endanger life if they had been initial doses. Tolerance is seldom a natural phenomenon. The same effect of habit occurs from the use of tobacco, alcohol, cocaine, morphine and other alkaloids. It is more usually a feature of natural substances, less of synthetic drugs, such as sulphonal, barbiturates, chloral, etc. Tolerance for mineral substances is limited, but it occurs in connection with arsenic to a certain extent.

(d) **State of Health**: A healthy person tolerates better than a diseased. General debility, senility, chronic or disabling disease may cause death of a person to a dose that is ordinarily safe, e.g., CO may kill at a blood saturation of only 25 to 30%. In some diseases, larger doses of certain drugs may be given without harmful effects, e.g., opium in tetanus, delirium tremens and mania, and strychnine in paralysis, while in other diseases certain drugs cannot be given even in small doses, e.g., opium in granular kidney and bronchial asthma and mercury in chronic nephritis.

(e) **Sleep and Intoxication**: The action of a poison is delayed if a person goes to sleep soon after taking it. The action is also delayed if one takes a poison in an intoxicated condition.

(f) **Cumulative Action**: Poisons which are eliminated slowly may accumulate in the body when given in repeated doses for a long time and may ultimately produce symptoms of poisoning.

**Types of Poisoning**: (1) **Acute poisoning** is caused by an excessive single dose, or several smaller doses of a poison taken over a short interval of time. (2) **Chronic poisoning** is caused by smaller doses over a period of time, resulting in gradual worsening. The poisons which are commonly used for the purpose of chronic poisoning are arsenic, phosphorus, antimony and opium. (3) **Subacute poisoning** shows features of both acute and chronic poisoning. (4) **Fulminant poisoning** is produced by a massive dose. In this death occurs rapidly, sometimes without preceding symptoms.

**Diagnosis of Poisoning**

**In the Dead**: Collect all information from the inquest report and from the relatives of the deceased.
(I) Post-mortem Appearances : External: (1) The surface of the body and the clothes may show stains or marks of vomit, faeces or the poison itself. The colour changes in the corroded skin and mucous membrane are: (a) sulphuric and hydrochloric acid: grey, becoming black from blood; (b) nitric acid: brown; (c) hydrofluoric acid: reddish-brown; (d) carbolic acid: greyish-white; (e) oxalic acid: grey, blackened by blood; (f) cresols: brown, leathery; (g) casustic alkalis: greyish-white; (h) mercuric chloride: bluish-white; (i) zinc chloride: whitish; (j) chromic acid and potassium chromate: organge, leathery. (2) Colour of post-mortem staining: The skin may be dark-brown or yellow in phosphorus and acute copper poisoning; cherry-red in poisoning by carbon monoxide; cholate-coloured in cases of death from poisoning by nitrites, aniline, nitrobenzene, acetanilide, bromates, chlorates, etc. due to the formation of methaemoglobin. (3) Smell about the mouth and nose: Substances which may be recognised by their odour are (a) Garlick-like: Phosphorus, arsine gas, arsenic (breath and perspiration), thallium, selenium, dimethylsulphoxide, tellurium, parathion, malathion. (b) Sweet or fruity: Ethanol, chloroform, nitrites, (c) Acrid: Paraldehyde, chloral hydrate, (d) Rotten eggs: Hydrogen sulphide, mercaptans, disulfiram. (e) Fishy or musty: Zinc phosphide. Other substances are: cyanides, phenol, opium, ether, camphor, etc. (4) The natural orifices, e.g., mouth, nostrils, rectum and vagina may show the presence of poisonous material or the signs of its having been used. (5) Injection marks should be looked for with care. (6) Skin should be examined for lesions, e.g. hyperkeratosis and pigmentation may be found in chronic arsenical poisoning. Jaundice may occur in poisoning from phosphorus, senecio, and in susceptible persons by potassium chlorate. (7) Any evidence of marks of violence, such as bruises, or wounds of any nature, may suggest some form of death other than poison.

The bodies of persons poisoned are not more rapidly decomposed than those of others. Some poisons may delay the action of the putrefactive bacteria to some extent.

Internal: There is no special routine peculiar to poisoning cases. All organs must be examined and all contents preserved.

(1) Smell: On opening the body, note any peculiar smell. The skull should be opened first to detect unusual odours in the
brain tissues, because such odours are masked by the opening of the body cavities. This is useful in cyanide, alcohol, phenol, cresol, ether, chloroform and camphor poisoning.

(2) **Mouth and Throat:** Examine the tongue, mouth and throat for any evidence of inflammation, erosion or staining. Areas of necrosis of the pharynx may be seen in death associated with agranulocytosis caused by amidopyrine, thiouracil, dinitrophenol, sulphonamides and barbiturates.

(3) **Oesophagus:** Corrosive alkalis produce marked softening and desquamation of the mucous membrane. In acute cantharidin poisoning, the mucous membrane is often swollen and engorged and may show patches of ulceration.

(4) **Upper Respiratory Tract:** Examine the larynx, trachea and bronchi for evidence of volatile irritants or inhaled poisonous matter. Oedema of glottis, and congestion and desquamation of mucous membrane of the trachea and bronchi may be seen in corrosive acid or alkali poisoning when it enters the respiratory tract.

(5) **Stomach:** The colour and appearance may be normal, though poison is present.

(a) **Hyperaemia:** Hyperaemia of the mucous membrane caused by an irritant poison is generally marked at the cardiac end and greater curvature of stomach, but rarely at the pyloric end. It is usually patchy and of a deep crimson colour. The ridges are more involved in poisoning than in disease. The mucous membrane is often covered with a sticky secretion and shows small haemorrhagic foci. Redness of mucous membrane of the stomach is found after death, but is usually limited to the posterior wall.

In this case, there is no thickening of mucous membrane, nor any thick mucus over its surface. Redness of the mucosa is also found during digestion, in asphyxial deaths due to general venous congestion, and when it is exposed to atmosphere. Hyperaemia caused by disease is uniformly spread over the whole surface and not in patches. Putrefactive changes will alter the colour of a healthy stomach, but the destructive changes of poisoning are usually present. Histological examination helps in cases of doubt.

**Colours other than** red may be present due to various causes. The green colour of ferrous sulphate tablets; the blue of amytal capsule; the pink of soneryl are characteristic Mercury
usually causes a slate-coloured stain; arsenic may show white particles adherent; strong sulphuric, acetic or hydrochloric acids, and concentrated oxalic acid are likely to blacken or char the wall; nitric acid may cause yellow colour, carbolic acid may produce buff or white colour and shrivelling; cresols produce brown colour; copper produces a blue or green colour. The colour may also be due to bile when there will be no signs of inflammation, or to fruit juice or food, when it is uniform and without signs of inflammation.

(b) **Softening**: Corrosives and irritants produce immediate contraction of the muscularis, due to which the superficial epithelium is damaged, while the depths of the glands are protected by compression of their necks by the spasm. Excess of mucus is secreted by the glands due to the neighbouring irritation. If life is prolonged, the poison passes deeper and deeper. Spasm of the pylorus holds a poison at this point, which is the site most often involved. Softening of mucous membrane of stomach, especially at its cardiac end and greater curvature is usually caused by corrosive poisons, chiefly alkaline corrosives. It is also seen in mouth, throat and oesophagus. In disease, it is limited to stomach and is usually found at its cardiac end. Softening due to putrefaction begins in most dependent parts and affects all the coats of the stomach without detachment of its mucosa, and softened patch is not surrounded by an inflamed area.

(c) **Ulcers**: Ulceration due to corrosive or irritant poisons is usually found at the greater curvature of the stomach. It appears as an erosion with thin, friable margins. The surrounding mucosa is softened due to inflammation, and there is diffuse hyperaemia. An ulcer from disease is usually seen on the lesser curvature and the margins are well-defined, thickened and indurated.

(d) **Perforation**: Perforation is occasionally observed, when the strong mineral acids have been taken, especially sulphuric acid; it is much less common with other acids. The stomach, in such cases is blackened and extensively destroyed, the aperture is irregular, the edges sloughing, and the adjacent tissues easily torn. The acid escapes into the abdomen and causes peritonitis. Perforation by irritant poisons is rare. In chronic gastric ulcer, it is oval or rounded and has a punched-out appearance and may show chronic adhesion to neighbouring organs.
In autolysis from post-mortem digestion, the change is confined to the stomach alone, and it is commonly found only at the cardiac end. The opening is large and irregular, with rough and pulpy edges. The surrounding mucous membrane is softened and gelatinous. Peritonitis is not seen.

**The Contents of the Stomach:** The ligatured stomach should never be sent for analysis without being opened, as putrefaction may obscure changes in the mucous membrane, and the gases produced may result in the lid of the jar being forced off in transit. The stomach is opened along its greater curvature in a clean porcelain dish. The wall is examined for fragments of poison adhered to it, such as powdered poisons, fragments of capsules, starch from tablets, fragments of leaves or fruit, cantharides, etc. The contents must be carefully observed and written notes made, regarding the volume, colour and contents, including food. The presence of seeds, leaves, capsules and foreign bodies, such as nails, pins, glass, etc., must be noted.

The cells of plants in the alimentary canal, retain their characteristic shape, dimensions, surface ornamentation and other characters, which can be identified in vomit or material from gastrointestinal tract, by microscopic examination.

(6) **The Duodenum and Intestines:** A strongly acid reaction is of greater significance here than in the stomach contents. Ulceration beyond the pylorus is usually due to natural disease. The only characteristic change which occurs in the intestine is seen in mercury poisoning. This change which usually involves the ascending and transverse colons, is a diphtheretic colitis, which may resemble the enteritis of acute bacillary dysentery.

A normal gastrointestinal tract rules out poisoning by corrosive acids and alkalis, phenols, mercury and arsenic.

(7) **Liver:** Substances, such as arsphenamine, phosphorus, chloroform, trinitrotoluene, carbon tetrachloride and senecio, may produce liver necrosis. Arsenic, carbon tetrachloride, amanita phalloides, yellow phosphorus and rarely ferrous sulphate produce a fatty liver. Jaundice may be produced by phosphorus, senecio and potassium chlorate, due to acute haemolytic anaemia.

(8) **Respiratory System:** Oedema of the glottis and congestion and desquamation of the mucous membrane of the trachea and bronchi may be seen in corrosive poisoning when the
acid or alkali has entered the respiratory tract. The lungs show non-specific signs of congestion and oedema.

(9) Kidneys: Parenchymatous degenerative changes are commonly found in irritant metal poisoning, and in cantharidin poisoning. Extensive necrosis of proximal convoluted tubules may be found in deaths from poisoning by mercuric chloride, phenol, lysol and carbon tetrachloride.

(10) Heart: Subendocardial haemorrhages in the left ventricle occur in most cases of acute arsenic poisoning.

(11) The bladder, and in females the vagina and uterus should be particularly examined, for poison is occasionally introduced into the body by these routes. In criminal abortion, it may be necessary to send the vagina and uterus for analysis.

Many poisons, such as alkaloids do not produce characteristic tissue changes. The presence of wounds or of a disease sufficient to cause death, does not rule out the use of poison. A poison can cause death without leaving any naked eye changes, and proof of poisoning must be obtained from other sources, or from chemical examination. No poison kills without producing some symptoms of illness, if no signs after death. Therefore, enquiry as to symptoms in life is very important. The conclusion that death was caused by poison depends on evaluation of clinical, toxicologic and anatomic evidence.

False Positives: Many poisons enter the body regularly in small amounts with food, water or air. Arsenic, lead and mercury compounds are sprayed and dusted on fruits and vegetables and are ingested regularly. Nicotine is present in the blood of smokers. Many poisons are used therapeutically, e.g. arsenic and strychnine, sedatives, tranquillisers, etc. These conditions produce false positive results.

Cause of Death: The blood level of the drug or chemical is useful to determine the cause of death, in correlation with clinical and anatomic findings. A lethal level does not by itself establish the cause of death. The blood level of a drug need not always be in the lethal range for it to reflect the cause of death, especially in a treated case. When the presence of a highly toxic material is established even in trace amounts, the inference that the poisoning is cause of death is justified.
**Toxic and Lethal Drug Levels:** Fatal concentrations of poisons vary depending upon: (1) analytical techniques which vary widely both in method and accuracy, (2) site of sampling, (3) fatal level being attributed to one substance without considering the levels of other toxic substances that the deceased may have taken, and of which the pathologist or analyst may not be aware. Many victims who die due to poisoning, have lower blood concentrations of the responsible agent than those usually regarded as fatal. The causes for this may be: (1) Unusual susceptibility to the drug; (2) combinations of drugs can interact in an additive fashion; (3) some pre-existing natural disease may have contributed to death; (4) rapid but not complete absorption of drug; (5) metabolic degradation of the drug during a prolonged survival in which respiratory complications and hypoxic encephalopathy maintain coma and act as the immediate causes of death.

**Toxicity:** The "therapeutic index", or the ratio of the toxic to the effective dose of a drug, indicates the relative toxicity of drugs. Toxicity of the chemicals have been devised depending on the amounts which produce harm.

The **lethal dose** is the dose that kills. "Minimal lethal dose" is the smallest dose that has been recorded as fatal to a healthy person.

**Interpretation of Toxicological Results:** The following factors should be considered in the interpretation of the result of toxicological analysis. (1) Age and weight of the deceased. (2) Presence of a natural disease condition. (3) Presence of traumatic lesions. (4) Degree of tolerance of the individual. (5) Hypersensitivity reaction.

**Putrefaction and Toxicologic Analysis:** In post-mortem decomposition, many poisons present in the tissues undergo chemical changes, and cannot be detected. Putrefaction of normal tissue may produce substances which give chemical reaction similar to those obtained from toxic compounds. Most volatile compounds are lost due to putrefaction, but ethyl alcohol and cyanide may be produced from normal tissue. Neurin, muscarin, mydalen, etc., are produced due to putrefaction, the toxicity of which is equal to the well-known alkaloids. In an embalmed body, it is very difficult to detect and identify most volatile poisons.
**Failure to Detect Poison:** In some cases, no trace of poison is found on analysis, although from other circumstances it is almost or quite certain that poison was the cause of illness or death. The possible explanations of negative findings are: (1) The poison may have been eliminated by vomiting and diarrhoea, e.g., in irritant poisons. (2) The whole of the poison has disappeared from the lungs by evaporation or oxidation. (3) The poison after absorption may be detoxified, conjugated and eliminated from the system. (4) Some vegetable alkaloidal poisons cannot be definitely detected by chemical methods. (5) Some drugs are rapidly metabolised, making extraction difficult. (6) Some organic poisons especially alkaloids and glucosides may by oxidation during life, or due to faulty preservation, or a long interval of time, or from decomposition of the body, may deteriorate and cannot be detected chemically. (7) Biological toxins and venoms which may be protein in nature cannot be separated from body tissues. Immunoassay procedures can detect these compounds. (8) If the poison acts slowly and death is delayed following production of irreversible organic changes (e.g. hydrogen sulphide or cyanide), the poison may be completely excreted. (9) Many drugs may be present in very small amount and these may require considerable amount of viscera for their identification. (10) The wrong or insufficient material may have been sent for analysis.

**DROWNING**

Drowning is a form of asphyxia due to aspiration of fluid into air-passages, caused by submersion in water or other fluid. Complete submersion is not necessary, for submersion of the nose and mouth alone for a sufficient period can cause death from drowning. About 150,000 person die from drowning each year around the world.

**Duration of Submersion in Fatal Cases:** When a person falls into water, he sinks partly due to the force of the fall, and partly to the specific gravity of the body which is 1.08. Shortly afterwards, he rises to the surface due to the natural buoyancy of the body. In sudden immersion into cold water, the victim may take a deep inhalation of water due to reflex from stimulation of the skin.
He may hold his breath for varying periods until the CO$_2$ in his blood and tissues reaches sufficient levels to stimulate the respiratory centre. At that time, an inevitable inhalation of water may occur. When he cries for help and struggles, he is likely to inhale water, which produces coughing and drives out large volume of air out of lungs, and leads to disturbance of the rhythm of the breathing. He may vomit and aspirate some gastric contents. His struggle increases and again he sinks. If this occurs during inspiration, he will inhale more water. The cerebral hypoxia will continue until it is irreversible and death occurs. With warm water, cerebral anoxia becomes irreversible between three to ten minutes. Consciousness is usually lost within three minutes of submersion. The struggle for life with rising and sinking of the body goes on for a variable period, depending on the vitality of a person, until he remains, submerged. Convulsive movements then occur, followed by coma or suspended animation and death.

**Types:** Drowning is of four types: (1) **Wet drowning:** In this, water is inhaled into lungs and the victim has severe chest pain. This is also known as primary drowning, in which death occurs within minutes of submersion secondary to cardiac arrest or ventricular fibrillation. (2) **Dry drowning:** In this type, water does not enter the lungs, but death results from immediate sustained laryngeal spasm due to inrush of water into the nasopharynx or larynx. Thick mucus, foam and froth may develop, producing a plug. This is seen in 10 to 20% cases of immersion and is commonly seen in children and adults under the influence of alcohol or sedative hypnotics. Resuscitated victims have panoramic views of past life and pleasant dreams without distress. (3) **Secondary drowning** (post-immersion syndrome or near drowning): **Near drowning** refers to a submersion victim who is resuscitated and survives for 24 hours. The person may or may not be conscious. These persons may develop hypoxemia resulting in brain damage, pulmonary oedema, haemoglobinuria, cardiac arrhythmias, pneumonitis, fever, sepsis, metabolic acidosis, chemical pneumonitis, cerebral oedema, cardiac arrhythmias and myocardial anoxia. Death may occur from half to several hours after resuscitation in about 20% of cases. In survivors, about 5 to 10% develop most serious neurologic damage. (4) **Immersion syndrome** (hydrocution or submersion syndrome)
inhibition): Death results from cardiac arrest due to vagal inhibition as a result of (a) cold water stimulating the nerve endings of the surface of the body, (b) water striking the epigastrium, (c) cold water entering ear drums, nasal passages, and the pharynx and larynx which cause stimulation of nerve endings of the mucosa. Falling or diving into the water, feet first, or "duck-diving" by the inexperienced, or diving involving horizontal entry into the water with a consequent blow on the abdomen cause such accident. Alcohol increases such effects, due to the general vasodilation of skin vessels, and possibly by some central effects on the vasomotor centre. This is seen in one to two percent of cases of drowning. Deprivation of oxygen is a factor in all types of drowning.

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The Pathophysiology of Drowning: The pulmonary alveolar lining is semi-permeable. If water enters the alveoli, an exchange of water takes place through the alveolar lining. The extent and direction of this exchange depends on the difference between the osmotic pressure of the blood and the water.

(2) Drowning in Sea Water: Due to the high salinity of sea water (usually over three percent NaCl), water is drawn from the blood into the lung tissue, and produces severe pulmonary oedema, and hypopatraemia. This causes haemoconcentration Simultaneously, In an attempt to re-establish osmotic balance, salts from the water in the lungs pass into the blood stream. A marked bradycardia occurs, probably due to the raised plasma sodium level. Slow death occurs from asphyxia.

Causes of Death: (1) Asphyxia: Inhalation of fluid causes obstruction to the air-passages. Circulatory and respiratory failure occur simultaneously, due to anoxia of both the myocardium and the respiratory centre. (2) Ventricular fibrillation: In fresh water drowning death may occur in three to five minutes from a combination of anoxia, and a disturbed sodium-potassium ratio. producing arrhythmias of the heart beat, ventricular tachycardia and fibrillation. (3) Laryngeal spasm may result from inrush of water into the nasopharynx or larynx. (4) Vagal inhibition is due to icy cold water, high emotion or excitement and unexpected immersion. (5) Exhaustion. (6) Injuries: Fracture of skull and fracture-dislocation of cervical vertebrae may occur due to the head striking forcibly against some solid object. Concussion may occur due to striking the head against some hard substance, or the water itself while falling from a height.

Fatal Period: Death usually occurs in four to eight minutes of complete submersion.
Post-mortem Appearances: External: The post-mortem signs are variable and none of them is pathognomonic. If the body is removed from the water shortly after death, the clothing is wet and the skin is wet, cold, moist and pale because of vascular contraction on the surface. The postmortem lividity is light-pink in colour (simulating the colour in CO poisoning) due to oxygenation, but in some cases it is dusky and cyanotic, or it may be a mixture of the two. Post-mortem staining is usually found on the face, the upper part of the chest, hands, lower arms, feet and the calves, as the body usually floats face down, buttocks up, with legs and arms hanging down in front of the body. The face may or may not be cyanotic, the conjunctivae are sometimes congested and few petechial haemorrhages are seen beneath the conjunctivae, especially in the lower eyelids. The pupils are dilated. The tongue may be swollen and protruded. Petechial haemorrhages are rarely seen in the skin. Rigor mortis appears early, especially when a violent struggle takes place before death. Vomiting, micturition, defaecation and seminal emissions may occur agonally.

A fine, white, lathery froth or foam is seen at the mouth and nostrils, which is one of the most characteristic external signs of drowning. The inhalation of water irritates the mucous membrane of the air-passages due to which the tracheal and bronchial glands secrete large quantities of tenacious mucus, and the alveolar lining cell irritation produces oedema fluid. Vigorous agitation of the seromucoid secretion, and the surfactant with aspirated water and retained air by the violent respiratory efforts converts the mixture of endogenous secretions and drowning medium into froth. The froth consists of protein and water and the fine bubbles do not readily collapse when touched with the point of a knife. The froth may project as a small balloon, mushroom-like mass, or in a curved horn from the mouth and nostrils for a distance of several centimetres. Froth is usually white, but may be blood-stained, because of slight admixture with blood from intrapulmonary bleeding. If wiped away, it gradually reappears, especially if pressure is applied on the chest. Froth is seen in death due to strangulation, acute pulmonary oedema, electrical shock, during an epileptic fit, in opium poisoning and putrefaction, but in all these cases it is not of such a large quantity as in drowning, and the
bubbles are also much larger. Putrefaction converts froth into bubbly, reddish, foul-smelling fluid.

**Cutis anserina** or goose-skin or goose-flesh, in which the skin has granular and puckered appearance may be seen. It is produced by the spasm of the erector pilae muscles, attached to each hair follicle, and can occur in living when the skin comes in contact with cold water. It may occur on submersion of the body in cold water immediately after death, while the muscles were still warm and irritable. It is also produced by rigor mortis of the erector muscles. It is rarely seen in India, as the water is usually warm. Retraction of the scrotum and penis is due to the same cause, and has the same value. These changes have been designated "reaction phenomenon".

Weeds, gravel, grass, sticks, twigs, leaves, etc. present in the water may be firmly grasped in the hands due to **cadaveric spasm**. This strongly suggests that the person was alive when he drowned, because it indicates the struggle of the person for his life. This is seen rarely. The old adage about a "drowning man clutching a straw" contains a large amount of scientific truth. Damaged nails and abraded fingers showing sand, mud, or other materials under the nails due to struggle has the same significance.

Soddening of the skin occurs due to absorption of water into its outer layer. It is first seen on the fingertips in two to four hours and spreads to the palm and the backs of the fingers, and the back of the hand, in that order in about twenty-four hours. The accuracy of timing is not possible due to variations in environmental conditions. Similar progress and changes are seen in the skin of the foot, but when shoes are worn, it takes almost twice as long. Wrinkling of the skin begins to appear shortly after immersion, bleaching of epidermis in four to eight hours, and the bleached, wrinkled and sodden appearance is seen in twenty hours. The skin becomes sodden, thickened, wrinkled, and white in colour, known as **"washerwoman's hands"** Similar changes are seen on the feet.

Contusions and abrasions produced during life may not be seen after removal from water, but are seen after the drying of the skin. In a few hours after removal from the water, the face may be
bloated, either livid or black, later changing to a deep green. The
discolouration is usually not found on surfaces which have been in
close contact, as in the armpits and upper limbs if they are in close
contact with the body, and the lower limbs if they are close
together. The stains are usually not seen on the parts which have
been closely wrapped in clothing due to pressure which prevents
accumulation of fluids.

**Internal:** The lungs are voluminous, may completely cover
the pericardial sac, and bulge out of chest when the sternum is
removed. This is known as ballooning and is due to presence of
fluid and air in the bronchi. The lungs are overdistended, and the
alveolar walls are torn. Peripheral displacement of air by water
distends the air spaces and the medial aspects of each lung
approach the midline. The oedema fluid in the bronchi blocks the
passive collapse that normally occurs at death, holding the lungs in
the inspiratory position. On section, an oedematous condition due
to the presence of a large amount of watery, frothy, blood-stained
fluid is seen. Drowning fluid actually penetrates alveolar walls to
enter the tissues and the blood vessels. This has been described
as **emphysema aquosum.** It is present in about 80% of cases and
is presumptive evidence of death from drowning. If the victim is
unconscious at the time of drowning, mere flooding of the lungs
with water, but without formation of columns of froth occurs, which
is known as **oedema aquosum.** The foam acts as a valve which
permits air entry to the lings but obstructs air exit from them.
Usually the lungs are congested moderately, but may be pale due
to the forcing out of blood from the lungs, and compressing of the
vessels in the internal alveolar septa by the air and water trapped
in the alveoli. The impression of ribs (grooves) are often seen on
the lungs; the lungs feel doughy and readily pit on pressure. The
alveolar walls may rupture due to increased pressure during forced
expiration, and produce haemorrhages, which when present
subpleurally are called **"Paltauf's haemorrhages".** Paltauf's
haemorrhages are shining, pale bluish-red, and may be minute or
3 to 5 cm in diameter. They are usually present in about 50 percent
of cases in the lower lobes of the lungs, but may be seen on the
anterior surfaces of lungs, and the interlobar surfaces. Red and
grey patches may be seen on the surface, due to Paltauf's
haemorrhages and to patchy interstitial emphysema, respectively.
The degree of ballooning is reduced in cases of pulmonary fibrosis and when extensive pleural adhesions are present. Petechial haemorrhages on the surfaces of the lungs are absent (or very rare), due to compression of the blood vessels in the interalveolar septa by the water.

In fresh water drowning, the lungs are ballooned but light in weight. They are pale-pink and appear uniformly emphysematous. They retain their normal shape and do not collapse when they are removed from the chest. A crepitus is heard on sectioning and each portion retains its normal shape. On compression, little froth is squeezed out, and there is no fluid in the tissue unless there is oedema. In salt water drowning, the lungs are ballooned and heavy, weighing up to 2 kg. They are purplish or bluish in colour, sodden and jelly-like in consistency and pit on pressure. When removed and placed on a flat surface, they tend to flatten out. On sectioning of the lung, crepitus is not heard. Copious amounts of fluid pour out of the cut sections even without compression. The shape of the sectioned portion is not retained. When squeezed the tissue is found to be filled with fluid in most parts of the lungs, i.e., they are wet and sodden. Occasionally, small intra-alveolar haemorrhages are seen in both fresh water and sea water drowning which causes the red staining of the foam in the respiratory tract. If the body remains in water for several hours, these changes become less marked, and the difference in appearance between the fresh water and sea water drowning lungs are not clear.

In death due to laryngeal spasm, very little water may enter the lungs but asphyxial signs are present. In laryngeal spasm, there is no anatomic evidence. It is a diagnosis of inference and exclusion. In many cases of drowning, relatively dry lungs (dry lung drowning) are observed. This may occur if circulation continues for a short time after removal of the victim from the water or if resuscitative measures are carried out. In such cases, most of the water in the lungs is absorbed into the hypertonic plasma while the lungs remain distended. It has been suggested that this is likely to occur if laryngeal spasm supervenes to prevent further water entry, so that continued circulatory function can remove intra-alveolar fluid into the plasma. Microscopic examination of lungs from freshly drowned persons shows distension of alveoli, alveolar
ducts and bronchioles, with extension, elongation and thinning of the septa and compression of the alveolar capillaries. Some alveolar walls may have been ruptured. Capillary congestion, intra-alveolar haemorrhages and protein-rich oedema fluid are often present. Intra-alveolar and intra-capillary red cells may not contain haemoglobin.

Froth appears within two minutes of drowning, and its quantity varies depending on the length of the submersion, and the violent respiratory efforts. The mucosa of the air-passages is congested. Froth in the air-passages varies from body to body. They may be completely filled by it, but usually the froth is seen in secondary bronchi and beyond. This is one of the characteristic signs of drowning, but if artificial respiration has been performed, especially by means of a respirator, the amount of troth and fluid in the air-passages may be greatly reduced. If there has been delay between death and examination, froth in the lungs and air-passages and over-distension of the lungs is not seen in most cases of drowning.

The fluid in the respiratory passages is of the same nature as the medium in which the body was found and substances like tine silt, grit, sand, weeds, diatoms, or various forms of algae can be found. If the matter has penetrated deeply into the lung it is useful evidence, but its presence in the trachea may be due to passive entry after death. Occasionally, the individual vomits during the unconscious gasping phase of drowning, and stomach contents may be found in the air-passages. The pleurae may be discoloured by haemorrhages, but petechial haemorrhages of asphyxial type are not found. Petechial haemorrhages may be present in the subepicardial region of the heart posteriorly.

If a dead body is thrown into water, due to the hydrostatic pressure water passes into the lungs. This "hydrostatic lung" will simulate the "drowning lung". According to Eisele, a "drowning lung" may be produced in a body remaining at the depth of 2 metres for 20 hours. A drowning lung together with the frothy fluid is diagnostic. Inhalation of water causes obstruction of the pulmonary circulation. This results in dilation of the right side of the heart and the large veins, which contain dark fluid blood. The blood is fluid due to the dilution by inhaled fluid and release of
plasminogen activator from the damaged endothelium of pulmonary capillaries. The intima of the aorta is stained red.

The stomach contains water in 70% of cases. If the chemical and microscopic nature of the water is same as that of the medium of submersion, it is a valuable confirmatory evidence of drowning, but it is possible that the victim might have drunk the same water shortly before death. When a disagreeable liquid which would not be swallowed voluntarily and which corresponds to the drowning medium, e.g., liquid manure, or muddy water, containing debris, is found in the stomach, it is a valuable indication of drowning. The amount should be measured and examined for foreign substances. Water is also not found in the stomach, if the person died from syncope or shock and in putrefaction. Gastric mucosa is often soft and heavy. The-small intestine may contain water in about 20% cases. This sign is regarded as positive evidence of death by drowning as it depends on peristaltic movement which is a vital act.

The brain is swollen with flattening of the gyri. The other organs are congested. When the struggle is violent, the victim may bruise or rupture muscles, especially those of the shoulder-girdle. Violent respiratory efforts may force some water into the middle ear through the Eustachian tubes. Haemorrhages are found in the middle ear in about half the cases of drowning. Haemorrhage in temporal bone or in the mastoid air cells, is seen in large-number of cases. The increased pressure transmitted from the surrounding water to the body and tending to be uniformly distributed, more easily compresses air in closed cavities than the body tissues. The lining of those cavities absorbs fluid and swell up followed by vascular engorgement and haemorrhages into the chambers. Temporal bone haemorrhages are also seen in deaths due to hanging, head injury and CO poisoning. Fluidity of the blood is due to fibrinolysis resulting from release of plasminogen activator from the damaged endothelial system.

Cutis anserina, washerwoman appearance of the palms and soles, pulmonary oedema, and haemorrhage into the petrous and mastoid bones can be found in a victim of drug overdose thrown in water, and the victim of heart attack collapsing into water.
DIATOMS: They are microscopic unicellular or colonial algae. They have a complex structure of their cell-walls which are usually strongly impregnated with silica and contain chlorophyll and diatomin, a brown pigment. Diatoms belong to class Bacillariophyceae. Diatom secretes hard siliceous outer box-like skeleton called a frustule (fig.14-16). They resist heat and acid. There are about 15,000 species. They vary considerably in size from 2 microns to one mm. in length or diameter. Most species are from 10 to 80 microns in length and if elongated, up to 10 microns in width. Diatoms measuring up to 60 microns in diameter are said to enter the pulmonary circulation during drowning. The diatom skeletons are readily recognisable as radially or axially symmetrical structures. They vary from place to place, and there are seasonal variations at the same place. They occur in cultivated soils and on surface of moist rocks and in the atmosphere. Large numbers of free floating diatoms are found in both fresh water and sea water. Their shape may be circular, triangular, oval, rectangular, linear, crescentic, boat-shaped, etc. They may be demonstrated in human organs by: (1) direct digestion of the material with nitric acid and sulphuric acid, (2) incineration in electrical oven and then dissolving the ashes with nitric acid, (3) direct microscopic examination of the lungs. Water is squeezed out from the lungs, centrifuged and sediment examined, (4) microscopic examination of tissue section, whereby optically empty sections are produced.

The drowning fluid and the particles in it, e.g., diatoms and planktons, pass from the ruptured alveolar wall into lymph channels and pulmonary veins and thus enter the left heart. Only a live body with a circulation could transport diatoms from the lungs to the brain, bone marrow, liver and other viscera, and skeletal muscle (fig. 14-18). They are also found in the bile and urine. The bone marrow is highly suitable and reliable. The bone marrow of long bones, such as the femur, tibia and humerus or sternum is examined for diatoms. The sternum is washed in distilled water. The periosteum is removed from the posterior surface. A piece of rectangular bone is removed with a sharp and clean knife and the marrow is curretted out from the gutter. Kidney, lung, liver or brain is also washed and 1x1 cm. pieces cut from the deeper tissue.
LITERATURE

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