CHAPTER 6
DEATH AND ITS CAUSE

Thanatology deals with death in all its aspects. The word "death" denotes the death of a human being (S.46, I.P.C.). 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 **(Bishop's tripod of life)**, but there is no legal definition of death. The question of death is important in resuscitation and organ transplantation. As long as circulation of oxygenated blood is maintained to the brain stem, life exists. Whether the person is alive or dead can only be tested by withdrawal of artificial maintenance. A person who cannot survive upon withdrawal of artificial maintenance is dead. The success of a homograft mainly depends upon the type of tissue involved, and the rapidity of its removal after circulation has stopped in the donor. Cornea can be removed from the dead body within 6 hours, skin in 24 hours, bone in 48 hours and blood vessels within 72 hours for transplanatation. Kidneys, heart, lungs, pancreas and liver must be obtained soon after circulation has stopped as they deteriorate rapidly.

THE MOMENT OF DEATH : Death is not an event, it is a process. 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 favour 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 centres 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).

BRAIN STEM DEATH: A current fairly conservative medical definition of death, that is "brain death" permitting removal of vital organs, such as heart or liver is irreversible coma (irreversible structural brain damage), consisting of : (1) Deep unconsciousness with no response to external stimuli or internal need. (2) No movements, no spontaneous breathing. (3) Cessation of spontaneous cardiac rhythm without assistance. (4) No reflexes (except occasionally spinal reflexes). (5) Bilateral dilatation and fixation of pupils. (6) Flat isoelectric EEG (confirmatory but not essential), provided that (a) all of the previously mentioned are present for a 24 hour period, (b) patient's body temperature should not be below 32°C., and (d) metabolic and endocrine disturbances, which can be responsible for coma should have been excluded. There should be no profound abnormality of the serum electrolytes, acid-base balance or blood glucose. Brain death is certified under S 3(6) of Transplantation of Human Organs Act, 1995.

BRAIN STEM: The tissue in the floor of the aqueduct, between the third and fourth ventricles of the brain contains ascending reticular activating system. The ascending reticular activating substance, which extends throughout the brainstem from the spinal cord to the subthalamus, determines arousal. Damage to the ascending reticular activating substance or damage to areas of the cerebral hemispheres results in disturbance of normal consciousness. If this area is dead, the person is ireversibly unconscious and apnoeic (incapable of breathing).

Functions of brainstem: A properly functioning paramedian tegmental area of the brain stem is a precondition for full consciousness which enables the cerebral hemispheres to work in an integrated way. Lesions of this part are associated with profound coma. The brainstem is also responsible for the respiratory drive, and in large measure (but not exclusively) for the maintenance of blood pressure. All motor output from the brain travel through the brainstem. Apart from vision and smell, all the sensory traffic coming into the brain arrives through the brainstem. The brainstem also mediates the cranial nerve reflexes.

Diagnosis of brainstem death: (1) The cause

of coma should be established. The cause must be irremediable structural brain damage. (2) It should be made sure that the patient is not suffering from drug intoxication, hypothermia, or profound metabolic disturbance. (3) Brainstem reflexes should be absent. (a) Pupillary response to light. (b) Corneal reflex. (c) Vestibulo-ocular reflex (tested by introducing iced water into the ear). (d) No grimacing in response to painful stimuli applied either to trigeminal territory or to the limbs. (e) No gag reflex to a catheter placed in larynx and trachea. (4) The patient should be deeply comatose, and the effects of depressant drugs, primary hypothermia or potentially reversible metabolic and endocrine distrubances as the cause of continuation of coma must be excluded.

The diagnostic tests should be determined by two doctors. The test should be repeated at least once, the interval depending on the opinion of the doctors. If it is proposed to request organ donation, the doctor who treated the patient or diagnosed brainstem death should not be part of the transplant team.

Whole or part of the brain can be irreversibly damaged due to hypoxia, cardiac arrest, intracranial haemorrhage, 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. They are not in need of life-sustaining treatment but require nutrition and hydration.

If the brainstem is damaged due to trauma, cerebral oedema, haemorrhage, hypoxia or infection, such as poliomyelitis, respiratory motor system fails and damage to the ascending reticular activating system causes permanent loss of consciousness, and higher centres in the cortex are also irreversibly damaged causing **'whole brain death'.**

Tissue and organ transplantation: (1) **Homologous donation** means grafting of the tissue from one part of the body to another in the same patient, such as skin or bone. **(2) Live donation** includes blood and bone marrow transfusion. Live organ donation include kidney and parts of the liver. **(3) Cadaveric donation:** Most organs must be obtained while the donor heart is still beating to improve chances of success.

Xenograft is grafting of animal tissue into humans, which has limited success.

STUPOR: The patient appears to be asleep and shows little or no spontaneous activity, responding only to vigorous stimulation and then lapsing back into somnolence.

VEGETATIVE STATE: In this the patient breathes spontaneously, has a stable circulation and shows cycles of eye opening and closing which may simulate sleep and waking, but is unaware of the self and the environment. Commonest causes are diffuse axonal injury and diffuse ischaemic brain damage. There is widespread bilateral damage to the neocortex, diffuse damage to white matter, and bilateral damage to the thalami.

THE BEATING-HEART DONOR: After brain stem death has been established, the retention of the patient on the ventilator facilitates a fully oxygenated cadaver transplant, the so-called "beating-heart donor". The results of the transplant are much improved. This has no legal sanction.

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.

Anoxia: According to Gordon (1944) the stoppage of vital functions depend upon tissue anoxia. **Anoxia** means "lack of oxygen". It may be: **(1) Anoxic anoxia:** In this type, oxygen cannot reach the blood, because of lack of oxygen in the lungs. This occurs: (a) from breathing in a contaminated atmosphere, e.g., from exposure to the fumes in wells and tanks, or from exposure to sewer gas, (b) from mechanical interference with the passage of air into or down the respiratory tract, e.g., in smothering, drowning, choking, hanging, strangulation, traumatic asphyxia and certain forms

of acute poisoning. **(2) Anaemic anoxia:** In this type, oxygen-carrying capacity of the blood is reduced, e.g., acute massive haemorrhage, poisoning by carbon monoxide, chlorates, nitrates, coaltar derivatives. **(3) Stagnant anoxia:** In this type, impaired circulation results in a reduction of oxygen delivery to the tissues, e.g., heart failure, embolism and shock. **(4) Histotoxic anoxia:** In this type, the enzymatic processes by which the oxygen in the blood is used by the tissues are blocked, e.g., acute cyanide poisoning. Anoxic anoxia due to lack of oxygen in the inspired air or mechanical obstruction to respiration is usually known as asphyxia or mechanical asphyxia. These four types of anoxia ultimately lead to cardiac failure and death.

Normal levels of oxygen in arterial blood (PO2) is 90 to 100 mm. Hg at 30 years and 65 to 80 at 60 years and above. Reduction to 60 mm Hg results in hypoxia. At 40 mm Hg, there is severe hypoxia, and death may occur at 20 mm Hg.

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 CO_2), causing unconsciousness or death. Nervous tissues are affected first by deficiency of oxygen and their functions are disturbed even by mild oxygen lack.The term asphyxia indicates a mode of dying, rather than a cause of death. 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 9 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 twently seconds of cardiac arrest, and heart stops within twenty minutes of stopping of breathing. If the heart functions for several minutes after stoppage of breathing, the weight of the lungs may increase to 450 to 500 g.

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. Paralysis of the respiratory muscles may result from acute poliomyelitis.

(3) Toxic: Poisonous substances prevent the use of oxygen. (a) The capacity of haemoglobin to bind oxygen is reduced, e.g., poisoning by CO. (b) The enzymatic processes, by which the oxygen in the blood is utilised by the tissues are blocked, e.g., cyanides. (c) Respiratory centre may be paralysed in poisoning by opium, barbiturates, strychnine, etc. (d) The muscles of respiration may be paralysed by poisoning by gelsemium.

(4) Environmental: (a) Insufficiency of oxygen in the inspired air, e.g., enclosed places, trapping in a disused refrigerator or trunk. (b) Exposure to irrespirable gases in the atmosphere, e.g. sewer gas, CO, CO_2. (c) Exposure to high altitude.

(5) Traumatic: (a) Pulmonary embolism from femoral vein thombosis 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 where an unconscious or stuporous person, either from alcohol, drugs or disease, lies with the upper half of the body lower than the remainder.

(7) Iatrogenic 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

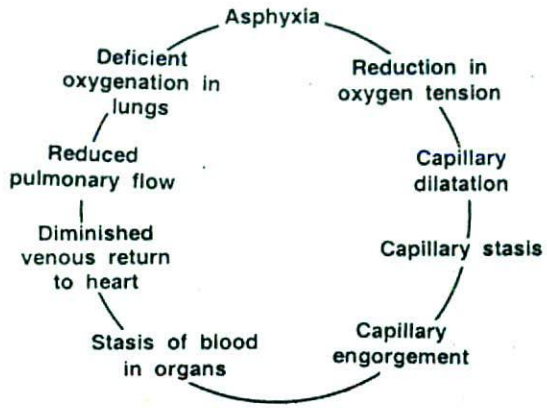


Fig. (6–1). Vicious cycle of asphyxia.

of blood in the dilated capillaries and 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 dilatation and the vicious cycle goes on. (Fig. 6–1).

Petechial haemorrhages are caused due to raised venous pressure from impaired venous return resulting in overdistension and rupture of venules, and not to hypoxia of the vessel walls. Petechiae vary in size from 0.1 to 2 mm. If larger than this they are called **ecchymoses.** 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. Petechiae are rarely seen in the parietal pleura and peritoneum except in haemorrhagic diatheses. 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. In the early stages, the transudation is followed by increased lymph drainage and oedema does not develop. 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. Generalised oedema is not prominent. 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) 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. Oxygenated blood is bright red. It becomes purplish-blue when oxygen is given up. Cyanosis is marked in the skin where hypostatic livid stains develop, and lips, ears, tip of nose, fingernils, cheeks, and internally the lungs, liver, spleen, kidneys and meninges are cyanosed. More than 5g/100 ml of whole blood should be in the form of reduced haemoglobin regardless of the total haemoglobin concentration. The essential cause of cyanosis is diminished oxygen tension in the blood with a rise in proportion of the reduced haemoglobin. Methaemoglobin and sulphhaemoglobin also cause cyanosis. Increased capillary permeability probably results from a combination of stasis and hypoxia.

When carotid arteries are obstructed, intensity of cerebral, facial, and orbital petechiae are much less prominent, than what is seen in cases where venous obstruction predominates. In sudden complete carotid obstruction, facial pallor can be striking. Bleeding from the ear and nose occurs when the back-pressure is severe to rupture small superficial vessels.

Dilatation of the heart chambers on the right side and fluidity of the blood in deaths due to asphyxia are absolete and should be disregarded. This is seen in any type of congestive death, including primary heart failure from many diseases, and is due to the generalised rise in venous and intracardiac pressure. Distension of the atria and ventricles is a common postmortem finding and may result from secondary muscular flaccidity. The light lungs (about 300 gm.) are more compatible with sudden rhythm disturbance and cessation of the action of the heart. Heavier lungs (450 to 500 gm. or more) indicate cessation of respiration with continuance of the heartbeat for several minutes.

Histology: (1) Partial disruption of alveolar septa with distinctive haemorrhage within the alveoli and intra-alveolar oedema fluid. (2) Brick-red discolouration of nerve cells in cerebral cortex seen in stained neurological sections. Pallor and vacuolar

degeneration of Purkinje cells in the cerebellum. (3) Vacuolar degeneration of liver cells in prolonged suffocation.

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. Reliable 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.

Post-mortem Fluidity of the Blood: Shortly after death the blood is usually fluid, and when it is removed from the body, it undergoes spontaneous coagulation. If the autopsy is done a few hours after death, the blood may be clotted and partly fluid. Uncoagulable fluid blood is normally present in the limb vessels and often in the heart of any healthy person who dies suddenly from any cause. It does not give any indication of the cause of death. In most deaths from asphyxia, the blood is fluid and incoagulable. The postmortem fluidity of the blood is due to presence of fibrinolysins, liberated from the vascular endothelium. Fibrinolysin activation occurs due to release of plasminogen activator, through the receptors on the vascular wall for various vasoactive materials that increase during the agonal period. Fibrinolysis is also activated by the leakage of plasminogen-activator due to increased permeability, and to degeneration and necrosis of the cell membrane, as a result of excessive acidosis after death. It is considered that the lysin is adsorbed on to the thrombi, and released into solution when the fibrin is lysed. Under certain conditions, the fibrinolysin may be so active that fibrin is destroyed as rapidly as it is produced, and post-mortem clots never develop in the vessels. In other cases, thrombi are formed, but they undergo lysis. In deaths associated with infection and cachexia, and slow natural death, fibrinolysins may fail to develop and abundant clots are seen in the heart and in the limb vessels. The concentration of circulating fibrinolysins is more in healthy persons, which is further increased during exercise and also due to emotional stress. Therefore, blood is fluid in young persons dying of unnatural causes.

Post-mortem clotting of blood in the heart and venous system is a most erratic process. The condition of the blood at autopsy depends upon the concentration of fibrinolysin, and the rate of intravascular coagulation after death. The blood is likely to be fluid, when the fibrinolysin is active and the rate of coagulation is slow. Post-mortem thrombi are formed when the firbrinolysin is less active, and the rate of coagulation is rapid.

SYMPTOMS: (1) STAGE OF DYSPNOEA: The excess of carbon dioxide in the blood stimulates the respiratory centre. The respiratory movements become increased in rate and amplitude, blood pressure is increased, pulse 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, pulse is fast, veins in the neck become swollen. There are frequently convulsions which cease as the victim becomes insensible and the reflexes are abolished.

(3) STAGE OF EXHAUSTION: The respiratory centre is paralysed, the muscles become flaccid. There is complete insensibility, reflexes are lost and the pupils

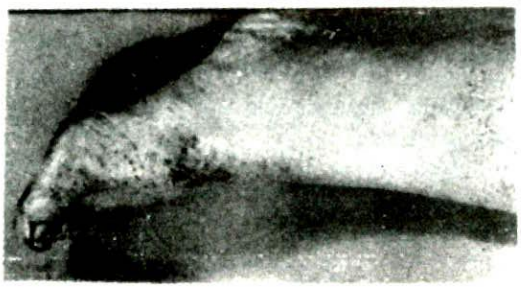


Fig. (6–2). Tardieu spots on foot and leg in a case of hanging.

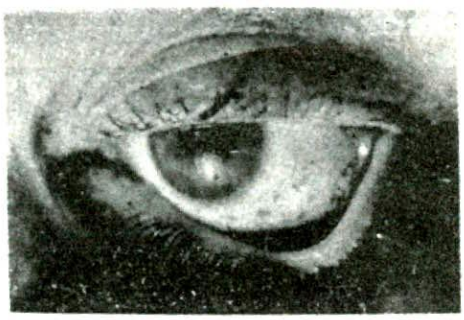


Fig. (6–3). Subconjunctival petechial haemorrhages.

are widely dilated. 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.

Post-mortem Appearances: External: Post-mortem hypostasis 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, eyelids 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, especially in lax, unsupported tissues, such as forehead, skin behind the ears, eyelids, circumoral skin, conjunctivae and sclerae, neck, buccal mucosa, epiglottis, visceral pleura, pericardium, thymus and rarely in the serosa of the bowel (Fig. 6-3). In many cases unconsciousness can occur in several seconds.



Fig. (6–4). Petechial haemorrhages in lungs.

Internal: The blood is fluid and dark, because of increased amount of CO_2. The large veins are full of blood. Vessels may burst in the eardrum and in the nose causing bleeding. The larynx and trachea are usaully congested, and contain a varying amount of slightly frothy mucus. The lungs are dark and purple. If the backpressure persists, there is exudation of serous or serosanguineous fluid in the alveoli, producing oedema. The amount of pulmonary oedema, does not indicate the time interval between injury and death. Accumulation of fluid in the posterior and dependent parts of the lungs after death, should not be mistaken for pulmonary oedema. Some of the marginal portions of the lungs may show emphysematous changes. The abdominal viscera show marked venous congestion. The brain is often congested. The cranial sinuses are usually filled with dark blood. **Tardieu spots** are numerous where the capillaries are least firmly supported, as in subconjunctival tissues and under the pleural and pericardial membranes (Fig. 6-4) 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. In the brain, petechiae occur in the white matter, and there may be larger patches of bleeding in the subarachnoid space, because of acute venous engorgement. Often profuse petechiae and ecchymoses are seen under the scalp due to the same mechanism. The time taken for these various signs to occur depends on the circumstances, from a few seconds to several minutes. 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. Petechiae are likely to occur in association with cyanotic congestion and may not be visible until the area is drained of blood

during autopsy. Someimes, it is difficult to distinguish petechiae from the cut ends of congested vessels, especially in the brain. Microscopic examination will confirm the nature of haemorrhage. Shapiro (1955) pointed out that some of these spots were spontaneous post-mortem artefacts, and some seen at autopsy, disappeared in a short time due to reduced intravenous pressure in the major pulmonary vessels at autopsy.

The natural diseases which produce haemorrhages in the skin include bacterial endocarditis, meningococcal septicaemia and blood dyscrasias, especially purpura and haemophilia, and also in deaths from coronary thrombosis, acute heart failure, secondary shock, and rapid anoxia. Petechiae can be seen following any severe increase in intrathoracic pressure including asthmatic attack, heart failure, respiratory failure, straining at stool, and soon after delivery. These conditions produce relatively large haemorrhages which tend to combine. Their distribution is general, whereas Tardieu spots are present above the level of obstruction.

Asphyxial signs are found in many kinds of natural disease. Asphyxia causes vomiting due to medullary suboxia, due to which the air-passages may be filled at the end of asphyxial event by the inhaled vomit. This finding especially in infants, shoud not be assumed to be the cause of asphyxia; it is more likely to be the result. About 20 to 25% of all individuals aspirate food agonally whatever may be the cause of death.

There are variations in the intensity of asphyxial signs. When the asphyxial process is slight and prolonged, the congestive element will be diminished; when intense and short, lividity and congestion are marked. If, during the process of asphyxia, heart failure occurs before respiratory failure, the asphyxial signs may be less marked, depending at which stage in the asphyxial process, the cardiac arrest occurred. Sometimes, the findings are not sufficient to use an accurate term, and the cause of death has to be given within a broad framework, such as "consistent with asphyxia".

In the presence of putrefaction, mechanical asphyxia is indicated ~~by~~ presence of petechial haemorrhages under the eyelids, conjunctivae, sclerae and facial skin.

DELAYED DEATHS : The higher cortical centres suffer first from hypoxic injury, followed by basal ganglia and ultimately the vegetative centres, which explain delayed deaths. Such delayed deaths usually follow periods of unconsciousness, resulting from anoxic cerebral damage and subsequent hypostatic pneumonia. Other lethal sequelae include, massive subcutaneous and mediastinal emphysema from tracheal and laryngeal lacerations, or occlusion of airway due to oedematous or haemorrhagic swelling of pharyngeal tissues or the aryepiglottic folds. When death occurs hours, days or weeks after the asphyxial episode, the proximate cause of death is the traumatic incident.

COMA: It is a state of unarousable 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, CO, 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. Splanchnic pooling of blood occurs.

SYNCOPE: Syncope is sudden stoppage of action of the heart, which may prove fatal. This term is also not used as a cuase 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. Splanchnic pooling of blood occurs.

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 generalised 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. The recognition of the structural organic changes or chemical abnormalities, which cause stoppage of the vital functions is the first step. Understanding the mechanism by which the anatomical and chemical deviations from normal actually casued death, i.e. how they produced or initiated the sequence of functional disturbance which were sufficient to cause respiratory or cardiac arrest, which are the two ultimate lethal processes, is the second step.

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, labouring 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 workstress, 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, laryngeal spasm in drowning, etc. may not show any anatomical findings. **(2) Inadequate external examination** :The presence of fresh and old needle marks may be missed on cursory examination in a drug addict. Death from snake bites and insect bites cannot be explained unless the bite marks are identified. The burn may be missed in electrocution. (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.**

Obscure autopsies are those which do not show a definite cause for death, in which there are minimal, indefinite or obscure findings, or even no positive findings at all. They are a source of confusion to any pathologist. Frequently, these deaths are due to obscure natural causes, but they may be due to certain types of injury or complications of injury, or to poisoning. Mild degrees of natural disease should not be implicated unless other possibilities are most carefully eliminated.

The obscure causes are : (1) Natural diseases: (a) With obscure or some microscopic findings. (b) Death precipitated by emotion, work-stress, and (c) Functional failure, such as epilepsy, paroxysmal fibrillation.

(2) Biochemical disturbances : (a) Uraemia, diabetes, potassium deficiency. (b) Respiratory pigment disorders, such as anaemic anoxia, porphyria.

(3) Endocrine dysfunction: (a) Adrenal insufficiency. (b) Thyrotoxicosis or myxoedema.

(4) Concealed trauma : (a) Concussion. (b) Self-reduced neck injury. (c) Blunt injury to the heart. (d) Reflex vagal inhibition.

(5) Poisoning : Without microscopic change: (a) Delayed suboxic or narcotic poisoning. (b) Anaesthetic overdosage or maladministration. (c) Neurotoxic or cytotoxic poisons. (d) Plant poisoning.

(6) Miscellaneous : Allergy, drug idiosyncrasy.

Non-medical persons believe that the cause of death can always be determined by autopsy. The police may press the doctor for giving a positive statement with regard to the cause of death. In such cases, the doctor should admit his inability to give a positive opinion. Such obscure cases require clinical and laboratory investigations and interview with persons who had observed the deceased before he died, to know the signs and symptoms shown by the deceased before his death. Laboratory investigations may be bacteriological, virological, histological, biochemical and toxicological. In the absence of positive findings from these sources, a

careful assessment of possible functional causes of death must be made, before any cause of death is given as undetermined. Even in these disappointing cases, the negative morbid anatomical and chemical examination is of great value in excluding injuries or poisons, which may have been alleged to have caused or played some part in the death. The presence of infectious, malignant, occupational and other diseases are also excluded. Though the pathologist cannot establish the cause of death, he can exclude many conditions which have been incorrectly attributed to have caused death.

(1) NATURAL CAUSES: CARDIC LESIONS: Acute rheumatic carditis may cause sudden death in a young adult. In some such cases, naked-eye cahnges may be absent, but typical Aschoff bodies of rheumatic fever are found in the myocardium on microscopic examination. Acute toxic myocarditis of diphtheria may cause sudden death, in which the primary lesion in the nose or throat may be overlooked. Idiopathic myocarditis may cause sudden death. Brown atrophy of the heart and senile heart may prematurely fail suddenly. Myocarditis, fibrosis, and necrosis of conducting tissue may escape detection. Small coronary thrombosis and easily dislodged emboli may be overlooked.

Acute occlusion of coronary artery may result from thrombosis or haemorrhage within the wall of the artery. Zones of occlusion are usually less than five mm. in length, and most of the occlusions occur within three cm. of the orifices of the vessels. First part of the anterior descending branch of left coronary artery within two cm. of its origin is commonly affected, followed by the proximal part of the right coronary artery, first part of the circumflex branch of left coronary artery, and the short main trunk of left coronary artery. Fresh thrombi are dark-brown, and attached to the vessel walls. Old thrombi appear as homogeneous yllowish or grey, firm plugs blocking the vessels. Most infarcts occur in the left ventricle in the anterior wall. Posterior infarcts may be due to blockage of either the right vessel or the circumflex branch of the left artery. Infarction usually occurs when the lumen is reduced to twenty percent or less. Right ventricle is involved in less than ten percent cases. Coronary insufficiency from narrowing of the lumen of major vessels may lead to chronic ischaemia and hypoxia of the muscle distal to the stenosis. Hypoxic myocardium is electrically unstable and liable to arrhythmias and ventricular fibrillation, especially at moments of sudden stress, such as exercise or an adrenaline response, such as anger or emotion.

MYOCARDIAL INFARCTION: There is no need for the ischaemia to be severe enough to produce a myocardial infarct. At autopsy, no naked eye changes are seen for the first 12 to 18 hours. After this period, there is oedema and pallor of the affected area which is the first sign, and the cut surface looks granular and dull. After 24 hours to third day, the area becomes better demarcated, and turns yellow and is surrounded by an area of hyperaemia. In one to three days there is a progressive softening and thinning of infarcted area, which is maximum about the tenth day, and rupture may occur. With breakdown of myocytes, red streaks appear giving a 'tigroid' appearance. From the third week and later, the centre of the infarct becomes gelatinous and the colour becomes grey. From ten days onwards, the infarct is slowly converted into a fibrous scar, which is completed in three months. During the next month or two, fibrosis replaces the dead muscle to form a scar. Infarcts may be transmural, or may be confined to inner zone. The papillary muscles are usually involved.

HISTOLOGY: By the second to fourth day nuclei become hollow, shadowy and ghost-like. During the first few days neutrophils infiltrate, which are replaced by monocytes (mostly macrophages) within a week. By the end of first week, disintegration of muscle fibres and new capillaries and fibroblasts appear. By the fourth week early fibrosis is seen, leucoytes diminish and fibroblats and macrophages increase. Firm fibrosis occurs in about three months.

Cardiomyopathies account for the second largest number of sudden death after coronary artery disease.

Immersion of tissue slices in a solution of triphenyl tetrazolium chloride (TTC) gives brick-red colour to intact area (where dehydrogenase enzymes are preserved) and infarcted area appears pale. This is seen in about 4 hours, but the results are variable.

SEVERE NARROWING OF CORONARY ARTERIES (AT LEAST 75%) WITHOUT THROMBOSIS OR MYOCARDIAL INFARCTION IS THE COMMON CAUSE OF SUDDEN DEATH. A physical or emotional stimulus may demand sudden increase in cardiac effort, and if the circulation of the deceased is unable to satisfy the immediate need for increased oxygen, death may result (acute coronary insufficiency). Fresh thrombotic lesions are seen in less than 25% of the cases. Transitory coronary artery spasm can cause death in persons suffering from angina without narrowing of the coronary arteries and without significant atherosclerosis or congenital anomalies. **A thrombus or an occlusive lesion** in the terminal part of the artery may be overlooked. The ostia of the coronary arteries may be occluded by atherosclerosis of the aorta. Spasm of the coronaries may lead to cardiac arrhythmia and death. A ventricle which is overworked and under-nourished may suddenly go into ventricular fibrillation or asystole. This

can occur even if the arteries are not completely blocked and is often precipitated by a sudden demand for an increased cardiac output. The lesions of the conducting system of the heart may sometimes cause arrhythmias and death. Any person with a heart in excess of 420 gm. is at risk of sudden death, even though the coronary arteries are normal. In these cases a state of electrical instability occurs from chronic hypoxia, so that sudden stresses, such as exercise or emotion can suddenly cause the arrhythmias.

Sympathetico-adrenal stimulation causes increased myocardial irritability resulting in ventricular extrasystoles and ventricular fibrillation and death occurs after several minutes. At autopsy, coronary artery sclerosis, fatty heart, chronic valvular disease, etc. may be seen. Lungs are congested and oedematous with pleural petechial haemorrhages.

PRIMARY CARDIAC ARREST DURING EXERCISE: During or immediately after exercise, ischaemia of myocardium, arrhythmias and sudden death can occur. During exercise there is progressive increase of systolic pressure and heart rate, but diastolic pressure is affected very little, and peripheral resistance is decreased. Levels of catecholamine increase, which continue to rise following cessation of exercise, with tenfold increase of epinephrine. Sudden death occurs with ischaemia sensitising the heart to the arrhythmogenic properties of the catecholamines. Sudden death following assaults or even threats may occur due to existing heart disease.

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 downstair 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 without 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 (located in the internal carotid artery just above the bifurcation of common carotid artery, and situated about the level of angle of mandible) 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, characterised by bradycardia and cardiac arrhythmias ranging from ventricular arrhythmias to cardiac arrest. This mechanism acts through a reflex arc in which the afferent (sensory) nerve impulses arise

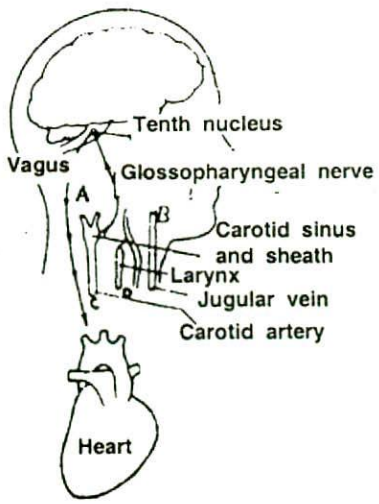


Fig. (6–5). The coarse distribution of the receptor system in vagal inhibition reflexes.

in the carotid complex of nerve endings, but not in the vagal nerve trunk itself. These impulses pass through glossopharyngeal nerves to the tenth nucleus in the brain stem, then return through the vagus (efferent) supply to the heart and other organs. This reflex arc acts through the parasympathetic side of the autonomic nervous system and is independent of the main motor and sensory nerve pathways (Fig. 6-5). There is wide network of sensory nerves in the skin, pharynx, glottis, pleura, peritoneum covering viscera or extending into the spermatic cord, cervix, urethra, perineum and coeliac plexus. Afferent fibres from these tissues pass into the lateral tracts of the spinal cord, effect local reflex connections over several segments and also pass to the brain. The vagal nucleus is controlled by the synaptic connections in the spinal cord, which may be facilitated from both the sensory central cortex and from the thalamic centres. The latter may be responsible for emotional tone noted in the vagal reflex.

Parasympathetic stimulation of the heart can be initiated by high neck compression, pressure on carotid sinus or sometimes by direct pressure over the trunk of the vagus nerve

Causes: (1) The commonest cause of such inhibition is pressure on the neck particularly on the carotid sinuses as in hanging or strangulation. (2) Unexpected blows to the larynx, chest, abdomen and genital organs. (3) Extensive injuries to the spine or other parts of the body. (4) Impaction of food in larynx or unexpected inhalation of fluid into the upper respiratory tract. (5) Sudden immersion of body in cold water. (6) The insertion of an instrument into the bronchus, uterus, bladder or rectum. (7) Puncture of a pleural cavity usually for producing a pneumothorax. (8) Sudden evacuation of pathological fluids, e.g., ascitic or pleural. (9) Sudden distension of hollow muscular organs, e.g., during attempts at criminal abortion, when instruments are passed through the cervix or fluids are injected into the uterus. (10) In degenerative diseases of the heart, e.g. sinus bradycardia and partial or complete A-V block; parasympathetic stimulation further depress the heart rate and may induce a Stokes-Adams attack which may be fatal. There is great variation in individual susceptibility. Death from inhibition is accidental and caused by microtrauma. The stimulus should be sudden and abnormal for the reflex to occur. The reflex is exaggerated by a high state of emotional tension, and also any condition which lowers voluntary cerebral control of reflex responses, such as a mild alcoholic intoxication, a degree of hypoxia or partial narcosis due to incomplete anaesthesia.

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.

A soldier was dancing with his girl friend in the presence of many others in a hall. While dancing, he playfully "tweaked" (pinched) her neck. She dropped down dead on the spot. There were no injuries or signs of asphyxia. Death was as a result of vagal inhibition.

ANAPHYLACTIC DEATHS : (1) Deaths due to allergic reactions are rare. Most deaths occur within 1 to 2 hours and are preceded by signs and symptoms suggesting hyperacute bronchial asthma. Sometimes, deaths are delayed for several hours with nervous symptoms, such as coma or with symptoms of circulatory failure resembling traumatic shock.

AETIOLOGY: Allergic reactions may occur due to: (1) Drugs: Penicillin, aspirin, horse-serum products, iodine-containing agents. (2) Insect bites. (3) Foods: fish, schll-fish, eggs, nuts, fruits. (4) Hormones: insulin, ACTH. (5) Enzymes; trypsin.

When an allergen is injected into the body, the antibodies formed by plasma cells combine with their antigens to form antigen-antibody complexes (allergens), which stimulate mast cells to release histamine and similar substances. About 75% of patients dying of penicillin anaphylaxis do not have allergic reactions during earlier course of penicillin therapy. Patients should be observed for 30 minutes after receiving the injection of antibiotics.

The anaphylactic syndrome is caused by local and systemic release of endogenous active substances. These include leukotrienes, C, D, E or histamine, eosinophilic chemotactic factor and other vasoactive substances, such as bradykinin and kallikrein. Anaphylactic shock is due to bronchospasm with contraction of the smooth muscle of the lungs, vasodilation, and increased capillary permeability. Death occurs due to laryngeal oedema, bronchospasm and vasodilation. Serum tryptase levels are an indicator of mast cell activation and if elevated suggest, an allergic mediator release, particularly in anaphylaxis.

SIGNS OF SYMPTOMS: The onset of symptoms is within 15 to 20 min. **General:** malaise, weakness, sense of illness, oedema of face. **Dermal:** skin rashes, urticaria, pruritus, erythema. **Mucosal:** periorbital oedema,

nasal congestion, angio-oedema, cyanosis. **R.S.:** sneezing, rhinorrhoea, dyspnoea. **Upper airway:** laryngeal oedema, hoarseness, stridor, oedema of tongue and pharynx. **Lower airway:** dyspnoea, acute emphysema, asthma, bronchospasm. **G.I:** nausea, vomiting, dysphagia, abdominal cramps, diarrhoea **C.V.S:** tachycardia, palpitation, shock. **C.N.S.:** anxiety, convulsions.

AUTOPSY: Findings are non-specific. There is usually oedema of the larynx (recedes soon after death) emphysema, cyanosis, petechial haemorrhages, congestion and oedema of the lungs, focal pulmonary distension alternating with collapse and bronchiolar constriction, froth in the mouth and nostrils, visceral congestion, and infiltration of bronchial walls with eosinophils.

History is very important. In cases of insect bites, Ig E antibodies are found in the serum. Ig E binds to the mast cells and basophils. When the cells interact with specific antigens, they release a number of potent compounds, including histamines and an eosinophilic chemotactic factor, which causes an immediate hypersensitivity reaction.

DIAGNOSIS: To establish diagnosis of death from allergic shock, the following should be established: (1) The injected material must be non-toxic to normal persons. (2) Characteristic allergic symptoms should be present before death. (3) Autopsy should not reveal the presence of any other lesion capable of causing death. (4) By the passive transfer technique, a specific sensitising substance corresponding to the allergen must be demonstrated.

DRUG IDIOSYNCRASY : The administration of drugs in amounts which are known to be harmless to normal person may cause death due to drug idiosyncrasy, e.g., many persons are hypersensitive to cocaine. The diagnosis is based mainly on clinical history.

DIABETES: An elevated vitreous glucose level is an accurate reflection of an elevated antemortem blood glucose level. Glucose levels above 200 mg% are diagnostic of diabetes mellitus even if i.v., glucose infusions were administered. Glucose level in vitreous falls gradually as the time after death increases.

ASTHMA: If airflow obstruction is not relieved, there will be steady progression to elevated CO_2, metabolic acidosis, exhaustion and death.

At autopsy, the lungs appear overexpanded, and completely occupy the chest cavity. A sticky tenacious white mucus deposit fills the bronchi. Microscopically, chronic inflammatory infiltrate with numerous eosinophils are seen around the bronchi. The basement membrane of the bronchi is thickened and has a wavy appearance.

EPILEPSY: Very few persons die in status epilepticus. Bite marks in the tongue are seen in about 25% of cases. Death is caused due to postictal respiratory failure.

Sudden death due to epileptic seizures is most likely due to a lethal cardiac arrhythmia induced or propagated by the disorganised neural discharges of seizure.

Negri bodies are found in the pyramidal cells (ganglion or nerve cells) of hippocampus and uncus and Purkinje cells of cerebellum.

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 (WHO). 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.

Causes : (1) Diseases of the Cardiovascular System (45 to 50%) : (1) Coronary artery disease (narrowing and obliteration of the lumen by atherosclerosis). (2) Coronary atherosclerosis with coronary thrombosis. (3) Coronary atherosclerosis with haemorrhage in the wall causing occlusion of the lumen. (4) Coronary artery embolism. (5) Occlusion of the ostium of the coronary artery associated with atherosclerosis or syphilitic aortitis. (6) Arterial hypertension with atherosclerosis. (7) Rupture of the fresh myocardial infarct. (8) Spontaneous rupture of aorta. (9) Angina pectoris. (10) Pulmonary embolism. (11) Systemic embolism occurring in bacterial endocarditis. (12) Rupture of aortic or other aneurysm. (13) Cardio-myopathies, alcoholic myopathy, asymmetrical hypertrophy of the heart. (14) Lesions of the conducting system: fibrosis, necrosis. (15) Valvular lesions: aortic stenosis, aortic regurgitation, mitral stenosis, rupture of the chordae, ball-valve thrombus. (16) Fatty degeneration of the heart. (17) Acute endocarditis. (18) Acute myocarditis. (19) Acute pericarditis. (20) Congenital heart disease in the newborn.

(II) Respiratory System (15 to 23%): (1) Lobar pneumonia. (2) Bronchitis and bronchopneumonia. (3) Rupture of blood vessel in pulmonary tuberculosis with cavitation. (4) Pulmonary embolism and infarction. (5) Air embolism. (6) Influenza. (7) Diphtheria. (8) Acute oedema of the glottis. (9) Acute oedema of the lungs. (10) Lung abscess. (11) Massive collapse of the lung. (12) Pleural effusion.

(13) Pneumothorax caused by rupture of emphysematous bleb. (14) Neoplasm of the bronchus. (15) Bronchial asthma. (16) Impaction of foreign body in the larynx and regurgitation of stomach contents into air-passages and bronchioles.

(III) Central Nervous System (10 to 18%): (1) Cerebral haemorrhage. (2) Cerebellar haemorrhage. (3) Pontine haemorrhage. (4) Subarachnoid haemorrhage. (5) Cerebral thrombosis and embolism. (6) Carotid artery thrombosis. (7) Brain abscess. (8) Brain tumour. (9) Meningitis. (10) Acute polioencephalitis. (11) Cysts of third or fourth ventricle. (12) Epilepsy.

(IV) Alimentary System (6 to 8%) : (1) Haemorrhage into the gastrointestinal tract from peptic ulcer, oesophageal varices, cancer oesophagus, etc. (2) Perforation of ulcers, e.g., peptic, typhoid, amoebic or malignant. (3) Acute haemorrhagic pancreatitis. (4) Strangulated hernia. (5) Twisting and intussusception of the bowel. (6) Paralytic ileus. (7) Appendicitis. (8) Bursting of the liver abscess. (9) Rupture of enlarged spleen. (10) Intestinal obstruction. (11) Obstructive cholecystitis.

(V) Genito-urinary system (3 to 5%): (1) Chronic nephritis. (2) Nephrolithiasis. (3) Obstructive hydronephrosis and pyonephrosis. (4) Tuberculosis of the kidney. (5) Tumours of the kidney or bladder. (6) Rupture of ectopic pregnancy. (7) Toxaemia of pregnancy. (8) Uterine haemorrhage due to fibroids. (9) Cancer vulva eroding femoral vessels. (10) Twisting of ovary, ovarian cyst or fibroid tumour.

(VI) Miscellaneous (5 to 10%) : (1) Addison's disease. (2) Diabetes mellitus. (3) Haemochromatosis. (4) Hyperthyroidism. (5) Blood dyscrasias. (6) Cerebral malaria. (7) Shock due to emotional excitement. (8) Reflex vagal inhibition. (9) Anaphylaxis due to drugs. (10) Mismatched blood transfusion.

CHAPTER 7
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.

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

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

Cessation of Circulation : The stethoscope is placed over the precordial area where the heartbeat can be heard readily. Under normal conditions, stoppage of heartbeat for more than 3 to 5 minutes is irrecoverable and is accepted as evidence of death.

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. Practitioners of yoga can pass into a trance, death-like in character. Involuntary suspension of animation lasting from a few seconds to half-an-hour or more may be found in newborn infants, drowning, electrocution, sunstroke, cholera, after anaesthesia, shock, cerebral concussion, insanity, etc. The patient can be resuscitated by cardiac massage or electric stimulator and artificial respiration.

CASE : (1) In 1964, a physician was called to examine an elderly man at his home. The doctor saw the man lying in bed motionless, quickly checked the man's heart beat and pulse and declared the man dead. In the autopsy room, while the body was being placed on the table, the doctor heard a gurgling sound and noticed a slight swallowing movement. The man was rushed to a hospital and lived for two more months.

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

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

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

minutes of death. The lips appear brownish, dry and hard due to drying.

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

(2) Opacity of the Cornea : This may occur in certain diseases (cholera, wasting diseases) before death. The opacity is due to drying and is delayed for about two hours if the lids are closed after death. If the eyelids are open for a few hours after death, a film of cell debris and mucus forms two yellow triangles of dessicated discolouration on the sclera at each side of the iris, which become brown and then black called ***"tache noir"*** within 3 to 4 hours, upon which dust settles and the surface becomes wrinkled (artefact).

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

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

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

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

COOLING OF THE BODY

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. After stoppage of circulation, convectional transport of heat inside the body stops. Exchange of heat between the core and surface of the body occurs only by conduction. The postmortem rate of heat production by anaerobic glycolysis is very low. With the start of cooling, a temperature gradient develops from the surface to the core of any part of the body. At first heat is lost from superficial layers of the body only. Due to the low velocity of heat transport inside the body, it takes some time for heat to be conducted from the deeper layers to the more superficial layers, until finally, the temperature gradient reaches the core. Conductive heat exchange occurs due to the temperature difference between the body and surroundings, e.g. clothing, covering, air, water, etc. At non-contact areas heat exchange occurs by convectional mechanism, which exceeds that of contact surface. Heat exchange by radiation is extensive for the first hour, but decreases later, depending on the rapid decrease in skin temperature. Only a small fraction of heat is lost by evaporation of fluid from the skin. For about half to one hour after death, the rectal temperature falls little or not at all. Then the cooling rate is relatively uniform in its slope. Then it gradually becomes slower, and when the temperature is within about 4°C of the environment, rate of cooling becomes very slow. The human body rarely reaches the ambient (atmospheric) temperature unless the latter is at or near freezing, probably because enzyme and bacterial action starts during early decomposition. The curve of cooling is sigmoid or inverted 'S' shaped in pattern, because of some residual enzymatic activity and due to retention of heat for some time. The body heat is mostly lost by conduction and convection. In serious illness, circulation begins to fail before death, and hands and feet become cooler than the rest of the body; this coolness gradually extends towards the trunk. In sudden death, the cooling starts after death. A laboratory thermometer 25 cm. long, with a range of 0 to 50°C. which can be read in single degrees is used. The rectum is the ideal

place to record temperature except in cases of sodomy. The thermometer should be inserted 8 to 10 cm. and left there for two minutes. The temperature can also be recorded by making a small opening into the peritoneal cavity and inserting the thermometer in contact with the inferior surface of the liver. The external auditory meatus or the nasal passages also can be used to record temperature. A small electronic thermocouple with a digital readout is better. Where the nose is used, the probe or bulb should be passed up to the cribriform plate, and in the ear, should be placed on or through the tympanic membrane. 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:

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

The normal body temperature varies between 36 to 37.2°C. The rectal temperature is between 36.5 to 37.5. Rectal temperature is about 0.6°C higher than oral. There are individual and daily variations up to 1 to 1.5°C. being lowest in the early morning and highest in the late evening. During sleep the rectal temperature is half° to one°C. lower. It cannot be assumed that the body temperature is normal at death. In cases of fat or air embolism, certain infections, septicaemia, heatstroke and in pontine haemorrhage, thyrotoxicosis, drug reactions, etc. a sharp rise in temperature occurs. Excercise or struggle prior to death may raise the rectal temperature up to 1.5° to 2°C. Low temperature occurs in cases of collapse, congestive cardiac failure, cholera, secondary shock, etc.

Factors Affecting Rate of Cooling : (1) The difference in temperature between the body and the medium : The temperature fall is rapid when the difference between body and air temperature is great. In India, during summer, the temperature of the environment may be higher than that of the body temperature, and as such the cooling is very slow. In tropical climates the heat loss is roughly 0.5° 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 cool more slowly in water containing sewage effluent or other putrefying organic material than in fresh water or sea water. **(5) Covering on or around the body:** The rate of cooling is slow when the body is clothed, as clothes are bad conductors of heat.

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

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

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

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

Development: In the early stage it consists of discoloured patches of 1 to 2 cm. diameter, having the same colour as blood, which can be mistaken for bruises. In doubtful cases, a portion should be removed for microscopic examination. Christison refers to two cases, in one of which two persons were convicted, and in the other three narrowly escaped conviction, upon a mistake of this kind. The areas then enlarge and combine to produce extensive discolouration. 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. The plasma tends to cause oedema of the dependent parts and contributes to the cutaneous blisters of early putrefaction. In the early stages mottled patches of hypostasis may be seen on the upper surfaces of the body, especially the legs due to uneven dilatation of the vascular bed. These patches soon join together and slide down to the dependent parts(Table 7-1). It is usually well developed within four hours and reaches a maximum between 6 to 12 hours and persists until putrefaction sets in. It is present in all bodies, but is more clearly seen in bodies of fair people than in those of dark. It may not be appreciated in old and anaemic persons.

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 and barbiturate poisoning, congestive cardiac failure, deep coma, and asphyxia. In such cases, hypostasis will be marked shortly after death.If death has been taking place slowly over a period, early hypostases may be present before death has actually occurred, especially when the affected part is already engorged. It is intense in asphyxia, where the blood may not readily coagulate, and is less marked in death from haemorrhage, anaemia and wasting diseases due to reduced amount of blood and pigment. It is also less marked in death from lobar pneumonia, and other conditions in which the blood coagulates quickly.

The distribution of P.M. hypostasis: The distribution of the stain depends on the position of the body. (1) In a body lying on its back (supine) it first appears in the neck, and then spreads over the entire back with the exception of the parts directly pressed on, i.e. occipital scalp, shoulderblades, buttocks, posterior aspects of thighs, calves and heels. Any pressure prevents the capillaries from filling, such as the collar band, waist band, belts, wrinkles in the clothes, etc. and such areas remain free from colour and are seen as strips or bands called vibices. Such pale areas (blanching) 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. The pattern of lividity may be modified by local changes in the position of the body , e.g., if the head is turned to one side and slightly flexed on the neck for some hours after death blood may

gravitate into a linear distribution determined by the folds formed in the skin and subcutaneous tissues. If such a body is examined after the neck has been straightened, the linear discolouration of the stains may be mistaken for marks due to beating. (2) If the body is lying in prone position, the lividity appears in the loose connective tissues in front, the colour is intense and Tardieu spots are common. Sometimes, the congestion is so great that minute blood vessels are ruptured in the nose, and cause bleeding. In persons who die in prone position, petechiae, ecchymoses and cutaneous blood blisters may develop after death, in areas of deep hypostasis especially in the shoulders or over the chest, which may be mistaken for asphyxial death. In sudden infant death syndrome and in drunken persons and epileptics who die face down on a pillow or other surface, white areas are often seen on the face around the nose and mouth due to pressure against the supportir g surface. This should not be mistaken for suffocation. Circulatory stasis in the aged, and sometimes the effect of cold, may resemble the effects of violence. Such marks are usually found on ears, shins, forearms and hands, where the circulation is comparatively poor and the skin is exposed. (3) If the body has been lying on one side, the blood will settle on that side, and if lying on back the staining will be seen on the back. (4) If the body is inverted as in drunken persons who slide out of bed, hypostasis will appear in the head and neck. The eyes may suffuse, and numerous haemorrhages may appear in the conjunctivae and hypostatic areas. This may give rise to suspicion of suffocation or strangulation. (5) Sometimes, blotchy areas of lividity appear on the upper surface of the limbs due to some irregularity of capillary dilatation at the time of death. The internal jugular veins are markedly engorged due to the blood which has drained from the head. This blood cannot drain away below due to the heart, and the valves in the subclavain veins prevent the drainage of blood into the upper limbs. As a result of this, the tributaries of the superficial veins in the neck cannot be effectively drained, due to which isolated areas of lividity may develop on the front and sides of the neck resembling bruises. In certain cases, isolated patches of lividity remain separate from the large areas of lividity resembling contusions.(6) If the body has been suspended in the vertical position as in hanging, hypostasis will be most marked in the legs and hands, and if suspension be prolonged for a few hours, petechial haemorrhages are seen in the skin. (7) In drowning, postmortem staining is usually found on the face, the upper part of chest, hands, lower arms, feet and the calves, as they are the dependent parts. If the body is constantly moving its position, as after drowning in moving water, the staining may not develop.

If the body is moved within few hours after death, patches of lividity 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 (complete shifting). This may take from a few minutes to up to one hour. In incomplete shifting, after turning the body over, lividity appears slightly in the downward facing parts. When coagulation in capillaries takes place, the stains become permanent and this is known as **fixation of post-mortem staining.** This usually occurs in 6 to 12 hours or more, but the condition of blood at the time of death exerts a considerable influence. Fixation occurs earlier in summer and is delayed in asphyxial deaths and in intracranial lesions. Some authors are of the opinion that hypostasis does not get fixed. Non-displacement and non-shifting of lividity is due to haemoconcentration by loss of fluid which penetrates the wall of those vessels related to the hydrostatic pressure.

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

Petechiae or larger haemorrhages and palpable blood blisters may develop in areas of hypostasis

Table (7-1). Hypostasis related to the time of death (hpm)

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

Computed by Mallach from literature data.

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

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

commonly in the back of the shoulders and neck, and sometimes on the front of the chest, even when the body is lying on its back. They are common in cyanotic congestive types of death, and appear more prominent with the increase in post-mortem interval, and may blacken the face and skin. They are more prominent when the body lies with the head downwards.

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, acetanilide, bromates, and aniline (causing methaemoglobinaemia) the colour is chacolate or copper brown. (4) In poisoning by phosphorus the colour is dark-brown or yellow.

In asphyxia, the colour of the stains is deeply bluish-violet or purple. A brownish hypostasis may be seen in methaemoglobinaemia and rarely a bronze colour in Clostridium perfringens septicaemia usually associated with septic abortion. 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. This may be most marked over large joints and dependent areas. Refrigerated bodies may also assume a pink colour. In mummification, lividity may turn from brown to black with drying of the body.

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

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, and the lowermost coils of intestine in the pelvic cavity. Hypostasis in the heart can simulate myocardial infarction, and in the lungs it may suggest pneumonia; dependent coils of intestine appear strangulated.

Changes in post-mortem lividity occur when putrefaction sets in. In early stages, there is haemolysis of blood and diffusion of blood pigment into the surrounding tissues, where it may undergo secondary changes, e.g., sulphhaemoglobin formation. The capillary endothelium and the surrounding cells show lytic changes. Microscopically, the cellular outlines are obscured and the capillaries are not identifiable. A contused area shows similar putrefactive changes and it becomes impossible to determine whether the pigment in a stained putrefied area originated from an intravascular (hypostasis) or/ and extravascular localised collection of blood (contusion). There is diffusion of blood-stained fluid in the chest or abdominal cavities. As decomposition progresses, the lividity becomes dusky in colour and turns brown and green before finally disappearing with destruction of the blood.

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.

COLOUR MARKINGS ON DEAD BODY: Various types of colour markings may be seen on the skin or in the internal organs of dead body.

(A) ANTE-MORTEM ORIGIN: (1) Trauma: Bruises and traumatic asphyxia. (2) Asphyxia: Cyanosis. (3) Inflammation: Congestion in organs or the skin. (4) Emboli: Fat embolism. (5) Physical: Exposure to severe cold, and heat.

(B) POST-MORTEM ORIGIN: (1) Hypostasis. (2) Putrefaction. (3) Poisoning: CO, HCN, nitrites, chlorate, etc.

(C) ARTIFICIAL: Paint, grease, dust, mud, coal, blood, semen, etc.

The cause of the colouration can be determined by the patterns of colour distribution, shape, relationship of clothing, etc.

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, eyelids loose tension, and joints are flexible. Body flattens over areas which are in contact with the surface on which it rests **(contact flattening).** Muscles are relaxed as long as the ATP content remains sufficiently high to permit the splitting of the actin-myosin cross-bridges. Muscular irritability and response to mechanical or electrical stimuli persist. Peristalsis may occur in the bowel, and ciliary movements and movements of white cells may continue. Discharges of the dying motor neurons may stimulate small groups of muscle cells and lead to focal twitching, although these decrease with time. Anaerobic chemical processes may continue in the tissue cells, e.g., the liver cells may dehydrogenate ethyl alcohol to acetic acid, and complex chemical changes may occur in the muscles. Pupils react to atropine or physostigmine. Muscle protoplasm is slightly alkaline.

RIGOR MORTIS

Rigor mortis (death stiffening; cadaveric rigidity) is a state of stiffening of muscles, sometimes with slight shortening of the fibres. Individual cell death takes place in this stage.

Mechanism : A voluntary muscle consists of bundles of long fibres. Each fibre is formed of densely packed myofibrils extending through its whole length. These myofibrils are the contractile elements, and are made up of protein filaments of two types, actin filaments (thin) and myosin filaments (thick) which form a loose physico-chemical combination called actomyosin, which is physically shorter than the two substances uncombined. In the relaxed condition, the actin filaments interdigitate with the myosin filaments only to a small extent. Under the influence of the nerve impulse, the arrays of actin filaments are drawn into the arrays of myosin filaments, rather like pistons into cylinders. This causes the muscle to contract.

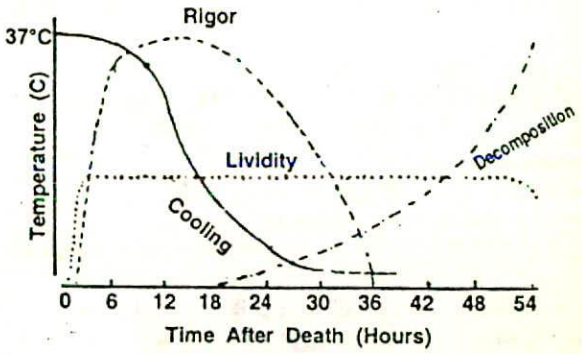


Fig. (7–1). Chart showing the major changes to estimate time since death.

During life, the separation of the actin and myosin filaments, and the energy needed for contraction are dependent on adenosine triphosphate (ATP). Three metabolic systems are responsible for maintaining a continuous supply of ATP in the muscle: (1) Phosphagen system. (2) Glycogen-lactic acid system. (3) Aerobic system. 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, which is dependent upon the supply of glycogen. 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 postmortem 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.

The Order of Appearance of Rigor : All muscles of the body, both voluntary and involuntary are affected. It does not start in all muscles simultaneously (Nysten's rule 1811). It first appears in involuntary muscles; the myocardium becomes rigid in an hour. It begins in the eyelids, neck and lower jaw and passes upwards to the muscles of the face, and downwards to the muscles of the chest, upper limbs, abdomen and 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 mortis always sets in, increases and decreases gradually.

Shapiro (1950) suggests that rigor mortis does not follow the anatomical sequence usually described. He suggests that as rigor mortis is a physicochemical process, it is most likely to develop simultaneously in all the muscles, although the changes are more easily first detected in the smaller masses than in the larger. The **proximo-distal progression** is more apparent than real, for the sequence is determined by the bulk and kind of muscle involved. This would explain the fixation of elbow or knee joints at an earlier stage than the shoulder or hip joints, but this does not explain why the small muscles of the fingers and toes should be the last to stiffen.

The development of rigor is concerned with muscles only. It is independent of the integrity of the nervous system, though it is said to develop more slowly in paralysed limbs.

When rigor is fully developed, the entire body is stiff, the muscles shortened, hard and opaque; knees, hips, shoulders and elbows are slightly flexed and fingers and toes often show a marked degree of flexion. Rigor of erector pilae muscles attached to the hair follicles, may cause roughness, pimpling or goose-flesh appearance of the skin with elevation of the cutaneous hairs, known as **cutis anserina** or goose skin. The testes may be drawn up into the groin; semen may be forced out of the seminal vesicles, and the pupils may be partially contracted. Rarely, if the uterus is in labour at the time of death, the rigor mortis may cause the uterus to contract and expel the foetus.

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 (Table 7-3). Skeletal muscle contains two types of fibres. (1) Type I (red) which are rich in mitochondria with dominant oxidative metabolism. (2) Type II (white) which are relatively poor in mitochondria with dominant glycolytic metabolism. Rigor occurs at different times in the above types of muscles. The fibres which are still slack and some others which are not fully contracted, retain capacity for reversible binding of myosin heads to actin filaments. Re-establishment of rigor occurs due to contraction of such fibres. If force is applied when rigor is fully developed, stiffness is broken up permanently and the rigid muscles may show post-mortem ruptures. Frequent handling of the body breaks the rigor in certain places, leaving a patchy distribution.

P.M. Changes: The contraction of the heart muscle due to rigor mortis should not be mistaken for myocardial hypertrophy. Secondary muscular flaccidity may result in distension of the atria or ventricles, which should not be mistaken for ante-mortem dilatation of the chambers, or myocardial degeneration. Because of these post-mortem changes, it is not possible to determine at autopsy whether a heart has stopped in systole or diastole.

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, because of many extrinsic and intrinsic factors. When rigor sets in early, it passes off quickly and vice versa.

Table (7-3) Rigor related to time of death (hpm)

Stage	Lower limit	Upper limit
Beginning	0.5	7
Maximum	2.0	20
Re-establishment after breaking	2.0	8

Computed by Mallach from literature data.

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. In healthy adults, it develops slowly but is well-marked and lasts longer, while in children and old people it is feeble and rapid.

(2) Nature of Death : The onset of rigor is early and duration is short 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 and lightning. The onset is rapid and the duration short in strychnine poisoning. In strychnine poisoning if death is rapid after a short convulsive stage, rigor appears rapidly and remains for a long time. In organophosphate poisoning rigidity appears early. CO poisoning delays disappearance. The onset is delayed in deaths from asphyxia, severe haemorrhage, apoplexy, pneumonia, nervous disease causing paralysis of muscle, and perfusion with normal saline. It may disappear very rapidly in case of widespread bacterial infection, especially in gas gangrene, where putrefaction begins early. Rigor mortis is frequently absent in persons dying from septicaemia.

(3) Muscular State : The onset is slow and the duration long in case where muscles are healthy and at rest before death. The onset is rapid, and duration short, if there is fatigue or exhaustion (violent or heavy exercise, severe convulsions) before death. In persons who run prior to death, rigor may develop rapidly in their legs, compared to other parts. Rigor may be delayed or very weak in emaciated persons. After insulin injection it develops quickly, as the muscle glycogen is reduced.

(4) Atmospheric Conditions : The onset is slow and duration long in cold weather. Rigor persists longer in cold wet air than in fresh dry air. It is prolonged by dry cold air and cold water. The onset is rapid due to heat, because of the increased breakdown of ATP but the duration is short. If the body is in an extremely hot environment and decomposition begins, rigor mortis may disappear in 12 hours after death. It may persist for 3 to 4 days in refrigerated conditions. When a body is frozen rapidly after death, and when it thaws, rigor mortis appears very rapidly and disappears very rapidly.

Because of the number and variability of the factors which influence the development of rigor mortis, it is not possible to draw any general rule for the rate of its onset, duration and disappearance.

Medico-legal Importance : (1) It is a sign of death. (2) Its extent helps in estimating the time of death, which is not reliable. (3) It indicates the position of the body at the time of death. If the body is lying on its back with its lower limbs raised in the air, it indicates that the body reached full rigidity elsewhere while lying in a position where the legs were flexed or the feet suspended and was later moved to the latter position where the support is no longer present.

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. The degree and depth of the change depends on the intensity of the heat and the time for which it was applied. It is seen in deaths from burning, high voltage electric shock and from falling into hot liquid. Heat causes stiffening of the muscles, because the tissue proteins are denatured and coagulated as in cooking. The muscles are contracted, dessicated or even carbonised on the surface. A zone of brownish-pink 'cooked meat', is seen under this, overlying normal red muscle. Changes in posture, especially flexion of the limbs occur due to muscle contraction. The stiffening remains until the muscles and ligaments soften from decomposition and the normal rigor mortis does not occur.

(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. When the joints are forcibly flexed, crackling of the ice occurs in the synovial fluid. If the body is placed in warm atmosphere, the stiffness disappears and after a time, the normal rigor mortis occurs rapidly and passes off quickly. Hardening of the subcutaneous fat especially in infants sometimes makes the skin-folds rigid, which may be mistaken for ligature mark.

(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. As such, the change preserves the exact attitude of the person at the time

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

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

of death for several hours afterwards. 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. The spasm is primarily vital phenomenon, in that it originates by normal nervous stimulation of the muscles. This is usually limited to a single group of voluntary muscles and frequently involves the hands (Fig. 7-2). 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 death. No other condition simulates cadaveric spasm and it cannot be produced by any method after death. Very great force is required to overcome stiffness. It passes without interruption into normal rigor mortis and disappears when rigor disappears.



Fig. (7–2). A case of suicidal cut-throat. The knife is firmly held in cadaveric spasm.

The mechanism of cadaveric spasm is obscure but possibly may be neurogenic and not the same chemical process as true rigor. The persistence of contraction after death may be due to the failure of the chemical processes required for active muscular relaxation to occur during molecular death. Adrenocortical exhaustion which impairs resynthesis of ATP may be a possible cause. It differs only in the speed of onset and the circumstances in which it occurs.

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

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

(2) Tidy also cited the incident at Sedan, described by Rossbach (1870), when six soldiers were killed by a shell. The head of one preserved his laughing

expression, present at the moment of death. The body of another, who also had his head blown off, remained in a sitting posture, with a cup still in his hand.

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

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

Mechanical excitability of skeletal muscle: **(1)Tendon reaction or Zasko's phenomenon:** Striking the lower third of the quadriceps femoris muscle about 10 cm. above the patella with a reflex hammer causes an upward movement of the patella because of contraction of the whole muscle. This can be seen up to 1.5 to 2.5 hours after death.

(2) Idiomuscular contraction or bulge: Striking at the biceps brachii muscle with the back of a knife causes a muscular bulge at the point of contact due to local contraction of the muscle. In the second phase lasting for 4 to 5 hours a strong and typically reversible idiomuscular pad develops. In the last phase, a weak idiomuscular pad develops between 8 to 12 hours, which may persist up to 24 hours.

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

DECOMPOSITION

It involves two processes, autolysis and putrefaction.

Autolysis : Soon after death, cell membranes become permeable and breakdown, with release of cytoplasm containing enzymes. The proteolytic, glycolytic and lipolytic action of ferments cause autodigestion and disintegration of organs, which occurs without bacterial influence. This chemical process is increased by heat and is stopped by freezing or inactivation of enzymes by the heat. The earliest autolytic changes occur in parenchymatous and glandular tissues and in the brain. Autolytic fermentation results in maceration of the dead foetus in utero, early softening and liquefaction of the brain of the newborn and infants, and softening of the internal organs. Autodigestion by acid gastric juice is a common finding in the newborn and infants, and is seen as softening and rupture of the stomach and lower oesophagus. In adults, such digestion may start before death in cases of intracranial lesions. Putrefaction usually follows the disappearance of rigor mortis. During hot season, it may commence before rigor mortis has completely disappeared from the lower extremities.

Putrefaction or decomposition is the final stage following death, produced mainly by the action of bacterial enzymes, mostly anaerobic organisms derived from the intestines. Other enzymes are derived from fungi, such as Penicillium and Aspergillus and sometimes from insects, which may be mature or in larval stage. The chief destructive bacterial agent is Cl. welchii, which causes marked haemolysis, liquefaction of 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, 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 hydrolyses 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. The fall in the oxygen concentration in the tissues and rise in hydrogen ion concentration after death favour bacterial growth and spread throughout the body. Because the protective agencies of the body are absent, the bacteria spread through the blood vessels using the proteins and carbohydrates of the blood as culture media. Streptococci and staphylococci multiply 10 to 100 times and even more in blood and tissues of corpses kept at room temperatures.

Putrefaction: 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 from the bowel into the tissues of the abdominal

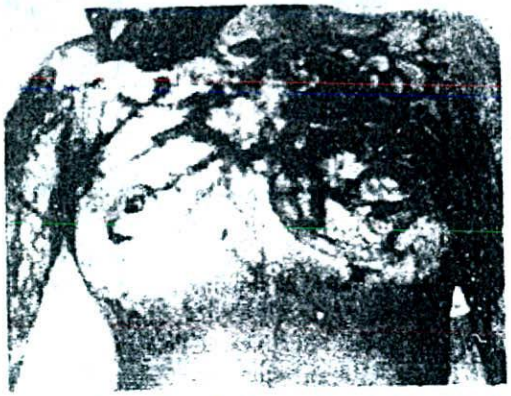


Fig. (7–3). Putrefactive network (marbling).

wall. 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. Internally, this is seen on the undersurface of the liver, where that organ is in contact with the transverse colon. The colour results from the conversion of haemoglobin of blood into sulphmethaemoglobin by the hydrogen sulphide formed in the large intestine and escaping into the surrounding tissues. The colour appears in 12 to 18 hours in summer and in one to two days in winter. Green colouration is more clearly seen on a fair skin than on a dark one. The green colouration then spreads over the entire abdomen, external genitals and then patches appear successively on the chest, neck, face, arms and legs. The patches become dark-green and later purple and dark-blue. They are at first scattered, but later on join together and the whole skin of the body appears discoloured. The putrefactive bacteria spread most easily in fluid and tend to colonise the venous system. The superficial veins especially over the roots of the limb, thighs, sides of the abdomen, shoulders, chest and neck are stained greenish-brown or purplish-red (linear branching pattern) due to the haemolysis of red cells, which stains the wall of the vessel and infiltrates into the tissue, giving a **marbled** appearance (red, then greenish pattern in skin resembling the branches of a tree) (Fig 7-3). This starts in 24 hours, but is prominent in 36 to 48 hours. The clotted blood becomes fluid, and as such, the position of the postmortem staining is altered, and the fluid blood collects in the serous cavities. Putrefactive effusion of foul-smelling bloodstained fluid into the pleural cavities usually starts at about the time when the skin becomes macerated. Such effusions usually do not exceed 60 to 100 ml. unless death resulted from drowning, when several hundred ml. of drowning medium which oozed out through the lungs and visceral pleura, may be present in the thoracic cavities. The reddish-green colour of the skin may become dark-green or almost black in 3 to 4 days.

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

(2) Development of Foul-smelling Gases : The chemical processes in this stage are those of reduction, the complicated proteins and carbohydrates being split into simpler compounds of aminoacids, ammonia, CO, CO_2, hydrogen sulphide, phosphorated hydrogen, methane and mercaptans. The gases are non-inflammable in the early stages, but as the decomposition progresses, enough of hydrogen sulphide is formed, which can be ignited to burn with a blue flame. Gases collect in the intestines in 12 to 18 hours in summer, and 1 to 2 days in winter and the abdomen becomes tense and distended. At about the same time, the eyeballs become soft, the cornea becomes white and flattened or compressed. Later, the eyes collapse. The gas formation in the blood vessels may force blood stained fluid, air or liquid fat between the epidermis and dermis forming small blisters in 18 to 24 hours (Fig. 7-5). Blisters are formed first on the lower surfaces of trunk and thighs, where tissues contain more fluid due to hypostatic oedema. Maggots are produced in one to 2 days which have proteolytic enzymes and penetrate the skin fairly rapidly. This allows air to enter under the skin and more maggots



Fig. (7–4). Advanced decomposition showing marked gaseous swelling of face, eye balls and tongue.

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 (Fig. 7-4). On opening the abdomen, the gas escapes with a loud explosive noise. Discoloured natural fluids and liquefied tissues are made frothy by gas. 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.



Fig. (7–5). Blisters of putrefaction.

Bloodstained frothy fluid has no particular significance. It can be due to rupture of small pulmonary or pharyngeal vessels. The compression of heart forces out its contents. Pressure of the gases may force food from the stomach into the fauces, and this may fall into the larynx. 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 early changes of decomposition in the face, especially when the head of the cadaver is dependent may be mistaken for signs of strangulation, especially when there is oozing of fluid from the nose. The subcutaneous tissues become emphysematous, due to which even a thin body appears obese. The breasts, scrotum and penis are greatly distended. The eyes bulge from their sockets, the tongue is forced out between the swollen and discoloured lips. In 2 to 3 days the sphincters relax and urine and faeces may escape. The epidermis becomes loosened (skin slippage) producing large, fragile sacs of clear or pink-red serous fluid. These gradually enlarge, join together and rupture, exposing large areas of slimy, pink dermis. The exposed subdermal tissue dries with a yellow parchment appearance. The breakdown of vessel walls and cell membranes leads to waterlogging of the tissues which helps the spread of bacteria. Putrefactive effusion of bad smelling bloody fluid into the pleural cavities usually begins at the time when the skin becomes macerated. The anus and uterus may prolapse after two to three days, and post-mortem delivery of a foetus may take place. Wounds caused before or after death bleed, due to pressure of the gases within the heart and blood vessels, and whether they are ante-mortem or post-mortem cannot be made out. Meningeal haemorrhage and haematoma persist well. Owing to the pressure of the gas in the blood vessels, hypostatic stains may be displaced in any direction. After three days, the face is so discoloured and bloated that identification becomes very difficult. Urine and faeces may escape due to intra-abdominal pressure. Blood stained fluid (tissue liquefaction stained by haemolysis) may leak from mouth, nostrils, rectum and vagina. In fat people, the fat, especially omental and mesenteric may liquefy into a translucent yellow fluid. The hair becomes loose and is easily pulled out. The nails are also loose. In 3 to 5 days or more, the sutures of skull especially of children are

separated and liquid brain comes out. Teeth (anterior teeth and often premolars) become loose and may fall out. The skin shows "slippage", and the skin of the hands and feet may come off in a "glove and stocking" fashion. Postmortem luminescence is usually due to contamination by bacteria, e.g., photobacterium fischeri, and the light comes from them and not from putrefying material. Luminescent fungi, Armillaria mellea, is another source of light.

When the nutrient material is used up, the formation of gas stops, and the swelling gradually subsides. The gas leaves the tissues, usually by escaping as a result of damage to the structures or by drainage through a post-mortem wound.

The distribution of the putrefactive changes may be influenced by the position in which the deceased was lying after death, e.g. if a person dies with his head in a dependent position, putrefactive changes will be advanced in the head and neck compared with the remainder of the body. A person dying upon a warm electric blanket may decompose rapidly, the decomposition will be more advanced in those parts of the body in contact with the heated blanket. If only part of the body is in contact with the blanket, there may be a sharp line of demarcation between the decomposition caused by the blanket and the unheated areas of the body.

Rigidity due to Inflation of the Tissues with Gases: In this condition changes of decomposition are well marked, (2 to 3 days) the lower limbs are abducted, flexed and rigid, the arms are abducted and flexed, the hands are open and the fingers are wide apart. The rigidity persists till the escape of gases due to advancing decomposition. This condition is especially seen in bodies recovered from water.

(3) Liquefaction of Tissues : Colliquative putrefaction begins from 5 to 10 days or more after death. The abdomen bursts and the stomach and intestines protrude. In children, thorax also bursts. In obese people, the body fats, especially omental, mesenteric and perirenal may liquefy into a translucent, yellow fluid filling the body cavities between the organs. The tissues become soft, loose and are converted into a thick, semi-fluid, black mass and are separated from the bones and fall off. The cartilages and ligaments are softened in the final stage. Small foetuses rapidly disappear in putrefied blood in cases of ruptured ectopic pregnancy.

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 an uncoffined body buried in a shallow grave, putrefaction is delayed to a moderate extent. In a deeply buried body, the lower temperature, the exclusion of air, absence of animal life, etc., markedly delay decomposition. The important factors are seasonal, climatic variation, the amount of soil water, the access of air, and the acidity or otherwise of the soil water. If a number of bodies are buried in a common grave without coffins, the bodies lying in the centre of the grave will be better preserved than those at the periphery. In India, an uncoffined buried body is reduced to a skeleton within about a year. Buried bones may decay at different rates, e.g. neutral soil may not destroy the skeleton at all. Acidic soil may cause decay in about 25 to 100 years. In bodies placed in airtight coffins, decay process may not occur for several decades, but in a poor coffin which admits air and water bodies will decompose quickly. In a hot climate, bones on the ground surface may decay in 5 to 10 years. The protein content of the bones decomposes. As the bones contain largely inorganic material, they will crumble, rather than decompose. Flat bones and the bones of the infants and old, breakdown faster.

Internal Phenomenon : As the blood decomposes, its colouring matter transudes into the tissues, which gradually change to greenish-yellow, greenish-blue and greenish black colour. The viscera become greasy and softened. The softer the organ, the more blood it contains, and the nearer to the sources of bacteria, the more rapidly it putrefies. The lining of the intestine, adrenal medulla and pancreas autolyse within hours of death. The capsules of the liver, spleen and kidney resist putrefaction longer than the parenchymatous tissues, which are usually converted into bag-like structures filled with thick, turbid fluid material. The organs composed of muscular tissues and those containing large amount of fibrous tissue resist putrefaction longer than the



Fig. (7–6). Cross-section of liver showing honey-comb appearance due to putrefaction.

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) Kidneys, bladder. (7) Prostate, uterus. (8) Skin, muscle, tendon. (9) Bones.

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

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

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

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

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

LIVER : It becomes softened and flabby in 12 to 24 hours in summer. Multiple blisters appear in 24 to 36 hours. Cl. Welchii form characteristic small clumps in a tissue space and produce gas, which soon increase in size. These lesions appear first as small, opaque, yellowish-grey, dendritic figures in the parenchyma. When bubbles develop, the organ has a honeycombed, vesicular or 'foamy' appearance (Fig. 7-6). The greenish discolouration extends to the whole organ, which finally becomes coal-black. In newborn children, the liver putrefies earlier than in adults.

The liver becomes deep blue if there is much passive congestion; green if there is oozing of bile from the gall bladder, or blue-black in the subcapsular area if the pigment comes from the adjacent loops of intestine. In case of jaundice, the liver becomes green a few hours after death, if the pigment is oxidised to biliverdin. In the early stage, the discolouration is seen round the branches of the portal vein. In advanced putrefaction, the liver becomes green or black.

GALL BLADDER : The gall bladder resists putrefaction for a long time, but the bile pigments may diffuse early through adjacent tissues.

PANCREAS : It becomes softened and haemorrhagic.

HEART : It becomes soft and flabby and the cavity appears dilated.

LUNGS : Gaseous bullae are formed under the pleura which are small, pale-red and scattered, and later join together. Later the lungs become soft, collapse and are reduced to a small black mass.

KIDNEYS :The renal tissue becomes flabby and the surfaces are dull and appear parboiled. The cortex darkens and later becomes green.

ADRENALS : The cortex softens from within forming a core around the softened medulla. Later, the medulla liquefies and the interior of the gland appears as a narrow slit or as a wide cyst-like cavity.

BLADDER : It resists putrefaction for a long time if it is empty and contracted. Urine may show albumin due to transudation of serum proteins from the blood, within 48 hours after death.

PROSTATE : It resists putrefaction for a very long time.

UTERUS : The virgin uterus is the last organ to putrefy. Gravid uterus or soon after delivery, it rapidly putrefies.

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. The rate of decomposition is about twice as rapid in summer as in winter. Advanced putrefaction may be seen within 12 to 18 hours in summer. Differences in temperature can cause varying areas in the same body to show different rates of decomposition. Pillows put over and under the deceased's head

prevent circulation of air and cause much more decomposition in the face than is seen in other parts of the body.

A frozen body will not undergo decomposition until it defrosts. If decomposition has already set in, refrigeration of the body may not stop decomposition completely.

(2) Moisture : For putrefaction moisture is necessary, and rapid drying of the body practically inhibits it. If organic substance is dried, putrefaction is arrested. After death from general anasarca, putrefaction is very rapid, and bodies recovered from water, if left in the air decompose rapidly. Organs which contain water decompose more readily than the dry one.

(3) Air : Free access of air hastens putrefaction, partly because the air conveys organisms to the body. In normal condition, the unbroken skin acts as an impermeable barrier to bacteria. Moist and still air helps putrefaction.

(4) Clothing : Initially clothing hastens putrefaction by maintaining body temperature above that at which putrefactive organisms multiply for a longer period. If the clothing is tight as under the belts, suspenders, socks, tight-fitting undergarments, and boots, the putrefaction is slow, for it causes compression of the tissues, which drives out the blood from the part, and prevents the entry of internal organisms. 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 less. Putrefaction is delayed if body is buried in dry, sandy soil, or in a grave deeper than two metres, and when the body is covered and placed in a coffin because of exclusion of water, air and action of insects and animals. When a body is buried in lime, decomposition is 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. Putrefaction is more rapid in porous sandy soil than in soils with an excess of clay. Putrefaction is more rapid if changes of decomposition are already present at the time of burial. In acid peaty soils even the bones may be destroyed.

(B) Internal : (1) Age : The bodies of newborn children who have not been fed, decompose very slowly because the bodies are normally sterile. If the child has been fed before death, or if the surface of the body has been injured in any way, decomposition tends to take place with great rapidity. Bodies of children putrefy rapidly and of old people slowly.

(2) Sex : Sex has no effect.

(3) Condition of the Body : Fat and flabby bodies putrefy quickly than lean bodies, due to larger amount of fluid in the tissues and excess fat, and greater retention of heat.

(4) Cause of Death : Bodies of persons dying from septicaemia, peritonitis, inflammatory and septic conditions, general anasarca, asphyxia, etc., decompose rapidly. In the case of generalised sepsis, bodies undergo rapid decomposition even if they were immediately refrigerated. Putrefaction develops very rapidly in infection due to Cl. Welchii, e.g. acute intestinal obstruction, some cases of abortion and in gas gangrene. 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 case of dismemberment, especially if this has been done while the blood is still fluid, the limbs putrefy slowly, because they are drained of the blood, and intestinal organisms do not gain entry. The trunk putrefies rapidly because of the action of intestinal bacteria, and the access of airborne organisms.

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 : Casper dictum states that a body decomposes in air twice as rapidly as in water, and eight times as rapidly as in earth. The variations are very real, and it is not of much practical value. The rate of putrefaction is slower in water than in air. Putrefaction is more rapid in warm, fresh water than in cold, salt water. It is more rapid in stagnant water than in running water. Putrefaction is delayed when a body is lying in deep water and is well protected by clothing, while it is rapid in a body lying in water contaminated with

sewage. As the submerged cadavers float face down with the head lower than the trunk, gaseous distension and post-mortem discolouration are first seen on the face and then spread to the neck, upper extremities, chest, abdomen and the lower extremities in that order. When the body is removed from the water, putrefaction is hastened as the tissues have absorbed much water. The epidermis of the hands and feet becomes swollen, bleached and wrinkled after immersion, and may be removed as a cast of the extremity, after 2 to 4 days. After several weeks in water, macerated flesh may be stripped off from the body by the action of currents or the contact with the floating objects. Fish, crustacea (crabs, lobsters, shrimps, etc.) and water-rats in a sewer may destroy the body. Moulds may be located anywhere on the body, but generally are found only on the exposed surfaces.

ADIPOCERE (Saponification)

Adipocere (cire=wax) is a modification of putrefaction. In this, the fatty tissues of the body change into a substance known as adipocere. It is seen most commonly in bodies immersed in water or in damp, warm environment. The change is due to the gradual hydrolysis and hydrogenation of pre-existing fats, such as olein, into higher fatty acids, which combine with calcium and ammonium ions to form insoluble soaps, which being acidic, inhibit putrefactive bacteria. Ultimately, the whole of the fat is converted into palmitic, oleic, stearic and hydroxystearic acid, together with some glycerol, and a mixture of these substances forms adipocere. These form a matrix for remnants of tissue fibres, nerves and muscles. 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. In a body immersed in water, this fluid contributes to the hydrolysis of the subcutaneous fat, but the formation of adipocere in deeper sites starts before the extraneous water enters the interior of the body. Water helps to remove glycerine which is formed during hydrloysis of the fats. Activation of lipid peroxidation is a significant component of the mechanism (Manulik, etal, 1999). Adipocere is delayed by cold and hastened by heat. The bodies enclosed in a water-tight coffin for many years may be converted to adipocere even in the absence of external water.

Properties : Adipocere has a distinct offensive or,sweetish smell, but during the early stages of its production, a penetrating ammoniacal odour is noticed. The smell remains in the clothing of those handling such bodies for several days. One's olfactory sense rapidly becomes accustomed to the smell of adipocere, and one cannot smell it after about two minutes exposure. The sense of smell rapidly returns after a few minutes in the open air. Fresh adipocere is soft, moist, whitish and translucent, but old samples are dry, hard, cracked, yellowish and brittle. It shows fragments of fibrous tissues and muscle in the fracture. It is inflammable and burns with a faint-yellow flame. It floats in water and dissolves in alcohol and ether.

Distribution : It forms in any site where fatty tissue is present. It is formed first in the subcutaneous tissues. 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. The epidermis disappears as adipocere forms, probably due to decomposition and shedding, and the dermis becomes darkened. Multiple whitish-grey, rounded outgrowths, varying from one to ten mm. in diameter are seen on the surface. They resemble moulds but are protruding clusters of crystals from the underlying adipocere. In widespread adipocere, the soft tissues are markedly dry, unless there has been prolonged immersion in water. Small muscles are dehydrated, and become very thin, and have a uniform greyish colour. The depths of large muscles have a pink or red colour in bodies with complete conversion of the fat to adipocere. The intestines and lungs are usually parchment-like in consistency and thinness. The liver is prominent and retains its shape. Histologically, the gross features of the organs can sometimes be appreciated, even though the cells are lacking. Adipocere may persist for decades, but finally either degenerates or is removed by mechanical forces or by animals.

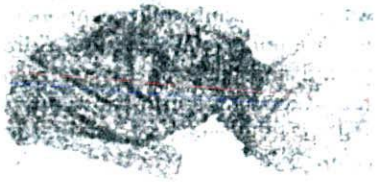


Fig. (7–7). Mummified foetus.

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. Stiffening, hardening and swelling of the fat occurs over a period of months. In most cases, the change is partial and irregular, but rarely the whole body may be affected. 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, its formation has been observed within 3 to 4 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

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, stretched tightly across anatomical prominences, such as the cheek bones, chin, costal margins and hips, adheres closely to the bones, and often covered with fungal growths. As the skin contracts, some of the fat cells in the subcutaneous tissues are broken, and the liquid oil is forced into the dermis which becomes translucent. Mummified tissues are dry, leathery and brown in colour (Fig. 7-7). The face will be greatly distorted due to shrinkage of soft tissues. 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. Collagen, elastic tissues, cardiac and skeletal muscle, cartilage, and bone are usually demonstrable histologically in the mummification material. The entire body loses weight, becomes thin, stiff and brittle. If a mummified body is not protected, it will break into fragments gradually, becomes powdery and disintegrates, but if protected, it may be preserved for years. Mummified bodies may be attacked by insects especially moths and larvae of various flies which destroy the body. A mummified body is practically odourless.

Table (7-5). A typical embalming fluid

Ingredient	Proportion
Formalin	1.5 litres
Sodium borate	600 g.
Sodium citrate	900 g.
Glycerine	600 ml.
Sodium chloride	800 g.
Eosin (one percent)	30 ml.
Soluble wintergreen	90 ml.
Water	up to 10 litres

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

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

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

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

Medico-legal Importance : It is the same as that of adipocere.

CONDITIONS PRESERVING THE BODY

(1) EMBALMING: Emblaming 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. Embalming produces a chemical stiffening similar to rigor mortis, and normal rigor does not develop. Embalming rigidity is permanent. Decomposition is inhibited for many months, if the injection is made shortly after death, and if done several hours after death, the body will show mixture of bacterial decomposition and mummification, and will disintegrate in a few months.

Emblaming alters the apperance 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.

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

Injection Methods: Arterial injection is forcing of fluid in an artery to reach the tissues through the arterioles and capillaries. Diffusion occurs into the cells and tissues for preservation at the capillary level.

(1) **Hand/foot pump.**

(2) **Stirrup pump.**

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

(4) **Gravity injector:** It is the simplest, safest and slowest of the injection methods. The gravity bottle or percolator should hold at least ten litres of fluid. A rubber tubing (preferable to use a transparent plastic tube) with a clamp to control the rate of flow is attached to the mouth of the bottle. A needle is attached to the other end of the tube. The bottle is filled with arterial fluid and raised above the body by a pulley and tackle and fixed at a height. A rise of one metre gives a fluid pressure of 0.6 kg/sq.cm., and two metres about one kg/sq cm. This method takes a longer time and the distribution of fluid is uneven with some areas untouched by the fluid.

(5) **Motorised injectors:** Fluid from an injection tank is forced into the vascular system using air from a compression tank. The pressure and flow rate are controlled by devices. About ten litres of arterial solution is injected into the vascular system within thirty minutes. The injection pressure is about 2 kg/sq cm.

METHOD OF INJECTION: (1) CONTINUOUS INJECTION AND DRAINAGE: The arterial injection is given continuously, against vein tube that is kept open throughout injection. The embalming time is much shortened. Venous drainage and tissue saturation is poor. This method is least satisfactory.

(2) CONTINUOUS INJECTION WITH DISRUPTED DRAINAGE: The injection is made continuously with vein tube closed. The blood in the veins build up a resistance for the arterial flow which helps in the better diffusion of the fluid.Thick blood is discharged when the drain tube is opened. This method is better than the continuous discharge and drainage.

(3) ALTERNATE INJECTION AND DRAINAGE: The arterial fluid is injected for some time with the drain tube closed. The injection is stopped when the superficial veins swell, and the drain tube is opened. When the flow of blood from the drain tube stops, it is closed and the injection started. This process is repeated several times till the embalming is complete.

(4) DISCONTINUOUS INJECTION AND DRAINAGE: This consists of repeated arterial injection of small quantities at two hour intervals. The total quantity of injection fluid is in excess of ordinary injection done at a time. The injection is continued for three or four times. The venous drain tube which is kept closed is opened a little before and kept open a little after starting another dose of injection. This is the best method.

NORMAL EMBALMING: (1) ARTERIAL EMBALMING: The embalmer should wear impervious apron, cap, mask and gloves. Place the body supine on the table. The clothing on the body and surgical dressing if any should be removed and the body washed with an antiseptic soap and warm water. Rigor mortis, if present should be broken by bending, massaging, rotating the head, etc. The nostrils are cleaned and plugged with a wad of cotton soaked in arterial solution. The cheek may be filled out with cotton soaked in arterial solution. The mouth should

be closed. The eyelids should be closed. If the eyeball is sunk arterial solution should be injected into the orbit and eyeball. The head should be elevated 8 to 10 cm. and placed on a head rest, and the feet raised to facilitate drainage. The anal orifice should be plugged with cotton wool soaked in cavity fluid. The vagina should be plugged similarly.

CHOICE OF VESSELS: The nearer the vessel to the heart, the better the result, especially for drainage. A single-point injection often leaves patches of areas unfixed by the embalming fluid. Multiple sites of injection may be required in cases of traumatic death, autopsied cases and postmortem mutilations. The 'six-point' injection involves right and left common carotid arteries for the head and neck, right and left axillary arteries for the upper limbs and the right and left femoral arteries for the lower limbs. The trunk may be injected sending the fluid through these arteries towards the heart. On completion of the injection, the vessels should be ligated to prevent leakage of embalming fluid. Each side of the face should be separately injected through the common carotid artery of the side to ensure equal distribution of the fluid and to prevent distortion of the face due to over-injection. After injection of one artery, it should be ligated before injecting into other artery.

The desired arteries and veins are dissected and raised to the surface. The vessels are cannulated with suitable tubes. It is better to drain one vessel from each of the high and low drainage points. Discontinuous method of injection with small quantities (about one to two litres) of arterial fluid, followed by drainage is the best method. The particular limb in relation to the artery exposed should be embalmed first and the artery is tied off. The vein is left open till the end. Mechanical injection is better than the gravity injector or manual pumps. The drainage tubes should be left in place, till the cavity treatment is over. All drainage points should be ligated after completion to prevent a leak.

CAVITY EMBALMING: (1) CLOSED CAVITY TREATMENT: If possible, cavity treatment should be done after half to one hour, which will allow for the hardening of the viscera, and facilitate piercing of the gut. A motorised aspirator if available is better. A 30 cm. long trocar is inserted into the abdomen through a small incision, about 5 to 6 cm above the umbilicus in the midline. The trocar is first directed upwards, backwards and to the left to pierce and aspirate the stomach. Then the trocar is slightly withdrawn and pushed up towards the right to pierce the right side of the heart. Next the right and left pleural sacs are reached by piercing the diaphragm and aspirated. Next several punctures are made in the small intestine, caecum and colon to suck out the contents. The urinary bladder, sigmoid colon and rectum should be aspirated. Next, one litre of cavity fluid should be injected distributing it evenly throughout the cavities. The following fluid is recommended.

Formalin	60%
Methanol	25%
Liquefied phenol	10%
Sodium lauryl sulphate	1%
Mercuric chloride	1%
Eucalyptus oil	1%

POST-EMBALMING DRAINAGE: Much of the undrained blood stagnates in the large vessels of the trunk. This should be removed by aspiration during cavity treatment.

If the body has to be transported to a distant place, it should be securely covered and fixed with an impermeable protective cover. To absorb unexpected leakage and discharges from the body, it should be covered with sawdust.

An embalmed body if left uncared will ultimately turn into a mummy due to dehydration. It will be shrivelled to skin and bones.

(2) Mummification.

(3) Adipocere formation.

(4) Freezing: If the body is frozen soon after death and kept in that state, it will be preserved for decades.

(5) Bodies which have been in water or soil containing antiseptic substances, sometimes become impregnated with these material and do not decompose.

(6) By injection of solution of arsenic, lead sulphide and potassium carbonate into the femoral artery or into the aorta, bodies are preserved for the purpose of dissection.

ESTIMATION OF POST-MORTEM INTERVAL

The interval between death, and the time of examination of a body is known as post-mortem interval. This is important (1) to know when the crime was committed, (2) it gives the police a starting point for their inquiries, and allows them to deal more efficiently with the information available, (3) it might enable to exclude some suspects and the search for the likely culprits started earlier, (4) to confirm or disprove an alibi, (5) to check on a suspect's statements.

In civil cases, it might determine who inherits property or whether an insurance policy was in force.

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. One should never give a single estimate of the time since death, but use a range of times, between which the death was presumed to have taken place. The longer the post-mortem interval, the wider is the range of estimate, i.e. the less accurate the estimate of the interval. In determining time of death, the doctor should not over-interpret what he sees and should not make dogmatic, unsupportable and potentially inaccurate statement. First all available history should be taken, and then local physical or environmental factors at the scene of crime, such as presence of fires and domestic heating, open windows, atmospheric temperature, etc. The range of time provided is at best an educated guess, based on knowledge and experience and subject to error.

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 : Entomology is the study of the form and behaviour of insects. **Myiasis** is a condition caused by infestation of the body by fly maggots. Flies (Musca domestica and M.vicinia) may deposit their eggs (pearly-white, one mm. long; about 120 to 150 eggs at one sitting) 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 8 to 24 hours in summer, larvae or maggots (white, segmented, 1 to 2 mm. in length at birth; 12 mm. when full grown) 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 (dark-brown, barrel-shaped, 6 mm. length) in 3 to 6 days, and the pupae become adult flies in three to six days. The complete life cycle from egg to adult may take 5 to 6 days in summer and 8 to 20 days in winter. Body lice usually remain alive for three to six days, after death of a person.

(6) Gastrointestinal and Urinary Tract: The amount of stomach contents and the extent of their digestion may be helpful to estimate the time of death, if the hour at which the deceased took his last meal is known. The gastric emptying of either liquids or solids is subject to relatively wide differences in the same and different individuals even if the same meal is ingested. The state of digestion and transportation rate of food from the stomach into the duodenum depends on several factors, such as anatomical, physiological, pathological, psychological, agonal, kind of food, etc., which contribute to great intra- and interindividual variability of gastric emptying. The quantity and digestive state of gastric contents is modified by the following factors: (1) The total quantity of food taken at a meal. (2) Additional snacks taken between a meal. (3) The ratio of solid to liquid in the meal. (4) The carbohydrate/fat content. (5) Marked variation between individuals. (6) Variation in the same individual from day to day. (7) Dramatic variations due to psychogenic and endocrine factors.

The stomach contents do not enter the duodenum after death. Digestion of the stomach contents may continue for some time after death. This may create further difficulties. It seems that there are no significant differences in solid food emptying rates between young and old persons. The emptying rate increases directly with meal weight. The stomach empties gradually. The stomach usually starts to empty within ten minutes after the first mouthful has entered. The bulk of the meal leaves the stomach within two hours. A light meal (small volume) usually leaves the stomach within 1 to 2 hours after being eaten, a medium-sized meal requires 3 to 4 hours and a heavy meal 5 to 8 hours. A head injury, physical or mental shock or stress, may completely inhibit the secretion of gastric juice, the motility of the stomach and the opening of the pylorus, and undigested food may be seen after more than 24 hours. Any illness or emotional stress, may prolong the emptying time for many hours. Digestion is delayed during sleep and is probably suspended during coma. Strong alcohols irritate the mucosa and tend to delay emptying. Hypermotility caused by emotional disturbance can result in rapid passage of food through the intestines causing diarrhoea. A carbohydrate meal leaves the stomach more rapidly than a protein meal, because carbohydrates are reduced to a semi-fluid state rapidly and a protein

meal leaves the stomach more rapidly than fatty meal. Fluids and semi-fluids leave the stomach very rapidly (within two hours), after being swallowed. If water is ingested with a solid meal, the water is emptied rapidly and separately and is not influenced by either the weight or total calories of the accompanying solid meal. Milk leaves rapidly, whereas meat and pulses are retained longer. Meat, green vegetables and roots cannot be recognised after four hours. The head of the meal reaches the hepatic flexure in about six hours, splenic flexure in 9 to 12 hours, and pelvic colon in 12 to 18 hours. The actual recognition of stomach contents may be useful as it may indicate what the last meal consisted of and narrow the time of death to the interval between two meals, provided the type of meal is definitely known. Unless death was sudden and unexpected, reliance cannot be placed upon the state of digestion or the volume of stomach contents. If the stomach is full containing undigested food, it can be said that death occurred within two to four hours of the eating of the last meal. Any estimate of a postmortem interval is only an opinion based upon probabilities and is subject to limitations. The presence of food in the small intestine or of faecal matter in large intestine is not of much value in determining the time of death.

(7) Cerebrospinal fluid: Cisternal fluid is examined. The amount of potassium increases at a constant rate in relation to the temperature of the body during the first 20 hours. The concentrations of sodium, calcium and magnesium have no obvious relationship to time since death.

(8) Blood: There is a progressive increase of lactic acid. The values increase by 50 to 75 fold in 12 to 24 hours, the steepest increase occurring in first 6 to 8 hours. Aminoacid nitrogen is less than 14 mg/dl up to 10 hours, but rises to 30 mg/dl by 48 hours. Acid phosphatase levels increase 20 times by 48 hours. Amylase levels increase 3 to 4 times on second day. Glutamate-oxalate transaminase (AST4) and lactate dehydrogenase (LDH) increase linearly over the first 60 hours. Sodium levels fall by 0.9m Eq/l per hour. Organic phosphorus level in serum reach 20 mEq/l at 18 hours after death.

(9) Pericardial Fluid: 10 to 15 ml can be obtained, as opposed to 1 to 2 ml of vitreous humour and 5 ml of CSF. Constituents are not helpful in estimating time since death.

(10) Synovial Fluid: There is linear rise of potassium, which increases more than double within the first two days.

(11) Vitreous Humour: The potassium concentration at the time of death is 5 to 8 mmol/l The rise of potassium concentration per hour is 0.17 to 0.238 mmol per hour. The reason for varying range may be due to electrolyte imbalances at the time of death. The varriation between the two eyes may be up to 10% The relationship between potassium concentration and time after death is not completely linear, as it depends on the degree and rapidity of decomposition. The slope is 0.2 mmol/l per hour. Madea and Henssge proposed the following formula.

PMI = 5.26 x K conc – 30.9

The 95% limits of confidence are ±20 hours up to 100 hours. The rise is mainly due to diffusion from the retina into the centre of the globe. Temperature, chillness and urea retention affect the range.

There is a linear rise of hypoxanthine(Hx) up to 120 hours. The rise begins immediately after death. There is a correlation between vitreous hypoxanthine and vitreous potassium values. The 95% confidence limits are ± 32 hours.

Chlorides decrease at less than 1 mmol/l/h and sodium by about 0.9 mmol/l/h. Urea exceeding 150 mmol/l indicates uraemia. The vitreous glucose usually falls after death and can reach zero within a few hours. A vitreous glucose of more than 11.1 mmol/l indictes diabetes, and less than 1.4 mmol/l of hypoglycaemia.

(12) Muscle Enzymes: Myofibrillar protease activity increases linearly and creatinine phosphokinase decreases linearly after death. A strong positive correlation is found between the ratio of non-protein nitrogen and total soluble protein and also creatinine concentration and time since death.

All the biochemical changes are temperature dependent. The above findings apply to cold and temperate climates.

(13) Hair: Hair does not grow after death; the contraction of the skin towards the hair roots gives the illusion of growth. A sample of hair is shaved from the chin and its length measured. From this, a rough estimation of the time since the last shave can be made, for beard hair grows at the rate of 0.4 mm. per day and nails at 0.1 mm per day.

(14) The Scene of Death : The dates on mail or newspapers, sales receipts or dated slips of paper

in the deceased's pockets, when the neighbours last saw the person, degree of coagulation of milk, state of food on a table, etc. may be valuable. The state of dress should be noted as regards whether the person is fully dressed or in the night dress. If the watch has stopped, the hour at which it has stopped should be noted. If it is still going, the time it takes to run down should be observed. In drowning, the watch commonly stops shortly after immersion. If a corpse lies undisturbed on growing grass, or plantbearing soil, underlying grass or vegetation soon dries, turns yellow or brown and dies.

Radioactive Carbon : C_{14} accumulates in living organic matter. The C_{14} content of the organism is steadily maintained as long as it lives. After death, the radioactivity gradually weakens taking about 5,600 years to reach half its initial activity. A simple carbon compound, such as CO_2 or acetylene, or even carbon itself is prepared from the bones, and the radioactivity is estimated. For medico-legal purposes, radiocarbon dating is not useful as the technique cannot date bones less than a century old.

Effects of keeping Cadavers in Refrigerating Chamber: If the body is refrigerated soon after death, the onset of rigor mortis is delayed. Reddish patches appear on the surface, especially in the hypostatic region and sometimes in internal organs. The blood is bright-red and injuries like abrasions and contusions have an intensified appearance. The tissues become hard.

Tower of silence is used by Parsees to dispose off dead bodies.

PRESUMPTION OF DEATH: This condition has only legal importance, and medical evidence is rarely necessary. This question arises in cases of inheritance of property or in obtaining insurance money, when a person is alleged to have been dead and the body is not found. Under the Indian Evidence Act (S107), a person is presumed to be alive, if there is nothing to suggest the probability of death within 30 years. But if proof is produced that the same person has not been heard of for seven years by his friends and relatives, death is presumed (S. 108, I.E.A.).

PRESUMPTION OF SURVIVORSHIP: The question of presumption of survivorship may arise in connection with inheritance of property, when two or more persons die in a common disaster, e.g., earthquake, shipwreck, plane-crash accident, etc. The question may arise as to who survived longest, and if no direct evidence on this is available, the question becomes one of presumption. The case is decided by the facts and evidence available. In the absence of such evidence, age, sex, constitution, nature and severity of injuries and the mode of death should be taken into consideration.

Commorientes: Persons who die together on the same occasion, where it cannot be ascertained who died first.

CHAPTER 8
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. **Trauma** is an injury inflicted by force on a living tissue. The question of wounds may be raised in a Court of law in both civil and criminal cases.

Classification of injuries : Medical : (I) Mechanical Injuries:(A) Due to blunt force: (1) Abrasions. (2) Contusions. (3) Lacerations. (4) Fracures and dislocations. (B) Due to sharp force: (1) Incised wounds. (2) Chop wounds. (3) Stab wounds. (C) Firearms: (1) Firearm wounds.

(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) Physical: Electricity, lightning, X-rays, radioactive substances, etc.

(V) Explosions

Legal : (1) Simple, and (2) Grievous.

Medico-legal : (1) Suicide. (2) Homicide. (3) Accident. (4) Fabricated. (5) Defence.

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. In the first case, the counterforce is provided by the inertia of the body, and in the second case by the rigidity of some stationary object against which he falls. A combination of these two events is seen in most cases. Due to the impact between the forward moving force and the counterforce, energy is transferred to the tissues of the body, which causes a change in their state of rest or motion. The human body contains many complex tissues which greatly vary in their physical properties, such as state of solidity, fluidity, density and elasticity, and because of this a change in the state of rest or motion of the body produced by a forceful impact does not affect the tissues uniformly. Some of the energy is spent in moving the body as a whole, but most of the energy may cause non-uniform motion of localised parts of the body, due to which the affected tissues will be subjected to compression or to traction strains or to a combination of both. All the body tissues, except those which contain gas, are resistant to compression, i.e., they resist force tending to reduce their volumes. Mechanical force does not cause compression of the tissue but causes their displacement and deformation, and traction strains are produced in the affected tissues. Such strains may be due to forces causing simple elongation of tissues, but they may be due to more complex mechanism, such as bending, torsion or shearing. A shear strain is a strain which is produced in a body by the forceful alteration of its shape but not its volume. It causes or tends to cause two parts of a body in contact with each other to slide relatively to each other in a direction parallel to their plane of contact. Because of the great variation in the resistance of the different tissues to traction, they rupture with varying ease, as their cohesiveness is exceeded. The rigid tissues like bones resist deformation, but if the limits of their elasticity is exceeded fracture occurs. The soft tissues are plastic, and as such, mechanical force alters their shape, which is limited by the cohesion between the tissue cells, connective and vascular tissue frameworks and capsules of organs. Soft tissues rupture when they are stretched beyond the limits of their tensile strength.

ABRASIONS

An abrasion (gravel rash) is a destruction of the skin, which usually involves the superficial layers of the epidermis only. Thickness of skin is 1.6 mm. They are caused by friction against a rough surface or by compression, such as 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.In its simplest form, the epidermal cells are flattened and their nuclei are elongated. If sufficient friction is applied, partial or complete removal of the epithelium may occur and the superficial layer of dermis is damaged. Many abrasions have some deeper areas of subepidermal damage which may result in superficial scarring. 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. Abrasions vary in size, depending on the

extent of the body surface exposed to the abrading force. 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 : A scratch (linear abrasion) may be an abrasion with length but no significant width, or a very superficial incision, depending on the agent. A scratch produced from the tip of the knife or razor can be called a **point scratch**. They are caused by a sharp or pointed object, not sharp enough to incise, but pointed enough to gouge or scratch, i.e. remove a portion of the skin's surface, 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. When the skin is gripped in a static fashion, fingernail abrasions may be straight or curved, one to 2 mm breadth, often about half to one cm. long, wide at the start, narrow at the end. They may be parallel linear scratches if the fingers are dragged down the skin. As the skin is put under lateral tension when it is indented by nails, it may distort, so that when the tension is released the elasticity of the skin causes it to return to its original position, carrying the nail mark with it. The curve may then reverse to form either a straight line or a convexity, but this is always not true. Pointed nails are more likely than those with straight edges to give these paradoxical results. A thorn or pin produces a narrow scratch which tails off.

(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 (grooves or furrows) 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 furrow may be broad at one end, and narrow in the opposite direction. Usually, the skin is uniformly denuded at the start. The epidermis is scraped away, destroyed or detached. A glancing kick with a boot also produces a graze. These abrasions are commonly seen in a road accident. Many abrasions extend into the dermis, because of the corrugations of the dermal papillae, and bleeding occurs. Deep abrasions have a typical punctate or spotty appearance. An abrasion caused by violent lateral (tangential) rubbing against a surface as in dragging over the ground is called **brush burn or gravel rash.** It is a scraping injury over a large area. **"Friction burn" (scuff or brush abrasion)** is an extensive, superficial, reddened excoriated area without serous ooze or bleeding and with little or no linear mark. It may occur due to tangential contact with a smooth surface or when the skin is covered by clothing.

(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. In this type, the movement is slight and largely directed inwards. 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. The abrasion is slightly

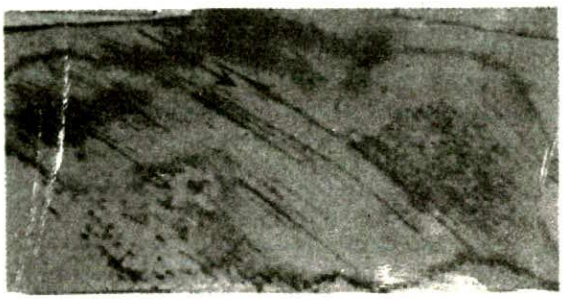


Fig (8–1) Abrasion indicating direction of force.

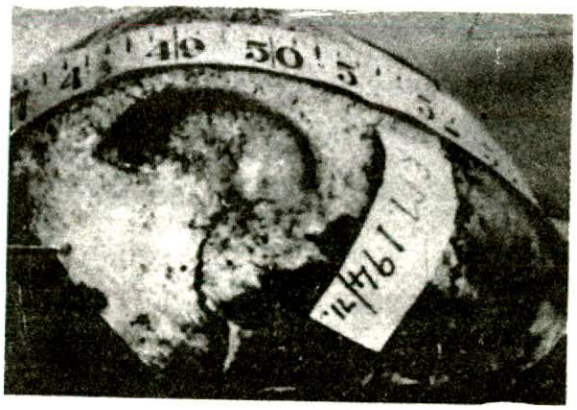


Fig. (8–2) Abrasion of the skull due to tearing force produced by the automobile wheel causing avulsion of the scalp.

depressed below the surface, unless there is bulging due to underlying contusion or local oedema. If the impact is forcible, the dermis is damaged with an underlying bruise. 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 (Fig. 8-3) Impact by a solid object may produce abrasion at the periphery where the skin is forced downwards. If a person strikes a flat and relatively smooth surface, an abrasion can be produced which shows little or no linear markings, as in traffic accidents.

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. If the skin is struck with a weapon having a patterned surface, the ridges of the object damage the epidermis, and the skin may be compressed into the cavities of the pattern, causing intradermal bruise. Other examples of patterned abrasion are : imprint of bicycle chain, weave of coarse fabrics, the spiral of electric wires, ropes, serrated knife, etc. Multi-thonged whip, such as a cat-o-nine-tails, leaves a series of linear abrasions or superficial tears.



Fig. (8–3) Patterned abrasion. Imprint of tyre tread across the back due to run over by a bus.

If there are multiple minor abrasions on a particular area, they can be described together as present over an area of (giving dimensions) on the particular anatomic region.

Age of the Abrasions : Abrasions heal from the periphery by new growth of epithelial cells. 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** : Dark brown to browinsh-black scab. Epithelium grows and covers defect under the scab. Reticulum fibres are seen in about 6 to 8 days. **After 7 days :** Scab dries, shrinks and falls off, leaving depigmented area underneath, which gets gradually pigmented.

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. The last stage is regression which begins about 12 days. During this phase, the epithelium is remodelled and becomes thinner and even atrophic.

Ante-mortem and Post-mortem Abrasions: Abrasions produced slightly before or after death cannot be differentiated even by microscopic examination. In superficial lesions or when decomposition is advanced, differentiation is difficult. After death, the abraded epidermis becomes stiff,

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

Trait	Ante-mortem abrasions	Post-mortem abrasions
(1) Site :	Anywhere on the body.	Usually over bony prominences.
(2) Colour :	Bright reddish-brown.	Yellowish, translucent, and parchment-like.
(3) Exudation :	More; scab slightly raised.	Less; scab often lies slightly below the level of skin.
(4) Microscopic :	Intravital reaction and congestion seen.	No intravital reaction and no congestion.

leathery, and parchment-like, brown, more prominent, and may be mistaken for burns. On drying, abrasions become dark-brown or even black. In a body recovered from water, abrasions may not be seen on first inspection, but they are easily seen after the skin dries. Abrasions may be produced after death when a body is dragged away from the scene of crime. The distribution of such abrasions depends upon the position of the body while it is being dragged. If the body was dragged face downwards, linear abrasions may be produced on the front of the face, trunk and lower limbs.

CIRCUMSTANCES OF INJURIES : Abrasions are usually seen in accidents and assaults. Suicidal abrasions are rare. 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 eyebrow, 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.

Differential Diagnosis : (1) Erosions of the Skin Produced by Ants : Ants produce brown erosions with irregular margins of the superficial layers of the skin. They are most commonly found at mucocutaneous junctions, about the eyelids, nostrils, mouth, ears, knuckles, axillae, groins, and genitalia. They are also seen in the moist folds of the skin. Sometimes they are localised, and may simulate ante-mortem abrasions. Examination by hand lens shows multiple crescentshaped, sand-like bite marks. Each one is separated by normal skin. Vital reaction is not seen. **(2) Excoriations of the Skin by Excreta:** In infants, slight inflammation with excoriation may be seen in the napkin area at the time of the death. After death, these areas become dry, depressed and parchment-like and the colour varies from pale- yellow to deep-copper. **(3) Pressure sores. (4) Drying of the skin** of the scrotum produces reddish-brown or yellow colouration resembling abrasion.

CONTUSIONS (BRUISES)

A contusion is an effusion of blood into the tissues, due to the rupture of blood vessels (veins, venules and arterioles), caused by blunt trauma. The bruise from a blunt impact is usually situated in the subcutaneous tissues. Contusions may be present not only in skin, but also in internal organs, such as the lung, heart, brain and 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. Petechial bruises are finely mottled or stippled. If the petechiae become larger and confluent, they are called ecchymoses. A fresh bruise is usually tender and slightly raised above the surface of the skin, and even a deep-seated bruise shows some swelling when compared with the opposite limb or part of the body. A bruise has lighter colour in the centre because extravasated blood is pushed outward by the impact. Mongolian spot (hyperpigmented skin in the lumbosacral region) should not be confused with contusion.

Size : Bruises vary in size from pinhead to large collections of blood in the tissues. 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. Development of marked tissue swelling

in the vicinity of a bruise usually results in loss of its original shape. As a general rule, the greater the force of violence used, the more extensive will be the bruise.

Factors modifying size and shape:

(1) 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. If 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. Bruising is not seen if the injured part is thickly covered, or if the weapon used is a yielding one, such as sand bag. Bruising may be absent if the pressure be continued until death occurs. Even a severe injury may produce little haemorrhage, if it was preceded by an injury which produced deep shock. Resilient areas, such as the abdominal wall and buttock bruise less. Bruising is relatively more marked on tissues overlying bone. Chronic alcoholics bruise easily. Bruising of the scalp is better felt than seen. Bruising of the scalp with fluctuant centres can simulate depressed fracture.

(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.

(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.

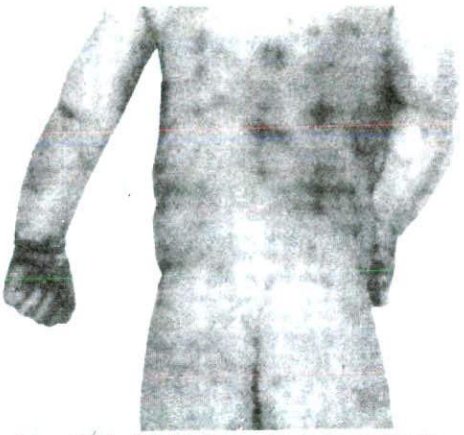


Fig. (8–4). Multiple contusions on back.

(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.

(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, or percolated or migratory contusion).** 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

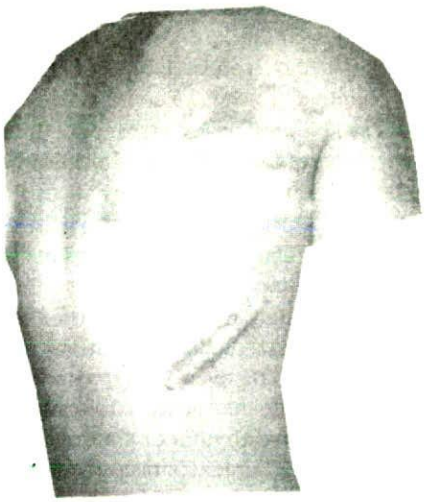


Fig. (8–5). Patterned bruise caused by a stick.

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 : (1) 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. (2) A blow from a solid body, such as a hammer, or the closed fist usually produces a rounded bruise. (3) Bruises 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. (4) 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. Blood from the base of the dent gets displaced to the sides due to the pressure by the object. 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., stick, the edges of the bruise may be irregular and the width may be greater due to infiltration of blood in the surrounding tissues along the edges of the bruise. A blow with a rigid weapon like a stick on a curved surface of the body, in a region where the soft tissues are particularly pliable, e.g., the buttocks, compress the tissues under the force of impact. In such case, the contusion is not limited to the maximum convexity of the affected part, but it may extend over the whole of the curved surface. (5) When the body is struck by a broad flat weapon, such as a plank, the edges of the plank may cause parallel bruises in the skin, separated by apparently normal tissue. (6) Bruises caused by blows from whips are elongated, curve over prominences, and may partially encircle a limb or the body. They are seen as two parallel lines, the distance between which is roughly equal to the diameter of the whip. (7) Bruises made by pliable canes are similar to those due to whip, but never encircle a limb or curve round the sides of the body. (8) Bruises from straps, belts or chains, leave a definite imprint. (9) A woven, spiral or plaited ligature may sometimes produce a patterned bruise. (10) Contact injuries from firearms may produce abrasion with bruising indicating the outline of the muzzle of the weapon. (11) Patterned bruising is also seen in motor car accidents. (12) Suction or biting on sides of the neck or the breasts, during erotic love-making or sexual intercourse produces elliptical patterned bruises. They frequently consist of a shower of petechial haemorrhages, which may be confluent. They may sometimes reproduce the shape of the upper and lower lips. (13) In a bruise caused by impact with a patterned object, the haemorrhage which is relatively small may be sharply defined if it lies in the immediate subepidermal layer, but the pattern is distinct due to translucency because of the thin layer that overlies it. Such contusions are commonly seen when the impacting object has alternating ridges and grooves (such as the tread of a motor tyre in a traffic accident) as the skin will be forced into the grooves and distorted. Intradermal bleeding will occur and the red lines may be produced not by the ridges but by the grooves, by a squeezing or bursting effect on the cutaneous capillaries. (14) Forceful compression with the sole or the heel may imprint an intradermal bruise of the pattern of the sole or heel of the shoe. Kicking and jumping on a person combined is known as **'stomping'.** (15) If a violent blow is struck on a clothed area of the body, or if the clothing is grabbed and twisted over the skin, petechial haemorrhages occur within the skin

reproducing the texture of clothing.

Delayed Bruising : A superficial bruise appears immediately as a dark-red swelling. A deep bruise may take several hours, or one or two days to appear and deeper extravasation of blood may never appear. Therefore, one more examination should be carried out 48 hours after the first examination, to note bruises of slower development. Occasionally, when an injury is produced before death, the bruise may appear some time after death, due to further escape of blood from the ruptured vessels due to gravitation, and rapid haemolysis of stagnant blood, the pigment diffusing locally and producing a stain on the surface **(come-out bruise).** This may explain the difference of opinion between two observers, who have examined the person or the body at different times. The pressure of the gases of putrefaction may cause the extravasated blood to extend along the tissue spaces and give rise to a false impression of the extent of ante-mortem bruising. Haemolysis of extravasated red cells and diffusion of pigment into the surrounding tissues may also cause post-mortem extension of bruise. Therefore, in assessing the extent of bruising, the postmortem interval should be considered. The examination of whole body by ultraviolet light will sometimes clearly show otherwise undetectable areas of bruising. At autopsy, when the superficial tissues are drained of blood, contusions may become more prominent and extensive than before. Surgical removal of the corneas for transplant purposes can cause haemorrhage in the eyelids simulating ante-mortem trauma. Removal of the vitreous soon after death can also cause scleral haemorrhage.

Deep Tissue and Organ Contusions : All organs can be contused. A contusion of the brain may initiate enough swelling with gradual accumulation of acid by-products of metabolism, with further swelling and impairment of function, confusion, coma and death. Contusions in vital centres, e.g., which 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 more vascular the area, the smaller the contusion, and the healthier the individual, the more rapid will be the healing. 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 starts at the periphery and extends inwards to the centre.

Table (8–2) Difference between hypostasis and bruise.

Trait	Hypostasis	Bruise
(1) Cause:	Due to distension of vessels with blood in the dermis.	Due to ruptured vessels which may be superficial or deep.
(2) Site:	Occurs over extensive area of the most dependent parts.	Occurs at the site of and surrounding the injury; may appear anywhere on the body.
(3) Appearance:	No elevation of the involved area.	Often swollen because of extravasated blood and oedema.
(4) Epidermis:	Not abraded.	May be abraded.
(5) Margins:	Clearly defined.	Merge with surrounding area.
(6) Colour:	Uniform bluish–purple in colour.	Old bruises are of different colour. Fresh bruises may appear more intense than the adjacent hypostatic area.
(7) Incision:	On incision blood is seen in blood vessels, which can be easily washed away. Subcutaneous tissues are pale.	Shows extravasation of blood into the surrounding tissues which is firmly clotted and cannot be washed by gentle stream of water. Subcutaneous tissues are deep reddish-black.
(8) Effect of pressure:	Absent in areas of the body which are even under slight pressure.	Little lighter over the area of pressure or support.

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.

The rate of colour change is quite variable, not only between persons, but in the same person and from bruise to bruise. Bruises in children change colour rapidly and may be completely absorbed in a few days. In interpreting the age of a bruise by colour changes, one should be very cautious.

When bruising is extensive and deeply situated, the colour takes a longer time to appear externally. Towards the end stage of healing process, large histiocytes containing coarse granules of haemosiderin pigment can be seen microscopically in section of contused area. The site of a bruise may contain crystals of haematoidin for a long period after the injury. It is difficult to estimate the exact age of a bruise with any degree of certainty even by microscopic examination. Subconjunctival ecchymoses do not undergo usual colour changes. They are at first bright red, then yellow before disappearing. In old people, healing of bruise is very slow. A bruise sustained at the time of carbon monoxide poisoning is likely to have a bright-red colour.

Ante-mortem and Post-mortem Bruising : In ante-mortem bruising, there is swelling, damage to epithelium, extravasation, coagulation and infiltration of the tissues with blood and colour changes. These signs are absent in post-mortem bruises.

Contusions and abrasions produced immediately before death show a marked decrease in the acidic mucopolysaccharides of the connective tissue ground substance, as demonstrated by Alcian Blue or dialyzed iron technique. Acidic mucopolysaccharide is absent in contusions more than an hour old, but reappears in the bruises several days old showing the increase of connective tissue. On microscopic examination, the presence of tissue reaction of a degree beyond a margination and limited emigration of the white cells indicates that the contusion was probably ante-mortem If the red cells have lost their shape and stainin characteristics, and if iron containing pigment is four either at the site of injury or in the regional lym nodes, probably 12 hours have passed after the inju It is of some value in distinguishing cereb haemorrhage occurring due to accident from natu haemorrhage, which could have been occurring f some time, and which may have caused the accide

Appreciable bruising does not occur 2 minut after death due to arrest of circulation, but by usin great violence, small bruises can be produced up t 3 hours after death, in areas where the tissues car be forcibly compressed against bone and also in hypostatic area, e.g., the back of the scalp, if the body is dropped on the ground, or on trolleys or post-mortem tables. The margins of post-mortem bruises are usually quite sharply defined, and those of ante-mortem bruises are less sharp or indistinct for the greater part, indicating vital reaction in the damaged tissues. Sometimes, haemorrhages are seen in areas of lividity on the arms or shoulders of fat persons without evidence of trauma on other parts of the body. These haemorrhages are produced by

Table (8–3) Difference between true bruise and artificial bruise.

	Trait	Artificial bruise	True bruise
(1)	Cause:	Juice of marking nut, calotropis or plumbago rosea.	Trauma.
(2)	Site :	Exposed accessible parts.	Anywhere.
(3)	Colour:	Dark-brown.	Typical colour changes.
(4)	Shape:	Irregular.	Usually round.
(5)	Margins:	Well-defined and regular, covered with small vesicles.	Not well-defined, diffuse and irregular; no vesicles.
(6)	Redness and inflammation:	Seen in the surrounding skin.	Seen in the site.
(7)	Contents:	Acrid serum.	Extravasated blood.
(8)	Itching:	Present.	Absent.
(9)	Vesicles:	May be found on fingertips and on other parts of the body due to scratching.	Absent.
(10)	Chemical tests:	Positive for the chemical.	Negative.

tearing of small veins in the skin when the body is lifted from the scene of death. At autopsy blood drains from blood vessels so that the deep bruises may show up against the white areas as the blood in the contusions will not drain.

In decomposed bodies, especially in the scalp, haemolysis of red cells produces a diffuse discolouration of the soft tissues, due to which it becomes impossible to differentiate between an ante-mortem contusion and an area of postmortem hypostasis. In hypostatic areas blood vessels break down with leakage of red cells into the soft tissue which haemolyse due to decomposition; erythrocytes in soft tissue in a contusion also haemolyse, and as such the apperances are similar.

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 of the neck can be demonstrated by reflecting the various structures of the neck in layers. 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. (a) When the arms are grasped, there may be 3 or 4 bruises on one side and one larger bruise on the opposite side, from the fingers and thumb respectively, indicating the position of the assailant in front of, or behind the victim. (b) Bruising of the arm may be a sign of restraining a person. (c) Bruising of the shoulder blades indicate firm pressure on the body against the ground or other resisting surface. (d) In manual strangulation, the position and number of bruises and nail marks may give an indication of the method of attack or the position of the assailant. (e) Bruising of thigh especially inner aspect, and of genitalia indicates rape. (5) In open wounds, dirt, dust, grease or particles of stone or sand are usually present, which may connect the injuries to the scene of crime.

Bruises are of less value than abrasions because: (1) Their size may not correspond to the size of the weapon. (2) They may become visible several hours or even one to two days after the injury. (3) They may appear away from the actual site of injury. (4) They do not indicate the direction in which the force was applied.

Complications: (1) A contusion may contain 20 to 30 ml. of blood or even more. Multiple contusions can cause death from shock and internal haemorrhage. (2) Gangrene and death of tissue can result. (3) The pooled blood can serve as a good site for bacterial growth, especially by clostridial group. (4) Rarely, in severe sudden compression of the subcutaneous tissue, pulmonary fat embolism may occur.

Artificial Bruises : Some irritant substances, when applied to skin produce injuries, which simulate bruises (Table 8–3). They are produced to make a false charge of assault.

CIRCUMSTANCES OF INJURIES : Accidental bruises are very common and may be seen on prominences, such as the forehead, nose, elbows and knees. Presence of mud, sand, grease or oil gives an idea of the manner of causation. Multiple contusions from minor trauma are often seen in alcoholics, which may be mistaken to be caused by physical violence. Self-inflicted bruises are rare, as they are painful. They are seen over accessible areas, usually on the head, especially in a hysterical individual or the insane. Homicidal bruises may be seen on any part of the body. It is not possible to differentiate an injury caused by a fist or weapon and a fall. Contamination of the wound should be looked for.

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. Localised portions of tissue are displaced by the impact of the blunt force, which sets up traction forces and causes tearing of the tissues. Displacement of tissues occurs most commonly when soft tissues are crushed against bone, e.g. scalp, shins, shoulders and face. Unless great force is used, most lacerations require a firm base to act as an anvil for the skin and underlying tissues to be pinned against. In lacerations of soft areas, such as buttock, thigh, calf, abdomen, upper arm, etc., the lacerating agent is either a projecting point or



Fig. (8–6). Multiple lacerated wounds on the scalp.

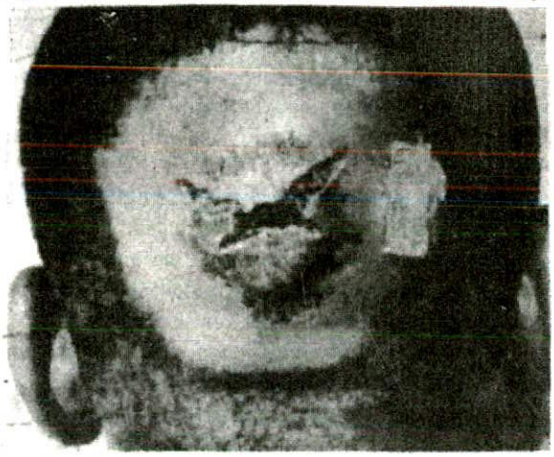


Fig. (8–7). Laceration of scalp (star-shaped).

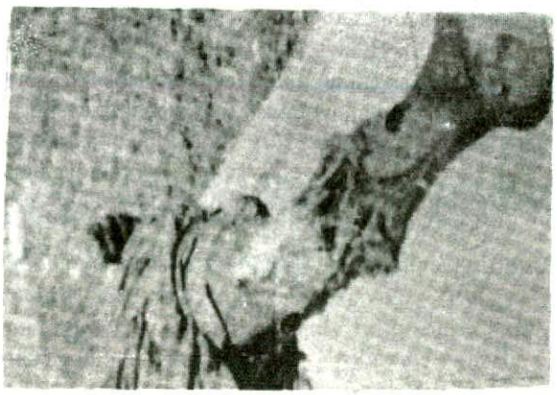


Fig. (8–8) Crush injury leg caused by run over accident.

edge, or a completely blunt object is pulled obliquely against the tension of the skin until it tears.

They are caused by blows from blunt objects, by falls on hard surfaces, by machinery, traffic

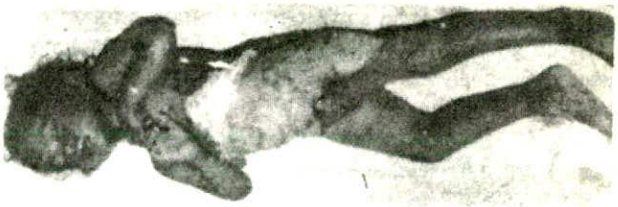


Fig. (8–9). Avulsion caused by running over of the wheel of automobile. The skin is torn away in a flap due to the grinding action of the wheel.

accidents, etc. If the force produces bleeding into adjacent tissues, the injury is a **'contused-laceration'** or **'bruised-tear'**. If the margins are abraded, it is called **"abraded laceration"** or **"scraped tear"**. If the blunt force produces extensive bruising and laceration of deeper tissues, it is called **"crushing"** injury (Fig. 8-5). 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. Scalp lacerations occur due to the tissues being crushed between skull and some hard object, such as the ground or a blunt instrument Fig (8-6&7).

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. A wound produced by a fall on the knee or elbow with the limb flexed, and by a broken glass or sharp stone also simulates incised wound.

(2) Stretch Lacerations : Overstretching of the skin, if it is fixed, will cause laceration. There is localised pressure with pull which increases until tearing occurs and produces a flap of skin, which is peeled off the underlying bone or deep fascia. This is seen in the running over by a motor vehicle, and the flap may indicate the direction of the vehicle. They can occur from kicking, and also when sudden deformity of a bone occurs after fracture, making it compound.

(3) Avulsion (shearing laceration) : An avulsion is a laceration produced by sufficient force (shearing force) delivered at an acute angle to detach (tear off) a portion of a traumatised surface or viscus from

its attachments. The shearing and grinding force by a weight, such as lorry wheel passing over a limb may produce separation of the skin from the underlying tissues (avulsion) over a relatively large area. This is called **"flaying"**(Fig. 8-8). The underlying muscles are crushed, and the bones may be fractured. The separated skin may show extensive abrasions from the rotating frictional effect of the tyre, but one portion is still in continuity with adjacent skin. Internally, organs can be avulsed or torn off in part or completely from their attachments.

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. In a case of extreme avulsion, an extremity or even the head can be torn off the body.

(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. It may be caused by blows by broken glass, or fall over a rough projected object. This is another form of overstretching.

(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. Frequently, the skin, at the margins is abraded due to the flatter portion of the striking object rubbing against the skin as it is indented by the forceful blow. The margins are contused due to the bleeding into the tissues caused by trauma. A single blow with a blunt weapon may produce more than one lacerated wound, e.g., a single blow over the side of the head may produce lacerated wounds over the parietal prominence, ear and the lower jaw. If a blunt object, e.g., a bottle strikes the skin surface, the edge at the point of impact will

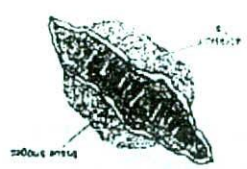


Fig. (8–10). Lacerated wound showing tissue bridges.

be sharp and turned inwards, whereas the other edge may be everted, exposing the hair follicles in the depths. In an impact over the scalp, external laceration may not occur due to the hair, but inner layers of scalp may be lacerated. If the instrument is padded or has a broad striking surface, severe fractures of the skull may occur without external laceration. Lacerations of the internal organs may be caused by : (1) direct injury of the viscera by fragments of fractured bone, (2) development of traction shears or strain shears in the viscera, (3) stretching of the visceral attachments, and (4) hydrostatic forces.

Characters : (1) Margins are irregular, ragged and uneven, and their ends are pointed or blunt, and they too show minute tears in the margins. The edges of lacerations, especially over a bony area, e.g., skull are undermined due to the crushing and tearing force of the impact. Tearing at the ends of lacerations, at angles diverging from the main laceration itself, so-called **swallow tails,** are frequently noted. (2) Bruising is seen either in the skin or the subcutaneous tissues around the wound. If the force is exerted by an object with a downward course, the lower margin of the wound is likely to be bruised more and undermined than the upper. (3) Deeper tissues are unevenly divided with tags of tissues at the bottom of the wound bridging across the margin (Fig. 8-10). **Tissue bridges** (bridging fibres) consist of nerves, blood vessels and elastic and connective tissue fibres. (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. A laceration is usually curved; the convexity of the curve points towards the direction of application of force. (a) A blunt round end may cause stellate laceration. (b) A blunt object with an edge, such as hammer head, may cause crescentic laceration (patterned laceration). (c) Long, thin objects, such as pipes, tend to produce linear lacerations, while objects with flat surfaces

produce irregular, ragged, or Y-shaped lacerations. (10) If the impact is from an angle, the skin on side of wound opposite to direction of motion is usually torn free or undermined for a variable distance. The other side, i.e. the side from which the blow was delivered, will be abraded and bevelled. If the impact was produced by an object with a downward course, the lower margin of the wound is likely to be more bruised and undermined than the upper. Gaping is seen due to the pull of elastic and muscular tissues.

Age: Age determination of laceration is difficult unless there are clear signs of healing, such as granulation tissue, fibroblast ingrowth, or organising infiltrate. Antemortem lacerations show bruising, eversion, gaping and blood-staining of margins, greater bleeding and vital reaction.

COMPLICATIONS : (1) Laceration of an internal organ may cause severe or even fatal bleeding. Temporal arteries may bleed freely as they are firmly bound and unable to contract. Multiple lacerations, involving only the skin and subcutaneous tissue, each causing some haemorrhage, may combine to cause shock and death. (2) Infection. (3) If it is located where skin stretches or is wrinkled, e.g. over joints, repeated and continued oozing of tissue fluids and blood may cause irritation, pain and dysfunction. (4) Pulmonary or systemic fat embolism may occur due to crushing of subcutaneous tissue.

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.

CIRCUMSTANCES OF INJURIES : Violent, uncoordinated muscular contractions can produce disruptive tissue stresses which produce fractures, and lacerations of tendons and muscles. Internal forces and hydrostatic pressure created by convulsions can cause mural lacerations in hollow viscera. Suicidal lacerations are usually situated on the exposed parts of the body, and mostly on the same side. In the case of a fall on the head, the abraded scalp surface will be circular and completely surrounds the laceration. A blow with a blunt, narrow object, such as the edge of an angle iron or a crowbar will produce a linear tear with finely abraded margins. Homicidal wounds are usually seen on the head.

Combinations of Abrasions, Contusions and Lacerations : Abrasions, contusions and lacerations are frequently seen together or as integral parts of one another. The same object may cause a contusion with one blow, a laceration with second, and an abrasion with a third. Sometimes, all three types of injury may result from a single blow. Sometimes, an imprint may result from an object, and it may be difficult to determine whether the imprint is primarily an abrasion or a contusion.

Punching: Punching, i.e. blows with the clenched fist will produce abrasions and contusions; laceration may occur over bony prominences. Punches on the face may split the lips, fracture the teeth, nose, jaw or maxilla and produce black eye.

Kicking: Kicking and stamping injuries are caused by a foot which is either swung or moved downwards (compression) with some force. They will produce abrasions, contusions and sometimes lacerations, which are more severe than punching. The feet may be used to stamp down on the recumbent body, causing deep injuries to abdominal organs and fractures of ribs and sternum. The pattern of the footwear may be imprinted on to the skin.

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 sharpcutting 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 blade.

Causes: (1) Striking the body with the edge of sharp-cutting weapon, (2) by drawing the weapon, and (3) by using the weapon like a saw in which case there may be more than one cut in the skin at the beginning of the wound which merge into one at the end.

A curved weapon like a sickle, produces a stab from the pointed end, and incised wound from the blade, sometimes with an intervening intact skin. When the skin becomes folded under the cutting edge of the weapon, a single movement of a sharp weapon may produce a series of incised wounds, separated one from another by bridges of normal skin.

Characters : (1)Margins : The edges are cleancut, well-defined and usually everted. The edges may be inverted, if a thin layer of muscle fibres is adherent to the skin as in the scrotum. The edges are free from contusions and abrasions. A

Table (8–4) Difference between suicidal and homicidal cut-throat wounds

Trait	Suicidal wounds	Homicidal wounds
(1) Situation :	Left side of the neck and passing across the front of the throat; rarely on both sides.	Usually on both sides.
(2) Level :	High; above the thyroid cartilage.	Low; on or below the thyroid cartilage.
(3) Direction:	Above downwards and from left to right in right–handed person. Sometimes horizontal cuts are seen which do not show variation in depth at either end.	Transverse or from below upwards. If attacked from the right side of victim, the wound runs from left to right; if from behind, it may resemble suicidal wound.
4) Number of wounds:	Multiple, may be 20 to 30, superficial, parallel and merged with the main wound; rarely single.	Multiple, cross each other at a deep level; not repeated in depths of the main wound.
(5) Edges :	Usually ragged due to overlapping of multiple superficial incisions.	Sharp and clean-cut; bevelling may be seen.
(6) Hesitation cuts :	Present.	Absent.
(7) Tailing :	Present.	Absent.
(8) Severity:	Usually less severe. One wound severe but sometimes 2 or 3.	More severe; all the tissues including the vertebrae may be cut.
(9) Wounds in other parts of the body :	Often present across wrists, groin, thighs, ankles, or knees; and rarely on the back of neck.	No wounds on wrists, etc. but severe injuries usually on the head and neck.
(10) Defence wounds:	Absent; unintentional cuts may be found on the fingers if a blade has been used.	Present.
(11) Hands:	Weapon may be firmly grasped due to cadaveric spasm.	Fragments of clothing, hair, etc., may be grasped.
(12) Weapon:	Usually present.	Usually absent.
(13) Vessels:	As head is thrown back, carotid artery is drawn beneath sternomastoid and against the spine and usually escapes injury.	Jugular vein and carotid artery are likely to be cut.
(14) Blood stains:	If standing, stains on the mirror and on front of body and clothes running from above down-wards and splashes on feet.	If asleep, blood runs down on both sides of the neck and collects behind the neck and shoulder; stains found on both palms, for the victim attempts to cover the wound.
(15) Clothes:	Not cut or damaged.	May be cut corresponding in position to those in the body; disarrangement, tears and loss of buttons.
(16) Circumstantial evidence:	Quiet place, such as bedroom or locked bath-room; usually stands in front of a mirror in order to direct the hand better; suicidal note or farewell letter may be found.	Disturbance at the scene, such as dis-arrangement of furniture in a room; trampling and crushing of vegetable matter and shrubs, or confused foot prints outside.

dull irregular-edged or nicked cutting edge may produce a wound with irregular, contused, and/or abraded margins, as the wound is caused more by the pressure applied by the weapon than by the cutting edge. A serrated knife may produce a saw-toothed cut on the body, if the blade passes obliquely or at shallow angle. The depth of the wound will not show bridging tissue.

(2) Width : The width is greater than the edge of the weapon causing it, due to retraction of the divided tissues. Shaking of the blade tends to make the wound wider than the weapon.

(3) Length : The length is greater than its width and depth, and has no relation to the cutting edge



Fig. (8–11) Suicidal cut-throat with hesitation marks.



Fig. (8–12) Homicidal cut-throat wound.

of the weapon, for it may be drawn to any distance. When the skin becomes folded under the cutting edge of the weapon, a single movement of the weapon may produce a series of incised wounds separated one from another by bridges of normal skin.

(4) Shape: It is usually spindle-shaped due to greater retraction of the edges in the centre. Gaping is greater if the underlying muscle fibres have been cut transversely or obliquely, and less when cut longitudinally. The wound may take zig-zag course if the skin is loosely attached as in axillary fold, because the skin is pushed in front of the blade before it is cut. If the blade is curved, the edges will be crescentic. If the surface is convex, the straight-bladed weapon may also produce a crescentic wound.

(5) Haemorrhage: As the vessels are cut cleanly, the haemorrhage is more. If the artery is completely cut, the bleeding will be more. Spurting of blood occurs if an artery is cut.

(6) 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.

(7) Bevelling cut : If the blade of weapon enters obliquely, the tissues will be visible at one margin, and the other margin will be undermined, and if the blade is nearly horizontal, a flap wound is caused. Bevelling can be produced by sharp weapon only. It is usually homicidal and may indicate the relative position of the assailant and the victim.

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.

Histopathology: Few minutes: Dilatation of capillaries and margination and emigration of neutrophils, reactive changes in the tissue histiocytes and swelling of the vascular endothelium. **12 hours:** Reactive changes occur in the firboblasts at the site of injury within few hours; monocytes appear in the exudate. **15 hours:** Monocytes begin to undergo mitotic division. **24 hours:** Epithelium begins to grow at the edges. Vascular buds begin to form. **72 hours:** Vascularised granulation tissue is formed. **4 to 5 days:** New fibrils are formed. **One week:** Scar formation in small wounds. The above changes are considerably modified if infection of the wound occurs.

In healing by second intention, inflammatory reaction is more intense and much larger amounts of granulation tissue are formed resulting in greater mass of scar tissue.

Wounds by Glass : Wounds produced by glass are lacerated, but can resemble incised and stab wounds. If a sharp-pointed piece of glass enters by its point, the wound has a stab-like appearance. Margins of the wound will almost always show tiny



Fig. (8–13). Homicidal incised wounds on the right side of the face and neck.

side cuts due to irregularities of the glass. Particles of glass may be found in the wound.

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

Circumstances of Incised Injuries : Suicide: The important features of self-inflicted wounds are: (1) They are multiple and parallel or nearly so, in any one area. (2) They are uniform in depth and direction. (3) They are relatively minor. (4) The fatal wounds are present on several limited accessible areas of the body, such as front of the neck, wrists, groin, and occasionally on the back of legs or on chest. **(5) Hesitation marks or tentative cuts or trial wounds:** They are cuts which are multiple, small and superficial often involving only the skin and are seen at the beginning of the incised wound (Fig. 11).

The sites of election of suicidal incised wounds are: throat, wrist and front of chest. When preliminary incisions are inflicted with some violence they may be few, but commonly they are superficial and multiple. The fatal incisions are usually made with great violence, and the large gaping wound produced by suicide should not be mistaken for homicidal wounds. When a safety razor blade is used, **unintentional cuts** are found on the fingers, where the blade has been gripped.

Suicidal incised wounds of extremities are usually found on the flexor surface of the wrists, outer side of the left arm, and forearm, the front and outer side of the thighs, and the front of the abdomen and chest. Fatal incised wounds of the arms are almost always suicidal. Suicidal wounds of chest are usually on the left side and directed downwards and inwards. A wound caused by running on knife tends to be more horizontal in direction. A person who commits suicide usually exposes the portion of the body to be incised, e.g. he may open his collar before cutting his throat, or pull up his shirt before cutting his chest or abdomen. Suicides usually do not injure the face.

HOMICIDAL WOUNDS: They are usually multiple and can occur in any region of the body. Wounds of the chest are usually present over a wider area and are more horizontal. They may be directed from below upwards which is rarely seen in suicidal wounds. Incised wounds situated on the back, or in such a position as cannot be easily reached by a suicide, are homicidal. Incised wounds on nose, ears and genitals are usually homicidal, and are inflicted on account of jealousy or revenge in cases of adultery, causing disfiguration. Cut-throat wounds cause immediate death from haemorrhage, air embolism, or inhalation of effused blood into the respiratory tract (Fig. 8-11&12).

ACCIDENTAL incised wounds are common usually in the home or workshop, and are of a minor nature. They may be caused by falling upon a sharp-cutting weapon held in the hand, or upon a sharp-pointed object or by a sharp piece of broken glass. They may be situated anywhere on the body, but are commonly seen about the hands.

Some mentally disordered persons may inflict hundreds of small wounds upon themselves. A knife, razor or broken glass may be used and a large number of cuts produced on the arms which may cross each other in groups. Small stabs may be produced on the chest or temples. Paranoid schizophrenics with a



Fig. (8–14). Child Sacrifice



Fig. (8–15). Chop wound on right upper arm.



Fig. (8–16). Cut injury of the skull. The wide gap anteriorly is caused by the heel of the axe in a frontal attack.

strong religious flavour to their delusions may remove penis, scrotum, and testes. Ears and nose may be mutilated.

In a decomposed body, it is difficult to differentiate a laceration from an incised wound. When a body with incised wounds, stab wounds, or lacerations is immersed in water soon after infliction of injuries, the blood in the wounds is lysed by water and passes into the water, and whether the injuries are antemortem or postmortem cannot be made out.

CHOP WOUNDS (Slash 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, sword, broad heavy knife, or meat cleaver. The dimensions of the wound correspond to crosssection 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. Undermining occurs in the direction towards which the chop is made (Fig. 8-15). 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. In the skull, the undermined edge of the fracture defect is the direction in which the force is exerted, and the slanted edge is the side from which the force was directed (Fig. 8-16). In case of long bones, the bone fragments get loosened on the opposite side of the force. A twisting force, after the chopping weapon has been embedded in bone, and as it is being removed can cause splintering of the bone, particularly near the ends of the chop wound. If the extremities are attacked, there may be complete or incomplete amputation of the fingers or other bones, and the joints may be separated or disarticulated. Wounds on the head and trunk are usually associated with injuries to important structures and are fatal. The cranium may be depressed, but if the weapon strikes obliquely, a piece of the skull may be removed. If the chopping blow is tangential, a disk-shaped portion of bone or skin or soft tissue may be cut away. The neck may be almost completely separated.

Most of these injuries are homicidal and usually inflicted on the exposed protions of the body like the head, face, neck, shoulders and extremities. Accidental injuries are caused by power fans, band saws or ship or airplane propellers, which may lacerate the soft tissues extensively or amputate parts of the body. Suicidal chop injuries are very rare.

STAB OR PUNCTURED WOUNDS

A stab wound is produced when force is delivered along the long axis of a narrow or pointed object, such as knife, dagger, nail, needle, spear, arrow, screw driver, etc. into the depths of the body. The wound is deeper than its length and width on skin. 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 wound of entry is larger with inverted edges, and the wound of exit is smaller



Fig. (8–17) Assessment of depth of penetration of a stab wound.

with everted edges, due to tapering of blade. 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 (hilt mark) 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. The mark will be symmetrical, if the knife strikes the skin at right angle. If the knife strikes in a downward angle, the mark will be prominent above the stab wound, and if the knife strikes in an upward angle, the abrasion will be below the stab wound. In oblique stab wounds, a knife striking from the right will have an abrasion on the right side and vice versa. In such cases, the suspected knife should be examined to determine the compatibility of the shape of the abrasion around the stab wound, with the handle of the knife in question.

(2) Length : The length of the wound is slightly less than the width of the weapon up to 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 tends to widen the wound, and the length will be more than the maximum width of the blade. If the instrument is thrust in, and is then completely withdrawn with the cutting edge dragging against one end, the wound would be extended superficially, producing a tail.

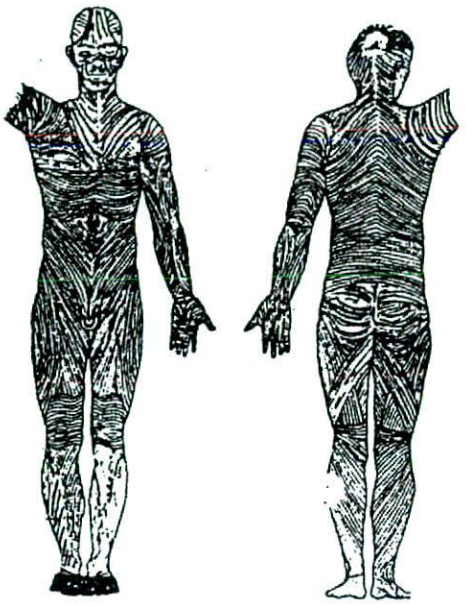


Fig. (8–18) Cleavage lines of Langer.

(3) Width: By examining multiple stab wounds in the body, the length and width of the knife blade can be determined approximately. The maximum possible width of the knife blade can be approximately determined if the edges of a gaping wound are brought together. Elasticity or laxness of the skin can change the width by one to two millimetres. A stab wound inflicted when the skin is stretched will be long and thin, which becomes shorter and broader when the skin is relaxed.

(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

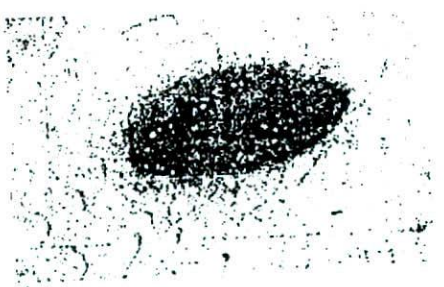


Fig. (8–19) Boat-shaped stab wound produced by a single-edged blade.

disturb a loose clot and may lead to fatal haemorrhage, or cause serious damage and may produce multiple false 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 (Fig. 8-17). In young persons, the chest is mobile and may be compressed during the stabbing. The breast, buttocks and thigh are indented by a full thrust, and the depth of wound may exceed the length of the weapon. Expansion and retraction of the chest during breathing must also be considered. Many of the internal organs are not fixed but have a variable degree of mobiliy. This should be taken into account when estimating the depth of the wounds. After the withdrawal of the weapon, the wound tends to close by expansion of the tissues along the track. The depth should be determined in the operation theatre when the wound is repaired.

The viscera of a dead body on the autopsy table are not in the same positions, as when the same person was alive and in standing position, or was bent over in a state of emotional tension, at the time of an assault. During fight, fright and flight, the victim may be moving or changing position in a variety of postures, which change by the second. When tense, the abdomen is usually contracted, and the distance between the abdominal wall and the spine is reduced. When the same body is on the autopsy table, the abdominal wall is relaxed and this distance increases. Similarly, the anatomical relationship between the lungs, liver and other viscera is not the same as when the person is bent at the hips and when lying flat. In a stab wound on the anterior wall of the chest, the post-mortem depth is greater than it was during life, because of the collapse of the lung. If the stab is on the back of the chest, the depth of the wound will be less, as the lungs will collapse posteriorly.

The force required to inflict stab wound is subjective, and can only be stated in comparative terms, such as slight pressure, moderate force, considerable force, and violent penetration. The depth of stab depends on: (1) **Condition of the knife:** The sharpness of the extreme tip of the knife is the most important factor in skin penetration. Once the tip has perforated the skin, the cutting edge is of little importance, and the rest of the blade will pass into the body with ease, without any further force being applied. A thin, slender, double-edged knife will penetrate more deeply than an equally sharp, wide, single-edged blade inserted with the same force. A blunt-pointed instrument requires considerable force to puncture the skin and penetrate the soft tissues. **(2) The resistance offered by the tissues or organs:** Apart from bone and calcified cartilage, the skin is most resistant to knife penetration. **(3) Clothing:** The amount of clothing and its composition, e.g. multiple layers of tough cloth, leather belts, thick leather jackets, or coats, etc. require greater force. (4) The speed of thrust of the knife. (5) Stretched skin is easier to penetrate than lax skin, e.g. chest wall. (6) When the knife strikes the skin at right angle, it usually penetrates more deeply than when it strikes from some acute angle. (7) When the knife penetrates the skin



Fig. (8–20) Appearances of stab wounds on the skin by different types of weapons.

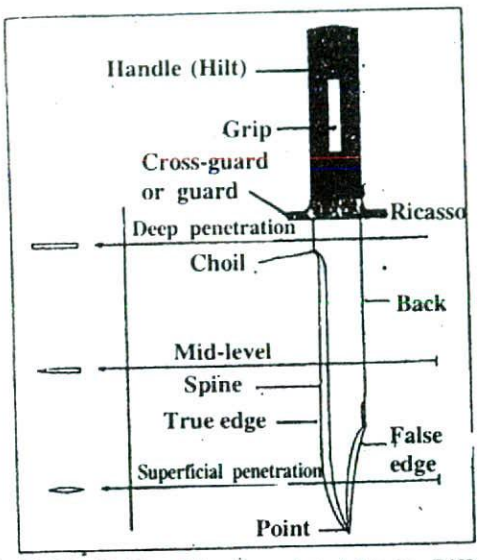


Fig.(8–21) Knife with clip-pointed blade. Differing skin wounds produced by same knife, at three different depths of penetration

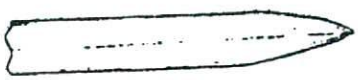


Fig.(8–22) Spear-pointed blade.

rapidly, e.g. if the body falls or runs on to the blade, the momentum of the forward moving body is sufficient to cause fatal injury. However, the knife must be sharp-pointed and held firmly so as to penetrate easily, as a blunt knife held loosely will be turned aside by the approaching body.

A piece of pliable tubing may be introduced gently, and if it goes in easily may reveal the true track. Later, the tubing can be made more rigid and straight by inserting a probe into it. Dissection in the tissues parallel to, but away from the wound, will reveal the track. Radio-opaque material or dyes can be injected into stab wound to demonstrate the wound track by X-rays.

(5) Shape : The size and shape of a stab wound in the skin is dependent on the shape of the weapon, the direction of thrust, the movement of the blade in the wound, cleavage direction, the movement of the person stabbed, and the condition of tension or relaxation of the skin (fig. 8-23). Stab wounds and incised wounds are slit-shaped with two acute angles, or gape open depending on their location and their orientation, with regard to the so-called **cleavage lines of Langer** (Fig. 8-18). The pattern of fibre arrangement of the dense feltwork of intimately intermingled dermal collagen and elastic fibres is called the cleavage direction or lines of cleavage of the skin, and their linear representation on the skin are called Langer's lines, which is almost same in all persons. Cleavage lines in the dermal layers of skin are mostly arranged in parallel rows. In the extremities they tend to run longitudinally; in the neck and trunk circumferentially. A stab wound which runs parallel to the cleavage lines will remain slit-shaped and narrow and the dimensions of the blade will be represented with considerable accuracy. A stab wound which cuts through the cleavage lines transversely will gape. If the knife is inserted in an oblique plane, the skin defect is wider and the wound may gape asymmetrically and assume a semicircular or crescentic shape. To ascertain the shape of the instrument, the edges of a wound may be manually approximated with slight twisting or they may be held together with a transparent adhesive tape. The resulting slit is considerably longer than the original oval-shaped wound. This will counter the claim of the defence that the suggested knife could not have produced a stab wound of the type as seen before reconstruction. The dimensions of the gaping wound are not useful to assess the shape of the blade.

The shape of the wound usually corresponds to the weapon used, but the shape of the wounds made

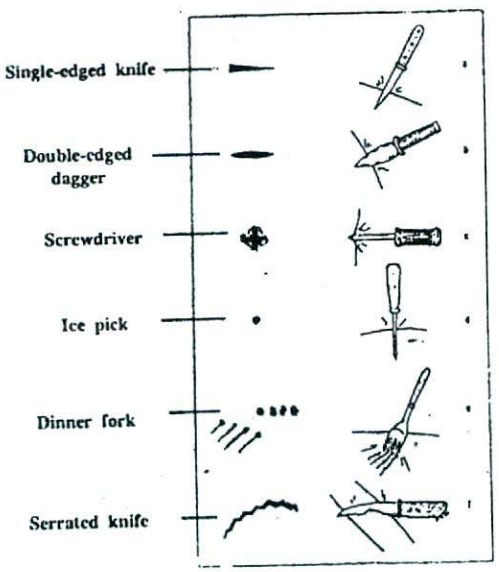


Fig.(8–23) Skin wounds produced by different weapons.



Fig.(8–24). Multiple homicidal stab injuries on the back.

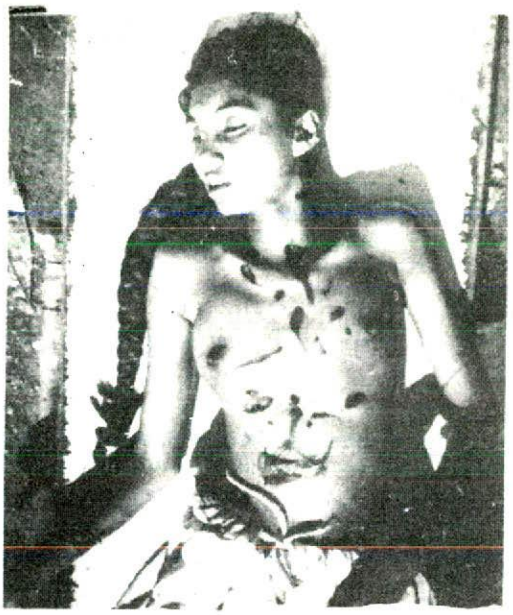


Fig.(8–25) Multiple stab injuries, some of them showing tailing.

by the same weapon may differ on different parts of the body. (1) If a single-edged weapon is used, the surface wound will be triangular or wedge-shaped, and one angle of the wound will be sharp, the other rounded, blunt or squared off (Fig. 8-19). Blunt end of the wound may have small splits (often in two places) in the skin, so-called **"fishtailing"**, if the back edge of the blade is stout. Some stab wounds caused by single-edged weapon have bilateral pointed ends like those due to double-edged weapons. This is due to: (a) The initial penetration by the knife point to a depth of about one cm., first produces a dermal defect with sharp angles at each end. As the knife penetrates more deeply, the end in contact with the cutting edge of the blade continues to be sharply angulated. The opposite end which is in contact with non-cutting surface of the knife, also remains sharply angulated because the dull surface does not imprint its shape to the skin defect. It merely causes further separation of the skin which continues to be torn along the course of its original direction. (b) The knife penetrating the skin at an oblique angle. As the knife perforates, it is pulled down with the cutting edge cutting through the skin and the blunt surface of the back of the knife does not impart its shape to the skin defect, as it does not contact it. (c) Many single-edged knives have a cutting edge on both sides at the tip. The initial thrust into the skin will produce a double-pointed wound, and as the blade passes through the skin, if it is pulled down slightly, the blunt back portion of the knife will not come in contact with the skin. The serosal planes and muscle fasciae (pleural surface, liver capsule and the pericardial sac) often clearly show the wedge-like shape of a knife. In some single-edged knives, both ends are blunt. This is caused if the knife penetrates to full length up to the guard, because of ricasso (short, unsharpened section of blade between the cutting edge and guard). By examination of a single wound, it is not possible to say, whether it was caused by a single-edged or double-edged weapon. If multiple stabs are produced by a single- edged weapon, examination of all the stab wounds will reveal the single-edged nature of the weapon. (2) 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. The blunter the tip of the object, the coarser or more stellate will be the hole it makes. (5) A pointed square weapon may produce a cross-shaped injury, each of the 4 edges tearing

its way through the tissues. (6) A fall on a pointed article, e.g., pieces of broken glass, will produce a wound with irregular and bruised margins, and fragments of glass may be found embedded in the soft tissues. (7) Stab wounds inflicted with a broken bottle, appear as clusters of wounds of different sizes, shapes, and depth, with irregular margins, and varying depth. (8) Stabbing with a fork produces clusters of 2 or 3 wounds depending upon the number of prongs on the fork. (9) A screwdriver will produce a slit-like stab wound with squared ends (rectangular) and abraded margins. (10) Thick relatively blunt-edged blades, e.g., bayonets, may produce cross-shaped stab wounds, because cutting and tearing of the skin occurs simultaneously and at right angles to each other. When a knife is twisted as it is withdrawn from the tissues, the external wound may have a cruciate appearance. (11) Ice-picks or similar instruments produce stab wounds, resembling small caliber bullet wounds. (12) Irregularly-shaped stab wounds such as L or V-shaped may be mistaken to be produced by two distinct stabbings in the same location. These atypical injuries are produced by stabbing, followed by simultaneous twisting and cutting (rocking), or the victim moving relative to the knife, or by a combination of the two. There will be a primary stab wound with an extension of it, due to the knife edge cutting the skin in a different direction as it exits. Small notches in margins of the skin defects or curving can be produced by the same mechanism. A great number of possibilities capable of causing atypically shaped stab wounds exist, the interpretation of which are usually difficult. (m) If the scissors is closed, the tip of the scissors splits rather than cuts the skin, producing a linear stab wound with abraded margins. Deep penetration will produce 'Z'-shaped wound. If the screw holding the two blades is projecting, there are small lateral splits in the wound centre. If the two blades of the scissors are separated, each thrust will produce two triangular stab wounds. (n) A knife with a serrated back edge will produce a stab wound, the back edge of which may be torn or ragged. If the knife enters obliquely, serrated abrasions may be seen on the skin adjacent to the end of the wound.

(6) Direction : When the knife penetrates at an angle, the wound will have a bevelled margin on one side with undermining (undercut) on the other, so that subcutaneous tissue is visible, indicating the direction from which the knife entered. In solid organs like the liver, the track made by the weapon is better seen. The principal direction should be noted first and other next, e.g., backwards and to the right. If the weapon is partially withdrawn and thrust again in a new direction, two or more punctures are seen in the soft parts with only one external wound. If the wound is perforating, it should be described in sequential order: the wound of the entrance, the path of the wound track, and the wound of exit. If the wound is penetrating, the wound of entrance should be described first, then the depth and direction of wound track, and specific termination.

An example of description of stab wound: Stab wound, wedge-shaped on the right upper chest, placed obliquely, with the inner end lower than the upper outer end. Length was 4 cm. The maximum width at the centre was 5 mm. The centre of the wound was just below the line joining the nipples, being 5 cm. from the midline, 6 cm. from the right nipple, and 18 cm. below right collar bone. The inner end was sharp and outer blunt. The lower margin was undermined. A track is established on the right anterior thoracic wall, passes through the fifth interspace, through upper lobe of right lung to a depth of 7 cm. A right haemothorax of 400 ml. of fluid and clotted blood is present.

Complications : (1) External haemorrhage is slight but there may be marked internal haemorrhage or injuries to internal organs. (2) The wound may get infected due to the foreign material carried into the wound. (3) Air embolism may occur in a stab wound on the neck which penetrates jugular veins. Air is sucked into the vessels due to the negative pressure. (4) Pneumothorax. (5) Asphyxia due to inhalation of blood.

Concealed Puncture Wounds : These are puncture wounds caused on concealed parts of the body, such as nostrils, fontanella, fornix of the upper eyelids, axilla, vagina, rectum, and nape of the neck. Fatal penetrating injuries can be caused without leaving any readily visible external marks, e.g., thrusting a needle or pin into the brain through the fontanelles, through the inner canthus of the eye, or into the medulla through the nape of the neck. These injuries may not be detected unless searched carefully.

Examination : The following points should be noted : (1) Identification and labelling of cuts and damage to clothing. (2) Distribution of blood stains.

Table (8–5) Differences between suicidal homicidal and accidental stab wounds.

Trait	Suicidal wounds	Homicidal wounds	Accidental wound
(1) Number:	Often single.	Frequently multiple.	Usually single.
(2) Site:	Accessible precordial area or upper abdomen.	May be anywhere.	May be anywhere.
(3) Tentative wounds:	May be present around site of fatal wound.	May be present rarely but away from fatal wound.	Absent.
(4) Clothing:	Removed from injured area.	Normally not disturbed.	Not disturbed.
(5) Defence wounds:	Absent.	Often present.	Absent.

Table (8-6). Difference between incised, lacerated and stabd wounds.

Trait	Incised wound	Lacerated wound	Stab wound
(1) Manner of production :	By sharp objects or weapons.	By blunt objects or weapons.	By pointed sharp or blunt weapons.
(2) Site :	Anywhere.	Usually over bony prominences.	Anywhere; usually chest and abdomen.
(3) Margins :	Smooth, even, clean-cut and everted.	Irregular and often undermined.	Clean-cut, parallel edges. Lacerated if weapon is blunt-pointed.
(4) Abrasion on edges :	Absent.	Usually present.	Absent.
(5) Bruising :	No adjacent bruising of soft tissues.	Bruising of surrounding and underlying tissues.	Rare.
(6) Shape :	Linear or spindle-shaped.	Varies; usually irregular.	Linear or irregular.
(7) Dimensions :	Usually longer than deep; often gaping.	Usually longer than deep.	Depth greater than length and breadth.
(8) Depth of wound :	Structures cleanly cut to the depth of wound.	Small strands of tissue at the bottom bridge across margins.	Structures cleanly cut.
(9) Haemorrhage :	Usually profuse and external. Spurting of blood may be seen.	Slight except scalp and external.	Varies; usually internal.
(10) Hair bulbs :	Cleanly cut.	Crushed or torn.	Usually clean cut.
(11) Bones :	May be cut.	May be fractured.	May be punctured.
(12) Foreign bodies :	Absent.	Usually present.	Usually absent.

(3) Removal of clothing, layer by layer. (4) Identification and labelling of wounds. (5) Wounds: (a) Position (height from heels), (b) location (measurements from fixed anatomical landmarks), (c) description including margins, size, shape, ends extension, (d) direction, (e) depth, (f) trauma to viscera, (g) estimation of force required, (h) foreign bodies. To indicate the general character of the instrument which inflicted the stab wound, the term "incised' or 'lacerated' should be included in the description of the stab wound, e.g., 'incised-stab wound', 'lacerated-stab wound', etc. The relationship between the wound and weapon can be established by the shape, width, length, and presence of blood on the weapon.

When multiple stab wounds are present, they should be numbered and photographed. A sketch or a printed body diagram should be used.

Incised-stab wound: Incised-stab wound is a wound which starts as an incised wound and ends as a stab wound by the sudden thrust of the blade into the body, or starts as a stab wound and becomes incised wound as the knife is pulled out of the body at a shallow angle to the skin surface producing an incised wound (fig. 8-25). If a nick or a pork-shaped cut is present at the end of the stab wound opposite to the incised portion, then the wound has started as an incised wound and ended as a stab wound. If the fork is at the end of the stab wound where the incised wound arises, then the wound has started

Table (8–7) Differences between suicidal, homicidal and accidental wounds.

Trait	Suicide	Homicide	Accident
(1) Nature of wounds :	Usually incised and stab.	Usually chop wounds, lacerations and stab.	Usually lacerations, abrasions and contusions.
(2) Number of wounds :	Multiple.	Multiple.	Usually single, may be multiple.
(3) Target area :	Accessible parts only, i.e., front and sides of the body, such as neck, wrists, left side of chest, groin, etc.	No fixed site; vital parts, such as head, chest, abdomen.	Anywhere; usually on exposed parts and bony prominences. Number of wounds on the same side.
(4) Wound grouping :	Arranged.	Irregular.	Vulnerable parts.
(5) Direction :	In right-handed persons from left to right and from above downwards.	Any direction.	Any direction.
(6) Severity :	Mostly superficial; one or two deep wounds.	Mostly severe and extensive.	Variable severity.
(7) Hesitation marks:	Usually present.	Absent.	Absent.
(8) Defence wounds :	Absent.	May be present.	Absent.
(9) Secondary injuries :	Absent.	May be connected with fight.	May be associated with accident.
(10) Weapon :	By the side of the body or may be grasped firmly due to cadaveric spasm.	Absent.	Present.
(11) Clothes :	Not damaged as they are usually removed.	May be damaged.	May be damaged and stained with oil, grease, mud, dirt, etc.
(12) Scene of crime :	Usually inside closed room; no disturbances of surroundings.	Disturbed and disorderly with signs of struggle and blood stains.	Varies with the nature of the accident.
(13) Motive :	Present, such as domestic worries, disappointment in love, chronic disease, failure in examination, etc.	Revenge, robbery, sexual offences.	Absent.

as a stab wound (Fig. 8-24).

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.

Examination of the Weapon : The doctor should note : (1) the length, width and thickness of the blade, (2) whether single-edged or double-edged, (3) the degree of taper from tip to hilt, (4) the nature of the back edge in a single-edged weapon, e.g. squared-off, serrated, etc., (5) the face of the hilt guard adjacent to the blade, (6) any grooving, serration or forking of the blade, and (7) sharpness of the extreme tip of the blade and the cutting edge.

In some cases of stabbing, the blade of the knife may not be bloodstained. In solid organs, bleeding occurs only after the knife is withdrawn, because bleeding is prevented by the pressure of the knife. During the withdrawal of the knife, the muscular and elastic tissue of the solid organs, and the elastic tissue of the skin may contract about the knife, wiping off the blood present on the knife blade. In such cases, analysis of wiping of the blade might still yield sufficient tissue to perform at least limited DNA analysis and typing. The clothing if present, may also wipe off the blood. An injury may cause deep shock and severe circulatory collapse. Subsequently, even if a deeply penetrating wound

is produced, there may be little bleeding.

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.

CIRCUMSTANCES OF INJURIES :

SUICIDE : Suicidal stab wounds are found over accessible areas of the body. The common site is the chest over the heart region. The direction of the wound is upwards, backwards and to the right. The depth is variable, some are superficial and others enter the pericardium or heart. The suicide may not withdraw the point of the weapon from the skin, and stab himself repeatedly in different directions through the same skin wound. Rarely one stroke is fatal, and the knife may be found sticking in the wound. In many cases, even multiple stabs do not cause death and the person may resort to other methods which present a puzzling picture. Stab wounds of the head are rare. Usually, a number of stabs extending only to the bone are seen in the temporal region. Suicidal stab wounds of the spine, abdomen, neck and extremities are rare. Wounds found on a part of the body usually covered by clothes without corresponding cuts or rents on them are suicidal, as a person who commits suicide exposes his body by opening his clothes and then inflicts wounds. In some suicides, the victim may be able to conceal the weapon.

Common methods of suicide are: poisoning, hanging, burning, drowning, jumping from a height, stabbing and cutting and railway injuries.

HARA-KIRI: It is an unusual type of suicide, in which the victim inflicts a single large wound on the abdomen with a short sword while in a sitting position or falls forward upon a ceremonial sword and pulls out intestines. The sudden evisceration of the internal organs causes a sudden decrease of intra-abdominal pressure and cardiac return, producing sudden cardiac collapse.

HOMICIDE: Most deaths from stab wounds are homicidal, especially if found in an inaccessible area. The wounds are multiple, widely scattered and deeply penetrating, involving the chest and abdomen. In case of a sudden surprise attack, a single wound is found at a vital spot. If there is a struggle, there may be a number of wounds, sometimes associated with defence cuts on the hands. In a few cases, a number of wounds may be found in a localised area, almost of the same depth. These occur when the assailant threatens the victim, who is held by another assailant. These wounds are usually seen on the face or neck, and may be far away from the fatal injury. Homicidal stab wounds of the chest may have any direction, but the common direction is at an angle from left to right and from above downwards. Fatal stab wounds of right chest usually injure right ventricle, aorta or right atrium. Stab wounds of the left chest usually involve the right ventricle when parasternal, and left ventricle as the stab wounds become more lateral and inferior. Severing of the left anterior descending coronary artery is rapidly fatal. Most stab wounds of the heart and lungs occur over the front of the chest, rarely the sides, and least on the back. Stab wounds of the lower chest can injure the heart and lungs and also abdominal viscera. Rarely a single stab may produce multiple skin wounds, such as tangential stab of the arm may pass through superficial tissues and then enter the chest wall, or stab through a sagging female breast can pass through the edge and re-enter the thorax. In OVERKILL HOMICIDE, assailant continues stabbing beyond the victim's death (Fig. 8-24&25) Postmortem stab wounds do not show bruising and are often yellow, or tan, due to absence of tissue perfusion, parchmented and sharply defined.

ACCIDENT : Accidental wounds are rare. They are caused by falling against projecting sharp objects like glass, nails, etc., or a person may be gored by the horns of a bull, buffalo, etc. An upward or downward track is quite inconsistent with an accidental stab wound. Impaling injuries are very rare and caused when a person falls or jumps from a building and lands on a fence or ornamental railings at play or mechanical catastrophe at work. Accidental stab wounds occur if a person is impaled after an accidental fall on to a sharp-pointed object, or by a moving sharp object striking the person.

ACCIDENTAL, SELF-INFLICTED OR INFLICTED BY OTHERS: The following factors are helpful to determine whether the wounds are suicidal, accidental or homicidal. (1) The nature, direction, extent and situation of the wounds. (2) The presence of foreign matter in the wound or adherent to the margins. (3) The nature of the suspected weapon or instrument. (4) Scene of the crime.

DEFENCE WOUNDS : Defence wounds result due to the immediate and instinctive reaction of the

victim to save himself, either by raising the arm to prevent the attack, or by grasping the weapon. If the weapon is blunt, bruises and abrasions are produced on the extensor or ulnar surfaces of the forearms, wrists, backs of the hands and knuckles. Defence wounds may be found rarely on the shins and feet if the victim was lying on the ground usually face up, as he kicks at the assailant, or curls up and tries to cover vital areas with his legs. The size and shape of the bruises depend upon the attacking object. Fractures of the carpal bones, metacarpals and digits may occur. There may also be defence bruises and abrasions on the thighs If the weapon is sharp, the injuries will depend upon the type of attack, whether stabbing or cutting. In stabbing with a single-edged weapon, if the weapon is grasped, a single cut is produced on the palm of the hand or on the bends of the fingers or thumb. If the weapon is double-edged, cuts are produced both on the palm and fingers. The cuts are usually irregular and ragged, because the skin tension is loosened by gripping of the knife. When attacked, the victim usually holds up the hand or forearm and receives cuts on the hand, wrist, ulnar border of the forearm and on the fingers. They are often irregular in depth and distribution. A typical knife defence wound may be seen in the web between the base of the thumb and index finger, when the blade is grasped. Defence wounds indicate homicide. In the female, they suggest sexual assault. Defence wounds are absent if the victim is unconscious, or is taken by surprise, or attacked from the back, or under the influence of alcohol or drugs.

Offensive Manual Injuries: Abrasions and contusions over the knuckles can be sustained due to offensive efforts by the victim, or by his defensive efforts. Ragged knuckle lacerations on the victim indicate that his fist struck the assailant's anterior teeth when he struck a blow on the latter's open mouth. Fractures of the fourth and fifth metacarpals (knuckle fractures) may occur when the assailant is punched on his head or chin. In manual assault, the hands of the suspected assailant may show injuries indicative of or compatible with his involvement in assault.

SELF-INFLICTED AND FABRICATED WOUNDS : Self-inflicted wounds are those inflicted by a person on his own body. Fabricated wounds (fictitious, forged or invented wounds) are those which may be produced by a person on his own body, or by another with his consent. They may be produced for the following reasons. (1) To charge an enemy with assault or attempted murder. (2) To make a simple injury appear serious. (3) By the assailant to pretend self-defence or to change the appearance of wounds, which might connect him with the crime. (4) By policemen and watchmen acting in collusion with robbers to show that they were defending the property. (5) In thefts by servants or messengers for the above reason. (6) By prisoners, to bring a charge of beating against officers. (7) By recruits to escape military service. (8) By women to bring a charge of rape against an enemy.

Fabricated wounds are mostly incised wounds, and sometimes contusions, stab wounds and burns. Lacerated wounds are rarely fabricated. Incised wounds are usually superficial, multiple and parallel, are of equal depth at origin and termination. They avoid vital and sensetive areas like the eyes, lips, nose and ears. The direction is from behind forwards on the top of the head, from above downwards on the outer side of upper arm, from below upwards on the front of forearms, variable on the legs and vertical on the abdomen and chest. Some mentally disordered persons may inflict hundreds of small wounds upon themselves. Paranoid schizophrenics with a strong religious flavour to their delusions may remove penis, scrotum and testes. Stab wounds are usually multiple and superficial and seen about the left arm or shoulder and sometimes on the chest. Burns are superficial and seen usually on the left upper arm. The clothes are not cut and if cuts are seen they are not compatible with the nature of the wounds. The history of the assault is incompatible with the injuries.

Old linear scars in sites of election suggest previous similar attempts. They are seen on the face, on the front of the trunk, on the arms and on the fronts of the legs.

CASE: A youth alleged that he had been the subject of a murderous attack and had received a number of letters over a long period threatening him with death. He had been placed under police protection. His story was that he had been followed by a man, and when he walked into a stairway he was brutally attacked with a razor. He shouted for help, and his assailant, whom he described in detail, ran away. He was in a weak and frightened state when he reported the matter to the police, and was bleeding from wounds in his back. On medical exam the wounds consisted

of a number of fine cuts just penetrating the skin between his shoulder-blades, more or less parallel to one another and apparently made from below upward by a right-handed man. They were undoubtedly self-inflicted, by means of a sharp instrument such as a razor used very carefully. On repeated questioning he admitted he had made the cuts by fixing a razor-blade on the end of a cleft stick and doing the job by adjusting two mirrors. The various threatening letters were in his own handwriting. Later, he duly confessed to the police.

Therapeutic Wounds : These are wounds produced by doctors during the treatment of a patient, such as surgical stab wounds of the chest for insertion of chest tubes, of the abdomen for drains, thoracotomy and laparotomy incisions, incisions on the wrists, antecubital fossae and ankles, and tracheostomy incisions. Some of these wounds may be mistaken for traumatic wounds, e.g. a surgical stab wound of the chest for putting a drainage tube. Sometimes, a traumatic wound may be enlarged and included in the surgical procedure, or a drainage tube may be put in a homicidal stab wound.

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. **Proximal (internal) ballistics** is the study of firearms and projectiles, **Intermediate (exterior) ballistics** is the study of the motion of projectile after it leaves the gun barrel till the time it hits the target.

Terminal ballistics involves the study of behavaiour of missiles once they penetrate their targets. **Wound ballistics** is the study of the effects of missiles on living tissue.

GENERAL MAKE-UP AND MECHANISM : Firearms consist of a metal barrel in the form of hollow cylinder of varying length, which is closed at the back end and is called the breech end; and the front open end is called the muzzle end. The inside of the barrel consists of three parts. (1) The chamber, at the breech end to accommodate the cartridge which is usually of large size than the bore, (2) the taper, called lead or leed in rifled arm and chamber cone in a smooth bore, connects the chamber to the bore, and (3) the bore, which lies between the taper and the muzzle. A breech action is attached to the barrel at the breech end to close the end of the barrel. It consists of a receiver which contains a bolt or block which closes and locks. The bolt or the block (consists of firing pin, spring, trigger and extractor) can be opened to insert a fresh cartridge into the chamber. Spent cartridges are removed by extractor claw. When the weapon is closed for firing, the barrel comes into position against a flat block of metal called breech face, which seals the breech end of the chamber. The block is pierced in its centre to accommodate the firing pin which is actuated by a spring and moves forward when trigger is pressed. When the weapon is cocked (ready to be fired), the hammer has been pulled back against a strong spring and is held back by the end of the sear, which rests in a notch known as the bent. When the trigger is pulled, the sear is disengaged from the bent and the hammer moves forward and strikes a small pushrod (firing pin or striker). In hammerless guns, the striker is used to fire the gun and the cocking is done by pulling back of the bolt action Fig. (8-26). The barrel of a "breakdown" action gun or rifle, i.e., a weapon which opens like a shotgun, has at its breech end a moveable limb called extractor, which moves backwards withdrawing the cartridge, when the breech is opened (Fig. 8-27). In a bolt action weapon, a small claw fitted to the front end of the bolt, pulls out the cartridge with it, when the bolt is withdrawn. The lock contains the lock and trigger mechanism, i.e. the apparatus for discharge of the weapon. The slide is the top part of semiautomatic that contains the barrel and on which the sights are mounted. A firearm is provided with sights with which one can aim, so that the bullet can strike the target accurately. The 'stock' is the supporting or handle part of the weapon. The

Table (8–8) Shotgun gauges with decimal equivalents.

Shotgun (gauge)	Bore diameter Inch	mm.
10	0.775	19.69
12	0.730	18.52
16	0.670	16.81
20	0.615	15.62
28	0.550	13.97
410	0.410	10.41

Table (8–9) Various degrees of choke.

Designation	Constriction in mm	inch
(1) Full choke	1.00	0.04
(2) Three-quarter choke	0.75	0.03
(3) Half-choke	0.50	0.02
(4) Quarter-choke	0.25	0.01
(5) True cylinder	0	0

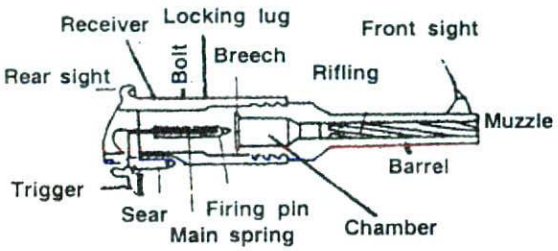


Fig. (8–26) Nomenclature of small arm.

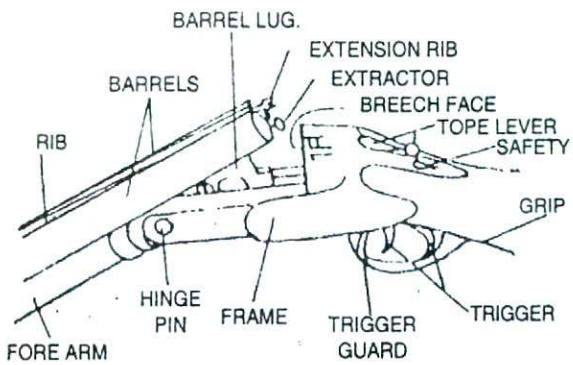


Fig. (8–27) Diagram showing details of twin-barrel (smooth-bore) shotgun.

slide is the top part of semiautomatic that contains the barrel and on which the sights are mounted. Terminal ballistics involves the study of behaviour of missiles once they penetrate their targets. Wound ballistics relates to the missiles effect on living tissue. the end of which is the 'butt'. The hand-arms are provided with grips for grasping them by hand. In long barrelled weapons, the butt is elongated to fit into shoulder. Those with long barrels are fired from the shoulder, i.e., rifle and shotgun, and those with short barrels may be fired from the hand, i.e., pistol and revolver. Firearms except revolvers and hammer guns have a safety device, which locks the firing mechanism when it is applied, known as safety catch.

Classification : (I) Rifled weapons: (1) Rifles: (a) Air and gas-operated rifles. (b) 0.22 rifles. (c) Military and sporting rifles. (2) Single-shot target-practice pistols. (3) Revolvers. (4) Automatic pistols. (5) True automatic weapons (machine guns). **(II) Smooth-bored weapons** (shotgun). Cylinder bore. (2) Choke bore. (3) Breech loader. (4) Muzzle loader. (5) Country made. **According to firing action:** (1) Over-bolt action (2) Under-bolt action. (3) Lever action. (4) Pump action or autoloading model.

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. 8-30). Rifling is made by a broach. 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. A handgun bullet usually makes a single turn about its longitudinal axis in 25 to 45 cm. of forward motion. Micro-groove system of rifling consists of 15 to 20 round grooves in bore of the barrel. 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).

Calibre or gauge: It is measured by the internal dimension of the barrel and is given in decimals of inch or millimetres. The dimension of the rifled weapon is measured between a pair of diametrically opposed lands and not grooves (Fig. 8-30). In smooth-bored weapons, the bore is measured similarly up to 1.27 cm. (half inch). For larger bores, the size is determined by the size of the lead ball which will exactly fit the barrel, and by the number of such balls of equal size and weight as can be made from 454 gm. (one pound) of pure lead. Thus the 12 bore gun is one whose diameter is that of a ball of lead of such a size that 12 balls may be made from 454 gm. The smaller the number of gauge, the greater the diameter of the barrel (Table 8-8).

Helixometer is the instrument to examine the interior of the barrel.

Shotgun : It may be single-barrelled or double-barrelled, the barrels lying side by side, or occasionally mounted one over the other. It is intended for firing a single ball, slug or a charge of shots. The barrel varies in length from 55 to 72 cm. Shotgun bores vary from 4 to 20. The common gauges are 12, 16 and 20. The weapon is made to 'break' or open on hinge across the breech facing for the insertion and extraction of cartridge cases. **Muzzle loading** guns are loaded entirely from muzzle end with the help of a rod using gunpowder, pieces of cloth, stones, metal fragments,

seeds, bolts, wood, screws, etc. The interior of the barrels is smooth (fig. 8-27). When the entire barrel from breech to muzzle is of the same diameter, it is called cylinder-bore. In **choke-bore** (fig. 8-28) the distal seven to ten cm. of the barrel is narrow. Different degrees are known as full-choke, half-choke and quarter-choke or improved cylinder (Table 8-9). Shotguns may have variable choke adapters. The choking lessens the rate of spread of shot after it leaves the muzzle, increases the explosive force and increases the velocity. There are some shotguns which have small portion of their bore near the muzzle end rifled, which are called **"paradox guns"**. Some shotguns have the whole of their bore rifled with very shallow grooves. **A musket** is a military shoulder arm. It has a long forestock and usually takes a bayonet (pointed knife-like weapon) at the muzzle. It is smooth-bore weapon, e.g., 0.410 musket. 0.410 musket fires a single shot at high velocity and is effective up to 90 metres. Many shotguns have removable and interchangeable barrels. The muzzle velocity of shotgun is about 240 to 300 m/s and of 0.410 musket from 350 to 600 m/s. Shotguns are effective up to 30 to 35 metres.



Fig. (8–28) Longitudinal section of a rifled barrel.

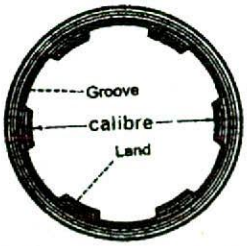


Fig. (8–29) Cross-section of rifled barrel.

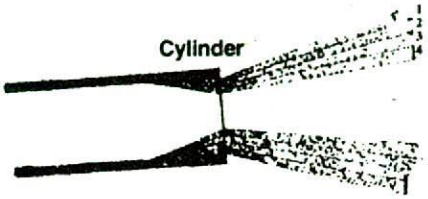


Fig. (8–30) Choking of shotgun.
(1) Improved cylinder (2) Quarter choke (3) Half choke
(4) Three fourth choke (5) Full choke

Cartridge : The shotgun cartridge (ig. 8-32) consists of a case of short metal cylinder which is continuous with a cardboard or plastic cylinder. The case is rimmed, which helps to keep the cartridge in correct position in the chamber, and makes extraction easy. The case helps to keep the various components in place, prevents the backward escape of the gases and provides a waterproof container for the gunpowder. The length of the cartridge varies from 5 to 7 cm. The cartridge case is filled as follows from the base: percussion cap (detonator cap; primer battery cup), which is set into the centre of the base of the cartridge cylinder, gun powder, a thick felt-wad with cardboard discs lying in front and behind it, the shot and finally the retaining cardboard disc, over which the edges of the cartridge

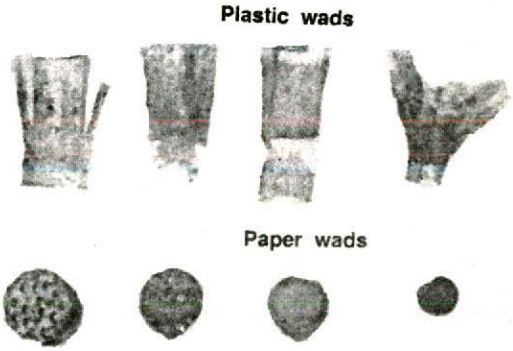


Fig. (8–31) Fired shot shell wads.

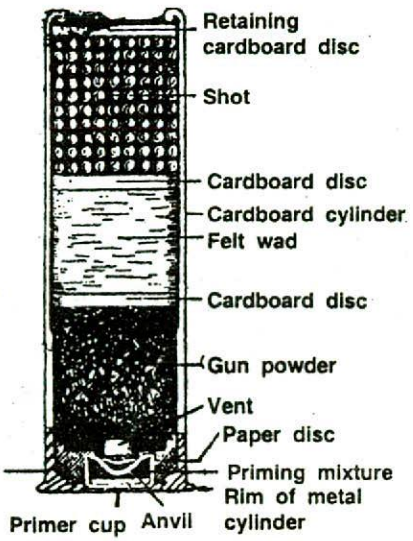


Fig. (8–32) Shotgun cartridge.

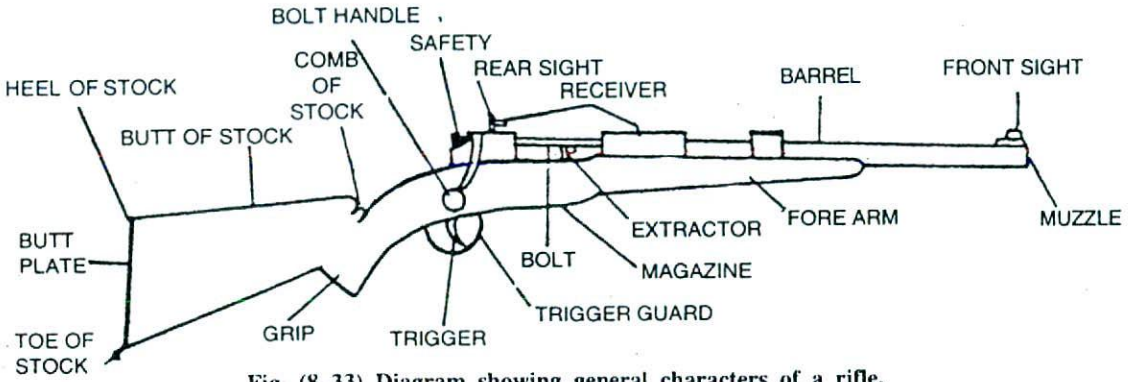


Fig. (8–33) Diagram showing general characters of a rifle.

cylinder walls are pressed (Fig. 8-31). Some cartridges have top wad over the lead shot to keep the shot in place. The diameter of the wadding used in the cartridge is greater than that of the bore of the gun. Wad acts as a piston and seals the bore completely, thus preventing the expanding gases from escaping and disturbing the shot charge. The felt wad contains grease, which lubricates the bore after the firing of each round. The modern one piece plastic wadding tends to have greater range due to its greater weight (Fig. 8-31) The powder is protected from the grease wad by a thin grease-proof card wad. Wads may be glazed-board, straw-board, plastic, cork, felt, etc. and may be disc-shaped, cup-shaped, or bizarre-shaped. Shots (pellets) are of two types. (1) Soft or drop shot is made of soft lead, (2) Hard or chilled shot is made from lead and hardened by antimony. The shots may also be plated with copper. Cartridges are numbered according to the size of the shot they contain. The shot consists of up to several hundred small lead shots, the number depending upon the size of the individual pellets. **"Shot size"** is expressed in number. The smaller the number, the larger the shot. **Dust-size shot** gun pellets number up to 2600 minute shot in 12 bore cartridge. **Bird shot** are small, ranging in diameter from one to 3.5 mm. A 12 bore shotgun shell contains 200 to 400 shot. **"Buckshot"** is the largest and has a diameter of 6 to 8 mm. **LG** is 9.14 mm. In a 12-bore shell they are nine in number. Pellets deform easily due to friction as they rub against the inside of the barrel. The heat can cause melting and fusion of pellets. **Rifled slugs** are single projectile, and are used in shotguns for big game hunting. They are similar in shape to a blunt bullet with a deep hollow cavity in the base and weigh from 23 to 33 g. The sides commonly have angularly inclined fins or ribs that resemble very coarse rifling marks on bullet. The slugs are usually a little smaller in diameter than the shotgun bore itself. The slugs have much greater range than pellets. The spiral grooves on the slugs impart spinning effect. The card-wad behind the shot charge prevents the shots from getting lodged in the felt-wad. (fig. 8-40) Some cartridges contain **'power**

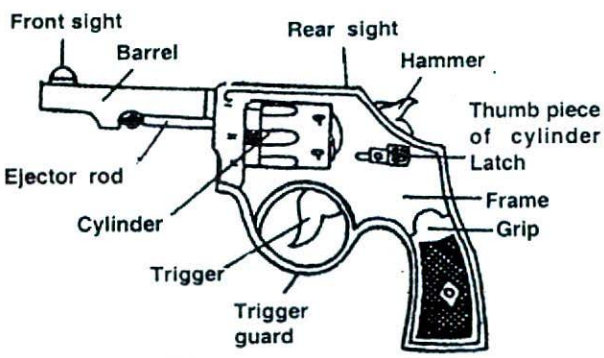


Fig. (8–34) Diagram showing general characters of a revolver.

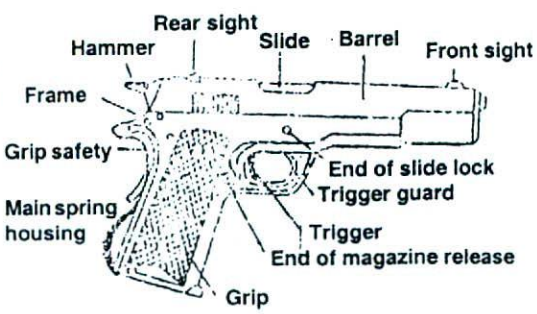


Fig. (8–35) Diagram showing general characters of a pistol.

piston' formed from four leaflets or petals folded so as to produce a cylindrical shape, which holds the shot inside a polythene cup, which may contribute to the wound at short range (Fig. 8-40). Some cartridges may have brightly coloured plastic granules as a filler between the shot, which may be found inside the wound.

Improvised firearms or country-made firearms are common in India. They are of various calibres, but 12 bore is the commonest. They are usually smooth-bored. Some of them can be mistaken for factory-made guns, but others do not appear to be guns at all. Walking-stick guns and folding guns are single-barrelled shotguns.

Rifle : A rifle (Fig. 8-33) is a gun with a long barrel, the bore of which is rifled. **A carbine** is a short-barrelled rifle, or a musket. Its barrel is less than 55 cm. in length and is meant for persons on horse-back. It may be self-loading or automatic or both. It is effective up to 300 metres. The bore of a rifle varies from 5.6 to 7.7 mm (0.22 to 0.303 inch). It has a firing range up to 1,000 metres. The military rifle has a magazine and bolt action, muzzle velocity of 800 metres per second and can kill at a range of 3,000 metres. The longer barrel gives an increased speed to the projectile. Velocity is the speed of the bullet or projectile at a predetermined point in its flight. The pressure in the firing chamber is about 20 tonnes per square inch. The bullet as it leaves the barrel, rotates at about 3,000 revolutions per second. The muzzle velocity is 450 to 1500 metres per second. Rifles may be single-shot, repeating, semi-automatic and automatic.

Revolver : Revolvers are so-called because the cartridges are put in chambers in a metal cylinder, which revolves or rotates before each shot, to bring the next cartridge opposite the barrel, ready to be fired. It has a cylindrical magazine situated at the back of the barrel, which has a revolving motion, and can accommodate 5 to 6 cartridges, each housed in a separate chamber (fig. 8–34). In the single action type, a bolt had to be moved manually to eject the spent cartridge and to bring a new round into the breech from a magazine cylinder and brings the cartridge in the proper position for firing. In the double action type, the hammer can be cocked by hand, or by a prolonged pull on the trigger. The bores vary from 5.6 to 11.25 mm (0.22 to 0.45 inch). The muzzle velocity is 150 to 180 metres per second. They are low velocity weapons. The effective range is 100 metres.

Automatic Pistol: It is a hand arm in which the cartridge is loaded directly into the chamber of the barrel (fig.8–35). When a cartridge is fired, the empty cartridge case falls on the ground several metres away, and a new cartridge slips into the breech automatically by a spring. The cartridges are contained in a vertical magazine in the butt, which can accommodate 6 to 10 cartridges. They are really semi-automatic or self-loading, because the trigger has to be pressed each time a round is fired. The bores vary from 6.35 to 11.25 mm. (0.25 to 0.45 inch). The muzzle velocity is 300 to 360 metres per second or more. They are high-velocity weapons. Their effective range is about 100 metres.

Air Rifle and Air Pistol : In these, compressed air is used to fire lead slugs. Some weapons use cartridges of liquid CO_2 as the propellant. The velocity is very little (80 to 105 m/s), and usually minor injuries are caused. Their range is about 40 metres. The missile is single and fired through rifled barrel. The bores vary from 4.4 to 5.6 mm. Death can occur from injury to the head, heart and abdomen. The slug can enter the skull and pass through the whole mass of the brain, but does not produce an exit wound.

ROCKETS are bombs that are propelled to their target by self-contained reaction motors. MINES are hidden explosive ordinance. GRENADES are small fragmentation munitions either hand-thrown, fired from rifles or ejected from special grenade launchers.

HUMANE (VETERINARY) KILLERS: They are used in abattoirs and by veterinary surgeons to kill large animals. Captive-bolt type weapon inflicts a clean, penetrating injury, about 5 cm. deep, which can be mistaken for a stab injury. Another type discharges 8 mm bullet, the injuries resemble a contact wound produced by a conventional weapon.

PEN GUNS : Tear gas pen guns may be modified so as to fire ordinary cartridge.

ZIP GUN: It is a crude home-made single shot rifled firearm.

STUD GUNS: They are tools used to fire metal studs into wood, concrete and steel.

AUTOMATIC WEAPONS: Machine-guns or sub-machine guns continue to fire through the action of gases of the previous explosion and of the ejector and recoil spring. Submachine guns have a firing rate of 600 to 800 shots/minute.

Cartridge: It consists of a metal cylinder with a flat base which projects as a rim except in an automatic pistol (Fig. 8-36). Rimless cartridge has an extractor groove near the base. 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. Usually there is no wad, but sometimes one piece wad is kept. Low-power rim-fire cartridge may not contain gunpowder but only a primer compound in a hollow rim (Fig. 8-37). As such, those cartridges cannot produce the tattooing. Many bullets have near the base, a circumferential groove called **"cannelure"**, into which the end of the case is crimped. A bullet without cannelure is held in position by stabs on the circumference of the case.

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 (fire holes or vents) in the centrefire case head, and ignites the powder charge. The primer used in shotgun cartridge is called a primer battery cup. This consists of a battery cup into which a primer cup fits. The battery cup supports the anvil and provides a flash hole. It is also held in place by friction. The priming mixture contains lead peroxide, lead styphnate, tetrazene, barium nitrate, antimony sulphide, pentaerythritol tetranitrate, etc. These minute particles can travel up to 7.5 to 15 cm. In rimfire cartridges no percussion cap is provided. The priming mixture is contained within the hollow rim of the cartridge and ignited when it is crushed between the rim walls of the cartridge head by the impact of the firing pin.

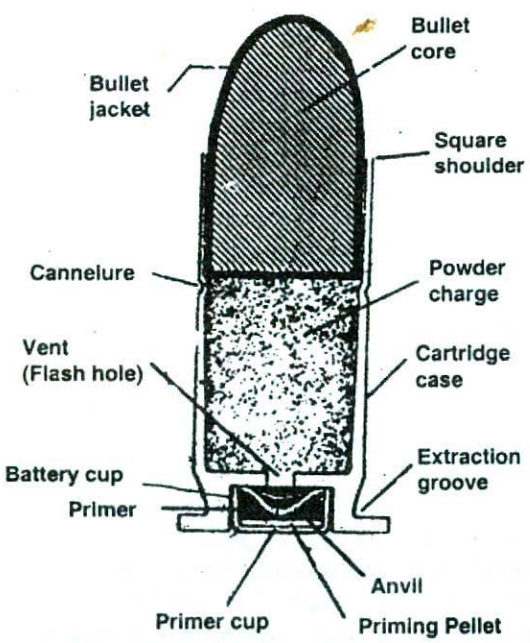


Fig. (8–36) Cartridge for rifled weapons.

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 more number of F's, the finer are the grains and the faster in burning. 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, CO_2, 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). Triple base powder consists of nitrocellulose, nitroglycerine and nitroguanidine. They produce much less flame and smoke and are more completely burnt than black powder.

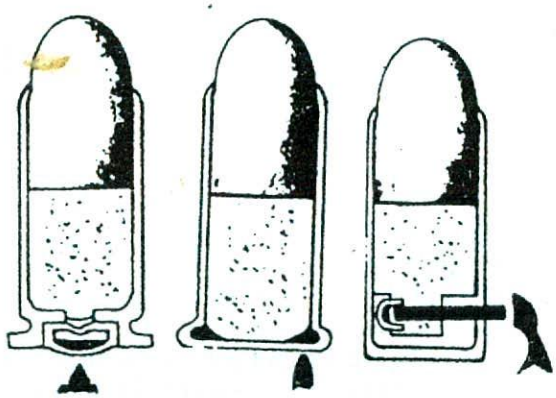


Fig. (8–37) Three types of cartridges. (1) Centrefire. (2) Rimfire. (3) Pin-fire

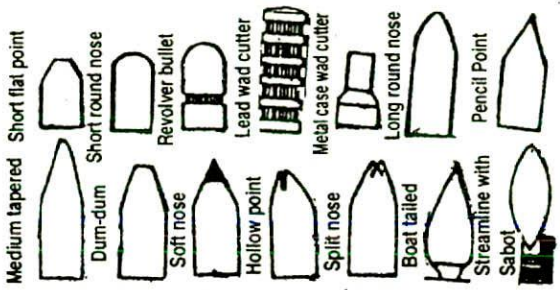


Fig. (8–38) Types of bullets.

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.

Bullets: The traditional bullet is made of soft metal and has a rounded nose. The metal is lead with varying amounts of antimony added to provide hardness. This is known as the round-nose soft bullet, and is usually used in rifles and revolvers. The tip of the bullet is called nose. The calibre of a bullet is its cross-sectional diameter. In revolver and pistol, the bullet is short and the point usually round or ogival. In rifle, the bullet is elongated with pointed end. Variations are: (1) Square-nosed, soft metal bullet, known as "wad-cutter", and used primarily for target shooting. (2) Hollow-point variety has a depression in the nose of the soft metal. This bullet is designed to expand or "mushroom" upon impact (Fig. 8–38).

Jacketed bullets are of two types: (1) The full metal jacket bullet in which a tough, heavy jacket covers all but the base, where the soft metal interior is exposed. The tough jacket may be made of steel, copper, nickel and zinc. (2) The semi-jacketed bullet is provided with a relatively thin but tough jacket, which covers the base and the cylindrical portion of the bullet, leaving the nose partly or fully exposed. This type is designed to expand or "mushroom" like the soft metal, hollow-point type. The combination of the above two is also available. **Mushroom bullets** produce more serious wounds. Other types of bullets are: (1) Short flat-point. (2) Medium flat-point. (3) Medium round-nose. (4) Long round-nose. (5) Medium long sharp-point. (6) Medium sharp-point. (7) Flat base. (8) Sharp-point boat-tailed. (9) Pencil-point. (10) Streamlined with sabot.

A DUMDUM BULLET is open at the base, and has the point covered with the jacket. When it strikes an object, the lead at the point expands or mushrooms, and produces a large hole. They are not in use. Frangible bullets completely disintegrate. A bullet with a hole in the point is called hollow-point or 'express' bullet. Incendiary bullets contain phosphorus, so that it catches fire on hitting the target. FRANGIBLE BULLET is made of iron which fragments on impact.

EXPLOSIVE BULLETS: If explosive bullets are present in the body, ultrasound or microwave diagnostic techniques must be avoided, as they can cause the projectiles to detonate. The exploding bullet is of various types. The wound produced is larger than the usual. If the missile explodes, there is greater fragmentation of the bullet and increased destruction of the tissues. The surgeon and pathologist should wear goggles and use long-handled instruments to manipulate the missile during surgical operation or autopsy, as they are vulnerable to detonations of explosive missiles. The removed bullet should be handled with long rubber-covered forceps and kept in a padded container to protect it from excess impact, vibration and heat.

PLASTIC BULLETS: Baton round (plastic bullet) is a solid cylinder of polyvinylchloride (PVC), 38 mm. in diameter, 10 cm. long, weighing 135 g. It is fired from a smooth-bore weapon and is effective up to 50 to 70 metres. They are used for riot control. It should not be fired at a person under 20 metres range. It causes bruising and pain. Fracture of the skull, eye damage, fracture of ribs, limb bones, and contusions of liver, lung and spleen have been reported.

MECHANISM OF DISCHARGE OF PROJECTILE: A firearm is fired when the trigger is pulled, which is usually situated below, or below and behind the breech. The trigger releases a pin or hammer, whose tip strikes the percussion cap at the base of the cartridge, which contains explosive mixture known as priming, which explodes by heat created by the strike of the firing pin and sends a flash through a tiny hole into the main body of the powder-filled case, which sets fire to the powder charge or propellent, producing instantaneously a large amount of gas which is under high pressure. The cartridge case swells outwards, due to which the hold on the bullet (missile, projectile) is released, and forces the bullet into the barrel and passes out. The confined gas behind it gives recoil thrust to the gun. Noise of gun firing is caused by muzzle blast, or due to the suddenly released gases disturbing the air. If the velocity of the bullet is more than the speed of sound, then there is also a crack from that. The bullet reaches its maximum velocity as it comes out at the open end of the barrel and this is called muzzle velocity. The bullet is followed by

a blast of highly compressed hot gas, particles of partially burnt and unburnt powder, smoke, flame and fragments of metal, cartridge and detonator, grease and wad or disc matter. The blast has the shape of a cone whose apex is located at the muzzle. All these things produce some effect on the body at short ranges.

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. Because kinetic energy increases in direct proportion to weight (mass) of the missile and the square of its velocity, a bullet travelling at twice the speed of a second bullet of equal weight and similar shape, possesses four times much energy or wounding power. Tissue damage produced by high velocity bullets is disproportionately greater than that produced by bullet of ordinary muzzle velocity. A wound produced by a bullet, whose impact speed equals its muzzle velocity is more severe, than that produced by the same bullet, discharged from the same gun, whose speed has been reduced due to travelling a long distance before it strikes its target.

(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.

FIREARM WOUNDS

They are usually recognised without difficulty. Wounds produced by rifled weapons may simulate wounds inflicted by a red-hot poker or a burning pointed stick. Glancing wounds may simulate incised or lacerated wounds. A bullet that passes through glass or some other object, and then strikes the body may produce a wound resembling a laceration. Firearm wounds cause an actual hole. A stab wound causes a slit-like opening which tends to seal after the knife is withdrawn.

WOUNDS FROM SHOTGUNS

The smoke extends up to 30 cm., flame up to 45 cm., and unburnt and partially burnt powder grains up to 60 to 90 cm. The larger the calibre, the greater the distance to which the powder is discharged. The cards travel for two metres, and wad for 2 to 5 metres. Wadding can produce minor injury, up to a distance of three metres.

The character of a wound depends on. **(I) The Distance From which the Weapon is Discharged (Fig. 8-42):** The discharge produces a long, shallow cone with its apex close to the muzzle of the shotgun and the further along the cone the victim is situated, the larger the wound pattern will be. **(1) Contact and near wounds:** They are single, usually round, equal to the bore in size, often ragged because of individual shot and tearing due to gases. In tight contact wound, soiling and burning are minimal or absent. The margins of the skin perforation are charred by flame, and the abraded border is usually soiled with powder residue. Usually smoke soiling of at least some of the margin of the wound is seen. As the gases are blasted within the wounds, the subcutaneous and deeper tissues show severe disruption. The rapid entry of gases causes a momentary vacuum immediately below the skin, which may cause extrusion of the soft tissues, e.g., fat through the wound. Particles of unburnt powder are driven to some distance through the wound and some of them are found embedded in the wound. These particles cause haemorrhage in deeper tissues and form aggregates of haemorrhages in the margins of the wound. Thus, the margins of the wound will be contused. If the contact is tight, muzzle impression **(copy or recoil abrasion)** is seen due to firm mechanical pressure of impact of the metal rim against the skin, and also due to the subcutaneous expansion of gases lifting the skin forcibly up against the muzzle. The muzzle imprint may be an incomplete, indistinct bruise or rarely may be a perfect imprint of the end of the weapon. Bruising can occur around the muzzle imprint. Muzzle imprint may be lost within the explosive damage associated with the discharge. But, in a double-barrelled shotgun the unused barrel often leaves a characteristic patterned abrasion. In many cases, muzzle impression is not produced due to rapid removal of the weapon by recoil. If the muzzle is not pressed firmly, and also due to the recoil of the gun, flame, gas and soot escape sideways and cause singeing and blackening of the surrounding skin. Corona may be present. CO in the gases combines with haemoglobin due to which the wound of entry and the wound track appear pink. This decreases gradually, but may be seen very rarely at an exit wound. Burning,

blackening and tattooing of the tissue also take place in the depths of the wound. The outer shots are deformed by their passage through the bore and choke.

Cruciate, stellate or ragged lacerations are seen especially if there is a thick bone immediately under the skin, such as skull due to the extreme force of the blowback phenomenon, as gases expand beneath the skin and lacerate the margins of the wound, as they exit through the original entry wound. Contact wounds on the head produce greater disruption of the margins, and often show subsidiary linear tears in the skin extending from the margins of the main wound. A large irregular hole is produced in the skull, with fissured fractures running from its margins. The bone may show burning and gunpowder blackening in the margins (burst head). The entire contour of the face and head may be destroyed, and the actual point of the muzzle impact of entry wound may be difficult to locate. Extreme mutilation is caused due to the explosive effect, because the gases have restricted space for expansion. Comminution of the vault of the skull is usually accompanied by extensive fissured fractures, or 'crazypaving' fracture, of the base of the skull, and the roofs of the orbits, and middle ears. A large exit wound may be produced with disruption of the cranium and projection of brain tissue for some distance. An eye may be blown out of its socket. An entrance wound in the mouth or nose may or may not be accompanied by an exit wound. Resistance by the hard palate reduces the power of the shot, and the shot lie mostly in the space between the skull and scalp, at the back or side of the head. Abrasions or bruising of one or both lips with or without laceration is seen. Splitting of the angle of mouth may occur due to blast. In the neck region a large exit wound or complete destruction may occur. In contact or near contact wounds on the abdomen, coils of small intestine may lie outside the abdomen due to entry of gas into the peritoneal cavity (Fig. 8-39). On the chest, the skin and subcutaneous tissues may be peeled away from the rib cage to form a pocket which then collapses.

If the part is clothed, smoke will escape sideways and may be found in each layer of clothing and on the skin. The cloth may be singed at the edge of the hole, and there may be a ring of burning around the skin wound.

(2) Close Range (up to one metre): In loose contct with skin, the blast effect is less as compared to tight contact and splitting of the wound margins usually does not occur. Within a distance of about 30 cm., tissues surrounding the wound are singed by flame, and blackened by smoke, and tattooed by unburnt or partially burnt powder granules. If the powder is smokeless, there may be greyish or white deposit on the skin round the wound. The deposit of smoke is known as smudging, fouling or blackening. This spreads more widely than powder tattooing. The hairs of the trunk and limbs may be completely burnt around the wound. If the distance is greater, the keratin of the hair may melt with the flame, and then solidify on cooling, producing clubbed appearance of the hairs because of rounded bulges at the tips. Soot soiling diminishes as the distance increases. There may be a wide flare or narrow rim of hyperaemia or even blistering from the flame. If the gun is fired at right angle to the body, the burnt area is circular, and if fired at an angle it is oval, the direction of the firing being indicated by the nearness of the wound to one or other end of burnt area. The end nearer the wound is the direction towards which the shot travelled. The blackening can be removed by wet cloth. Primer residues are easily removed from the skin by wiping, rubbing or washing. Unburnt particles of the powder are embedded in the skin producing tattooing (stippling or peppering). The particles sticking to the epidermis or lodging in the epidermis superficially



Fig. (8–39) Herniation of intestines through the entry wound of shotgun.

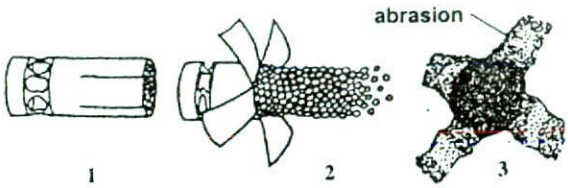


Fig. (8–40) Shotgun wad with enclosed lead shot in plastic cup. (1) Cup is not opened. (2) Between 30 to 60 cm. the sleeves of cup open and are forced backward. (3) Cross-like pattern of abrasion around the entry wound.

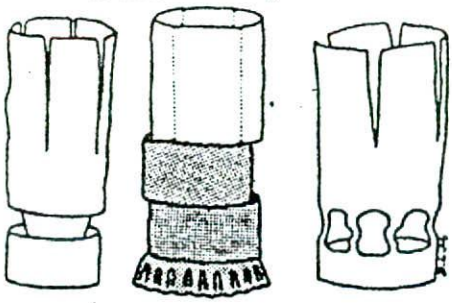


Fig. (8–41) Typical wads which may be recovered from the body at autopsy.

can be removed with pressure wiping, showing punctate wounds. The use of silencers to muffle the sound of discharge reduce the amount of smoke and powder significantly and cause misinterpretation of the distance from which gun was fired. The burnt area darkens and parchmentises on drying after death. Blackening and tattooing can be readily demonstrated by infrared photography on both skin and clothing.

The tissues up to 30 cm. along the track, and around the wound may be cherry-red due to absorption of CO. The wound is single, circular or oval, similar to contact wounds, though the blackening and tattooing are more extensive. The margins of the skin wound may be clean-cut or slightly ragged. There may be annular bruising around the wound due to tissue damage from the entry of gases. Felt, wads or plastic cups from the cartridge will be found in the depths of the wound. The deeper tissues show marked disruption. Carbon monoxide may be present in the blood absorbed from the damaged tissues. In some cartridges, plastic granules may be used as a filler between the lead pellets, and this coloured material may be found within the wound or on the skin up to 2 to 3 metres and produces very fine, punctate abrasions around the pellet holes. The plastic cup type wad opens up between 30 to 60 cm. so that four petals stick out, and a circular entrance wound is produced with a Maltese cross pattern of abrasion encircling it (Fig. 8-40). By 90 cm. air resistance folds back the petals and a single hole of entrance will be produced.

At a distance of 60 to 90 cm., small shot produce a single circular aperture, 2.5 to 4 cm. in diameter, with irregular and lacerated edges. There is no burning, or blackening. Some amount of tattooing is usually seen. The tissues within and around the wound are not cherry-red. Between 30 cm to one metre, the rim of the wound is irregular and shows some scalloping, often referred to as a "rat -hole" The shots are scattered after entering the body and cause much damage to the internal tissues. In the skull, there is less disruption than that at contact range; bursting open of the skull and scattering of its contents is not seen. The column of pellets has a cutting action which produce a clean hole. Shotgun wounds at contact and close range cause much more destruction of tissues than rifled weapons, due to the greater amount of gas produced, because of larger amount of gunpowder.

(3) Short range (1to 2 metres): A single circular aperture 3 to 4 cm. in diameter, with irregular and lacerated edges is produced. There is no burning, blackening or tattooing. Between one and two metres, the shot mass enters the body in one mass, producing a round hole, four to five cm. in diameter for all gauges and all chokes. Annular or linear abrasions are caused by the impact of the clothing against the stretched skin during the

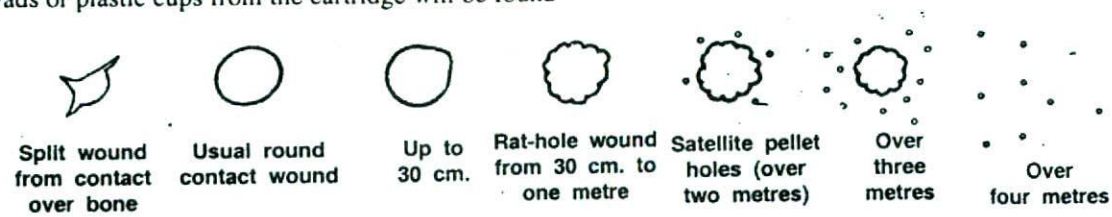


Fig. (8–42) Shotgun wounds at varying distances.

penetration of the shot *en masse*.

(4) Intermediate Range (2 to 4metres): The minimal distance at which shot mass begins to spread is extremely variable. It may be one metre with sawed-off shotgun but is usually about two metres with cylinder bore guns and four metres with full choke guns. At a distance of two metres, the shot mass begins to spread and individual pellet holes may be detected, which are usually round and show a rim of abrasion at their margins. The wound of entry is irregular. Wads may be found deep inside the wound up to two metres. Beyond two metres the wads often strike the body below the shotgun wound. It may penetrate the skin or it may only bruise the skin. The wads found within the wounds are useful to determine bore of the gun.

At a distance of three metres, the central aperture is surrounded by separate openings in an area of about 8 to 10 cm. in diameter. As muzzle-target distance increases, the main entrance defect progressively becomes smaller, and individual pellet wounds increase in number. Occasionally, several individual pellet entrance wounds are in contact producing scalloped defects which are larger than the individual pellet holes. As lead is soft, pellets deform easily due to friction as they rub against the inside of the barrel. The heat can cause melting and fusion of pellets.

(5) Long or Distant range (above 4 metres): At a distance of 4 metres, the shots spread widely and enter the body as individual pellets producing separate openings in an area of 10 to 15 cm. in diameter. Each individual pellet reproduces its own track. In the skull, the energy of the shots is greatly reduced perforating the skin and bone, so that they usually do not travel the entire brain substance. Cavitation is usually minimal or does not occur, and distant contusions are rare. Laceration of blood vessels by pellets or bone fragments may produce infarcts. The spread of pellets from a fully choked barrel is:

10 metres . . . 25 cm.
15 metres . . . 35 cm.
20 metres . . . 45 cm.
30 metres . . . 75 cm.

The spread is almost double from an unchoked barrel (Table 8-10 &11). At about 30 metres, the pellets only penetrate the skin or muslce. Death beyond a range of 30 metres is rare. Passage of shotgun pellets through any target before they strike the body, cause the pellets to spread, e.g., window glass, screen, or layers of clothing.



Fig. (8–43) X-ray photograph of the skull showing multiple pellets underneath the subcutaneous tissues of the face over an area of 12 x 8 cm. A case of shotgun injury from a distance of 5 to 6 metres.

Country-made Firearms: The injuries caused to the victim vary considerably depending upon the nature of projectiles. Metal scraps may produce small incised or incised-looking wounds; punctured wounds may be produced by nails, and lacerations and contusions by stone pieces. The distance up to which the smoke, flame and carbon particles travel is

Table (8–10) Approximate spread of shot in centimetres at various distances.

	5 metres	10 metres	15 metres	20 metres
Cylinder	20	50	65	75
Half-choke	13	30	40	50
Full-choke	8	25	35	45

Table (8–11) Percentage of shot falling within a 75 centimetres circle at various choke adjustments.

Choke adjustment	Average percentage
Full choke	70
Three-quarter choke	65
Half-choke	60
Quarter-choke	55
Improved cylinder	50
No choke	30 to 40

approximately half of that from factory made firearms. The pellets begin to disperse at a distance of 30 to 45 cm. As the characters of the firearms and components of the cartridge vary widely in country made firearms, the data applicable to factory-made firearms to calculate the distance of firing cannot be applied. For estimating the distance of firing, the only ideal method is to carry out test firing with the suspected weapon.

BILLIARD BALL RICHOCHET EFFECT: At close range, while the shots are bunched, they strike one another upon impact on primary target, i.e., the skin or clothes, and spread out in a wide pattern as they pass through the body. This causes the shots to spread widely and may suggest a greater range of fire than actually occurred. Similar spread is seen if the shots strike any other intermediary object, e.g., door or window, before reaching the victim. The phenomenon is termed the billiard ball ricochet effect. The final shot spread as seen in X-ray gives a true picture of the range of fire, only when the range of fire is great enough that the shots are spread out, before striking the target.

In decomposed and burned bodies where the skin pattern cannot be seen, the range of fire can be determined by X-rays.

BALLING OR WELDING OF SHOT: Balling of shotgun pellets results in the conversion of shot into a compact mass, which can travel for few metres in this form. In such cases, a circular or oval entrance wound of about 5 to 10 mm. in diameter, and widespread, small, circular punctures are seen, suggesting the use of two different weapons, one a shotgun at distant range and the other a rifle.This can be due to faulty manufacture or old ammunition, but this is rare. It can be due to hand-loading of cartridges, if too much powder is used, if wads of incorrect kind are inserted, or sealing pressure on the wads is too high. The most likely cause is due to pouring of paraffin wax into the cartridge after removing the outer cardboard or by replacing some of the pellets by a large ball-bearing which is held in place with wax.

(2) The Size of the Shot : The smaller the shot, the more minutely irregular are the edges. The penetrating power of the large shot is greater than that of the smaller shot. At close range, the wounds produced by shot of various sizes are similar, but small shots usually lodge in the body.

(3) The Nature of the Explosive : With smokeless powder, blackening and tattooing is less marked at all ranges than with black powder.

(4) The Gun itself : The pattern of wound depends on the length of the barrel, the bore, absence or degree of the choke present. Shorter barrels usually produce greater deposits over larger areas.

As range becomes greater, the intensity of blackening and tattooing decreases and the spread increases in a fairly regular manner. The diameter of the wound is measured from the outermost of the individual pellet holes in centimetres. For cylindrical barrels one-third of the spread of shot in cm. equals the range in metres. The spread for fully choked barrels is about half that of a cylindrical barrel. It is possible for a single pellet or shot to cause death. The estimation of range is difficult when replaceable or variable extensions called 'chokes' are used at the end of the muzzle of shotguns. Some chokes and even the barrels themselves are replaceable.

Irregular missiles, such as pieces of stone, iron, beads of metal, seeds, etc., used in muzzle-loading guns, produce several irregular lacerated wounds, and the exit wounds are larger than the entrance wounds. Death from shotgun injury is usually due to haemorrhage.

The direction of the fire may be determined from the passage of individual shots through the clothes and tissues, from the grouping of shot marks and the direction of glancing shots.

Exit Wounds : Usually shotgun pellets do not exit from the body except : (a) Contact wounds. (b) Tangential wounds where some of the pellets have a very short track through the body. (c) Thin part of the body, such as the neck or extremities. (d) Wounds caused by large calibre buckshot or rifled slugs.

At contact or near range, greater disruption of tissues occur than is seen in entrance wound. The margins are everted as the unsupported skin is struck from within, the tissues tend to burst outward, and the skin fragments, but there is no singeing, blackening or tattooing of the margins. There may be small, separate wounds made by individual pellets that have become separated from the mass. If the skin at the exit of a bullet is well supported, either by belt, corset, etc. or is pressed against a firm surface, e.g., wall, wooden or plaster board, etc. the skin surface will be rigid because of the pressure of the external substance, and the exit wound has clean margins.

WOUNDS FROM REVOLVERS AND AUTOMATIC PISTOLS

Entrace 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. Silencers will filter out a great proportion of soot and powder particles due to which the range appears greater than it actually was

(1) Contact Shot : 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 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. Singeing of the hair may be present due to the escape of hot gases by the sides of the muzzle end. The area immediately around the perforation is abraded, and this thin rim of abrasion is usually covered with powder residue. Tattooing is minimal or absent. Tattooing begins when the muzzle to target distance exceeds one cm. In firm or hard contact (muzzle pushed hard against the skin), 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

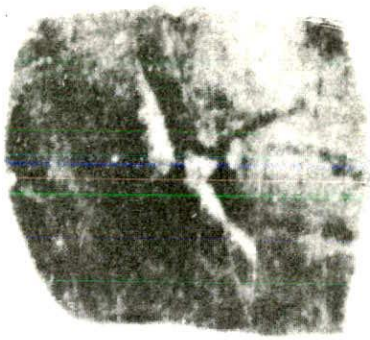


Fig. (8–44) Cruciate injury of the scalp caused by contact shot from a pistol

body. The wound is not eruptive or explosive in appearance. 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".** Back spatter is more common with shotgun. 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



Fig. (8–45) Appearances of wounds of entry by a rifled weapon.

discharge from the muzzle, i.e., gases, flame, powder, smoke, and metallic particles are blown into the track taken by the bullet (projectile or missile) 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. This results in undermined, ragged, stellate, triradiate or cruciform opening with everted margins from which tears radiate. The tearing may be severe, as the gas raises a large dome under the skin which then ruptures. Such wounds are usually produced by large calibered pistols. A subcutaneous pocket containing blood mixed with gunpowder is formed by separation of the tissues. When a small calibered pistol is used, the wound may be small and regular. Soot may be deposited on the bone surrounding the bullet hole. This should not be mistaken for lead rubbed off on the bone during the passage of the bullet, which is seen only in a localised area. Frequently, soot passes deeper into the wound track and a faint gray or black discolouration may be seen on the inner surfaces of the skull around the bullet hole and on the dura mater. Fissured fractures often radiate from the circular defect due to the considerable sudden expansion resulting from the muzzle blast. Fractures of the orbital roofs occur due to the same mechanism, but the dura over the orbits is usually not damaged. A bullet travelling the cranial cavity destroys the structures in its pathway and produces an expansile or explosive effect. A bullet fired from a short distance may produce an explosive effect sufficient to burst the scalp, shatter the skull and dislodge the brain. In less severe type of injury, a cone of damaged cerebral tissue is seen surrounding the wound track. When a bullet moves through the body, it transfers kinetic energy to the surrounding tissue, which is thrown forcefully away from the bullets path in a radial manner, and a temporary cavity is formed in its path. 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.

When the part is clothed, the bullet hole in the cloth touching the muzzle is sometimes surrounded by a flat ring corresponding to the outline of the muzzle. The loose fibres of the cloth in the centre of the bullet hole are often turned outward due to the expanding gases returning through the defect. These fibres are usually blackened by smoke. In synthetic fabrics, melting of the ends of these fibres may be observed sometimes. Varying amounts of soot is deposited on the edges of the bullet hole. If the clothes are bloodstained, deposits of soot may not be recognised, but the inside of the garment may show large deposit of smoke. This is due to the spreading of smoke by the muzzle blast between the skin and the clothing and is seen commonly if the shot passes through several layers of material. Each layer is blackened on both sides of the fabric, but the skin wound does not show blackening or tattooing. If there is no intervening clothing, the smoke passes into the wound but a small amount is deposited on the wound edges, especially if contact with the muzzle was loose. Fine granules of powder and deposits of soot can be demonstrated in the depth of the wound by a layerwise dissection of the injury.

(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 palms and soles are very resistant to powder tattooing. The blackening can be wiped off the skin by a wet cloth, but the tattooing cannot be wiped off. Carboxy-haemoglobin will be present in the wound track in diminishing concentrations. The length of the barrel of a firearm has considerable effect on the spread of smoke produced on the target, e.g., a gun with a 5 cm. barrel will spread the smoke over a much larger area than a weapon having a 15 cm. barrel, fired from the same distance and using the same type of ammunition. 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, slightly smaller than the diameter of the bullet, due to elasticity of the skin, with a bruised and inverted margin and a zone of blackening and tattooing. The individual tattoos are caused by the individual unburned powder grains being blown into skin. A small magenta-coloured zone, an actual micro-contusion is seen surrounding each tattoo point, which is caused by the trauma of the high speed impaction of the powder grain with rupture of small blood vessels and resulting minute haemorrhage. 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. Occasionally, when the range of fire is short, small fragments of metal derived from the interior of the barrel of the gun or the bullet itself, are embedded in the skin in the vicinity of the entrance wound.

(4) Distant Shot : The entrance wound is smaller than the missile due to the elasticity of the skin, circular, and margins are inverted (fig. 8-46). Distant entrance wounds of the palms and soles are irregular, often having a stellate appearance, without an abrasion ring, and look like exit wounds. 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. In case of semi-jacketed bullet, the jacket separates as it goes through the body, and the core mushrooms into small pieces. X-ray gives a picture of "lead snowstorm". If a semi-jacketed bullet passes through an intermediary target, the jacket may separate from the core, and both missiles may penetrate the body. The calibre of a bullet cannot be determined, if it strikes the skin surface obliquely.

Stretching and cavitation and dissipation of kinetic energy are the major causes of the lethal

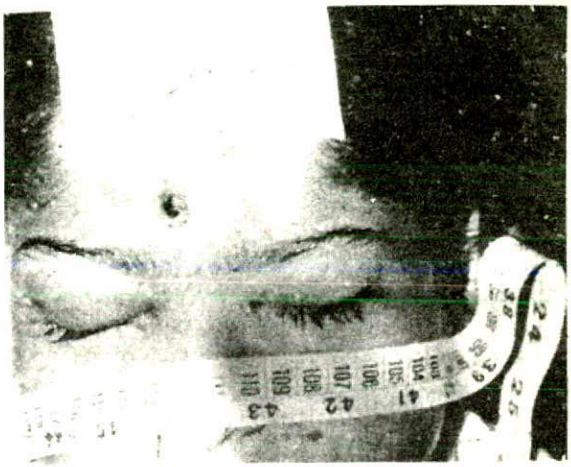
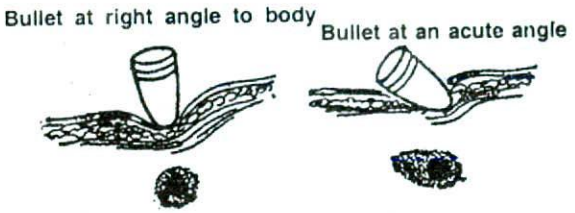


Fig (8–46) Homicidal rifle wound of entry.

effects of a bullet, together with deformation and fragmentation.

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.8-47). A black coloured ring **"grease or dirt collar"** (bullet wipe soiling) 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. It is more marked in a distant shot. 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 is reddish at first, but becomes reddish brown as it dries. Some contusion is present in abraded collar, and as such, it is also called **"contusion collar"**. 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. Abrasion collar may be absent when the tissues are soft and yielding, e.g. in the abdomen or buttocks. In addition to abrasion collar, there is often a slightly wide circle of peeled keratin, where the stratum cornem of the skin is raised to form a



Fig(8–47) Abrasion collar of an entrance bullet wound.

slightly frayed edge around the entry wound.

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.48 & 49). 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. Pieces of bone may produce short accessory wound tracks. As the bone fragments have to pass through the dura before entering the brain, lacerations are usually irregular and involve leptomeninges. At the point of exit, a punched-out opening is produced in the inner table and bevelled opening on the outer table. Bevelling is produced when the unsupported diploe everts and fragments on the side where the bullet leaves, this in contrast to the approach side where the rim of the defect is supported by the underlying bone. The exit wound is larger due to the deformity and tumbling of the bullet after entering the skull. There are often fissured, sometimes comminuted fractures radiating from the central hole. Asymmetry of the bevelling is useful in assessing the angle of fire.

The same appearance is seen in sternum, pelvis, 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.

Direction : If the bullet strikes the body at right angle, the abrasion collar is circular and uniform because the scraping by the bullet is equal on all

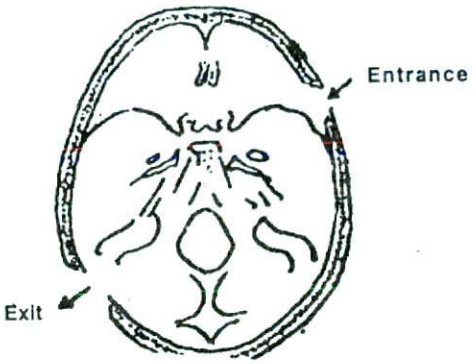


Fig (8–48) Shooting appearance of entrance and exit wounds in skull.

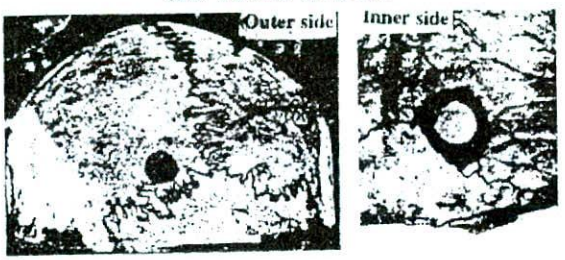


Fig. (8–49) (A) Skull cap showing the entrance hole of a revolver bullet with clean-cut margins. (B) The inner side of the same bone showing bevelling of edges.

sides of the wound. If the bullet strikes the body at an angle, the wound itself is round, but the marginal abrasion is oval or elliptical, due to increased width on the side from which the bullet comes, due to the bullet moving across a wider surface of the skin on that side. The direction of the bullet is from the wide to the narrow side. Oblique angle will cause an elliptical mark, the length of which increases as the angle decreases. When the bullet enters the body from an oblique angle, one edge of the wound is shelved or undercut, which indicates the direction from which the bullet entered. Shelving is usually seen in the deeper layers of the skin rather than in the tissues below.

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 square 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. The tissues show considerable damage due to the wobbling of a bullet within the body. 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. It is easily deviated and deformed by striking some hard object, and often lodges in the body. Large bullets cause greater damage than small ones. Round bullets produce larger wounds than conical ones. A bullet of smaller size than the calibre of the weapon may cause only bruising.

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.

(B) 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. Full metal-jacketed bullets usually exit undeformed. Exit wounds may vary considerably in size and shape. They may be round, stellate, cruciate, elliptical, crescent shaped. An exit wound produced by a tumbling bullet or a bullet passing sideways through the tissues may be elongated or slit-like resembling a stab, incised or lacerated wound. The same appearance may be seen in a part of the body in which the skin folds or changes direction, e.g. in the buttock crease, under the arm, or in the groin or umbilical area. Exit wounds of the head are usually star-shaped resembling contact entrance wounds. In some cases, the entrance and exit wounds may look alike. The exit wound is of help in determining: (1) the direction of fire, (2) posture of the victim at the time of the shooting, and (3) the number of bullets in the body. When the weapon has been fired in contact with the bone or at very close range, the exit wound is usually smaller than the wound of entrance. With increased range, the exit wound is larger than the wound of entrance. With high velocity bullet, the two wounds may be of the same size. 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 (usually broader than that of entry wound), resembling a wound of entrance **(shored or supported exit wound)**. The shored type of exit wound caused by

Table (8–12) Difference between wounds of entrance and exit of a bullet wound.

Trait	Entrance wound	Exit wound
(1) Size :	Smaller than the diameter of the bullet. In close discharge, skin is torn.	Bigger than the bullet.
(2) Edges :	Inverted.	Everted, puckered or torn.
(3) Bruising, abrasion and grease collar:	Present.	Absent.
(4) Burning, blackening, tattooing:	May be seen around the wound.	Absent.
(5) Bleeding:	Less.	More.
(6) Fat:	No protrusion except in contact shot.	May protrude.
(7) Tissues within and around the wound:	May be cherry-red due to CO of explosive gases.	No colour change.
(8) Fibres of clothing:	Turned in and may be carried into the wound.	Turned out.
(9) Lead ring or metal ring:	May be seen around the wound by radiological examination.	Absent.
(10) Spectrography:	More metal is found around entrance wound, if bullet has only passed through soft tissues.	The exit wound may contain more metal if a bone is struck nearer to it.



Fig. (8–50) Appearances of wounds of exit by a rifled weapon.

a bullet fired at long range or through clothing, or when the firearm is of small calibre and discharged in contact with the skin at a point where bone is not immediately below the skin surface, resembles wound of entry. 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, door, car seat, 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). The variation in the shape and large size of exit wound are due to : (1) The bullet tumbles in the body and fails to exit nose-end first. (2) The bullet is deformed. (3) The bullet breaks up in the tissues and exits as several pieces. (4) Fragments of the bone may be blown out of the body with the bullet. (5) The unsupported skin at the exit tends to tear and break into pieces. Rarely, slit-like exit wounds are seen probably due to the deformity of the bullet, caused by impact of the bullet on bone during its passage through the body. Such wounds are mostly seen on the head and over the shoulders. Occasionally, a bullet may be found protruding from an exit wound. In a fatty person, the edges of both the wounds of entrance and exit may be everted due to protrusion of fat. They may also be everted in decomposed bodies (Fig. 8-50).

Entry wound may be larger than exit due to: (1) tearing of soft tissues by inrushing gases, (2) tumbling or yawning of bullet, (3) breaking of bullet with only a portion of it exiting, (4) tangential entry with focal avulsion of tissues, (5) bullet entering through folded or creased skin.

Revolvers and automatic pistols cause similar wounds, but penetrating power of bullets of pistols is much greater because of the greater velocity and because of their being coated with hard metal.

WOUNDS FROM RIFLE : The wounds inflicted on the body are similar to pistol wounds but produce more damage. They vary considerably and produce most unexpected results. The flame may extend up to 15 to 20 cm. Unburnt powder grains and small metallic particles are not found beyond 75 cm. Smoke is absent beyond 30 cm. from the muzzle.

In contact wounds, the burning and tattooing is not much. The blast effects are also much less and the splitting of the clothes or tissues is the same as that with the revolvers. The entrance and exit wounds may be of the same size and shape, if the bullet passes through the body without touching the bone; but even when no resistant structure is touched, there may be explosive effects and severe lacerated exit wounds. The entrance wound is usually smaller than the diameter of the bullet and looks like a wound made by forcing lead pencil into skin. The edges are depressed, may have microtears 1 to 2 mm in length, radiating from the surface, and surrounded by a reddish zone which becomes brown on drying. Bruising of the deeper tissues around the track of the bullet is seen. If the bullet strikes at an angle, the skin may split or be turned up. If it strikes a bone, extensive shattering and comminution of bone takes place. In such cases, the wound of exit is usually a lacerated hole, varying from about 2.5 cm. to the size of the palm of the hand. Frequently, several small holes will be found around the large exit caused by fragments of bone being driven out. Bullet fragmentation is much more common in rifle injuries than those due to handguns. In wounds of the head within 300 metres, the brain is frequently pulped and a great part of the cranium slashed into fragments.

A bullet leaving the muzzle of service rifle rotates or spins round its long axis at the rate of about 2,500 revolutions per second, and for the first 200 to 300 metres, the base of the projectile has also a circular motion round the axis of flight, gradually losing this motion and continuing to spin in the true axis. If a resistant body is struck within a range of 200 to 300 metres, the effect produced by the spin of the bullet together with the intense liberation of energy is similar to that of an explosion, the tissues themselves exploding ne the bullet. This explains the shattering of bon suc : pelvis, femur, skull, etc. It also explains t seve. iceration seen even when a rifle bullet fired at short range passes through soft tissues only. At



Fig. (8–51) Rifle bullet passing through forearm and abdomen.

these ranges, the bullet commonly disintegrates, and causes effects which give the impression that more than one shot has been fired. Between 300 to 1,000 metres, the spin of the bullet becomes regular, and it passes easily through the tissues and cuts a clean hole through the bones. Beyond this, it behaves like a low-velocity bullet.

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.

PECULIAR EFFECTS OF FIREARMS

(1) Atypical Entrance Wound : (a) For a few microseconds after the bullet leaves the muzzle (up to about 50 metres for a pistol or 150 metres for a rifle), there may be a "tail wobble' or "tail wag". This is partly responsible for the great tissue damage and the large atypical entrance wound at short range.

(b) The gyroscopic effect of the bullet diminishes as it reaches the end of its fligh, until it begins to wobble, and then to tumble. In wobble, yaw or tumble, the impact of the bullet may be sideways or even backwards with an irregular lateral motion, due to which an irregular lacerated wound is produced. A bullet travelling in an irregular fashion instead of travelling nose-on is called a **yawning bullet** (fig.8–52), and a bullet that rotates end-on-end during its motion is called a **tumbling bullet** (fig.8–53). The amount of tissue crush might be three times greater when the bullet yaws to 90°.

(c) The tattooing seen on the skin may be altered by the use of 'silencers'. Muzzle-brakes and flash-hiders may produce peculiar blackening and tattooing patterns by allowing gases to pass in specific direction. Bullets fired through a shortened or sawn-off barrel, may be deformed or squeezed.

(d) In revolvers, weapon defects, such as cylinder misalignment will cause deformities of the missile, with breaking off of the metal fragments from the bullet occurring when the misalignment is great. Lesser defects of this type may cause tumbling.

(E) RICOCHET BULLET : A ricochet bullet is one which before striking the object aimed at, strikes some intervening object first, and then after recocheting and rebounding from these, hits the object. They are rare, as most bullets on striking a hard surface break up or penetrate the surface. The critical angle of impact for ricochet for hard surfaces varies from 10 to 30°. The bullet ricochets off at an angle smaller than the impact angle. The path of a ricochet is completely unexpected. The bullet may ricochet before or after striking the body and may produce a non-penetrating or a penetrating injury. Ricocheting of a bullet may occur with inferior firearms and low velocity bullets. The bullet may be deformed and flattened before striking the skin. The degree of deformity varies depending on the texture of the bullet. This produces a large irregularly oval, triangular or cruciate entrance wound with irregular abraded margins. As the bullet loses



Fig. (8–52) Schematic depiction of "yaw' associated with missiles of very high velocities.



Fig. (8–53) Schematic depiction of "tumble' associated with missiles of very high velocities.



Fig (8–54) Internal ricochet. (1) Single ricochet. (2) Double ricochet. (3) Inner tangential at contralateral side. (4) Inner tangential at contralateral side and ricochet. (5) Inner tangential at entrance side.

gyrating movements, abrasion collar is absent. Burning, blackening and tattooing are also not seen. Sometimes, a bullet may strike an object and tumble, and hit the body side on, producing an elongated wound of entrance like a keyhole. Particles of the substance against which the bullet is deflected or has struck, e.g., soil, fibres, paints, etc. may be found adhering to the bullet. The nose of the bullet may be found facing the entrance wound due to deflection in the body. When the velocity is lost, the bullet only produces an abrasion or contusion.

Sometimes, after passing through the brain, there is not enough energy left in the bullet to penetrate the skull. It may rebound (ricochet) from the inner table of the skull like a billiard ball, producing a second track. If it ricochets for a second time, a third wound track is produced. If the bullet enters the skull at an acute angle towards the inner table, it may move along the dura tearing the leptomeninges and blood vessels and produce a shallow wound track along the surface of the brain. The inner divergent course of the bullet may be combined with internal ricochet (fig. 8–54).

(f) The bullet may strike the surface producing a contusion and fall to the ground. This is seen with relatively soft non-jacketed bullet fired from a firearm that has an eroded or worn barrel; and also when the ammunition used is of smaller size than the barrel of the gun.

(g) **Bullet Graze or Slap :** If a bullet strikes skin at so acute an angle that it does not perforate the skin, a grazing or slap wound is produced. It usually appears as reddish-brown, elongated, elliptical or triangular furrow or abrasion. The underlying dermis may or may not be involved, but the adjacent intact skin may be ecchymosed.

(h) The bullet strikes the sternum at just the right angle and is deflected, so that it continues around the rib cage, between the bone and the skin. It may come out at the back of the body after causing only a superficial injury; or it may lodge between the skin and the backbone.

Bullets that strike in unusual location may cause injury and death, but the wound of entry may be difficult to locate, e.g., ear, nostril, mouth, axilla, vagina and rectum.

(2) Single entrance and multiple exits : If the bullet splits up within the body and divides itself into 3 or 4 pieces, there will be only one entry hole, but several exits. The bullet striking a bone may break the bone into fragments, which act as secondary missiles producing multiple exits. Sometimes, the jacket of a bullet may separate from the inner part upon impact on bone. In such cases, the jacket and the core each produce separate tracks. With the semijacketed bullet, lead core usually exits, and the jacket remains in the body. Sometimes, a fragment of metal separates from the bullet during its passage through the skull. The separated fragment proceeds under the skin until it exits at a distance of 3 to 5 cm. or remains lodged between the skull and the skin. The major part of the bullet will enter the skull and may exit in a different position. In such a case, a single bullet will produce two exit wounds. In bullet wounds of the head, many lead fragments may be found scattered throughout the brain and on X-ray may closely resemble a shotgun injury.

(3) Bullet Striking the Skull but not entering it : Bullets may graze or rub the top and sides of the cranium without entering it. In such cases, entry and exit wounds are found on the scalp about two to three cm. apart, while the skull between the two wounds shows an oval or elongated gutter-like depression **(gutter wound).** When the bullet strikes the skull at an angle, it may rarely deviate from its path by impact on the bone, to produce a continuous wound track under the skin without penetration of the skull. The track may proceed for considerable distance, the bullet following the curvature of the skull.

(4) Multiple Wounds of Entrance and Exit from a Single Shot : A bullet may pass through an arm and the chest so that four wounds result. A bullet passing through the chest or abdomen and thigh and lower leg, produces six wounds. This occurs when the person is running or sitting in an unusual position. When the body surface is irregular, such as the breast or buttocks, several re-entries and exits can take place. In such cases, examination of clothing and detection of nitrites and nitrates and

microscopic examination of the sections of the wound is useful. The features of re-entry wound resemble those of wounds caused by long range fire.

(5) Entrance Wound is present but Bullet is not found in the Body : This occurs when (a) the bullet entering the stomach may be vomited, (b) entering the windpipe may be coughed up, (c) entering the mouth may be spit out, (d) entering the gastrointestinal tract may be passed out in the faeces, and (e) when it is so deviated or turned on coming in contact with the bone, that it passes out by the same wound as it entered.

(6) Unexplained Bullets in the Body : Occasionally, more bullets are found than there are entrance wounds. This occurs, due to defect in the weapon, or due to faulty ammunition, or with loaded firearm unused for several years. When such a weapon is fired, the bullet may fail to come out from the muzzle. When it is fired again, the second bullet may go off carrying the lodged bullet with it, and both the bullets may enter the body through the same entrance wound. This is called a **tandem bullet or piggyback bullet** (tandem=one behind the other) (Fig. 8–55) The bullets may separate within the body, or before they hit the target. The features caused by flame, smoke and gunpowder may be diminished or absent and the wound may appear as if caused by long-range fire. This is because the pushing force of the second bullet is directed backwards due to obstruction caused by first bullet impacted in the barrel.

Souvenir Bullets : If a bullet is present for a long time in the body, there will be no fresh bleeding in the surrounding area. A dense fibrous tissue capsule usually surrounds it. A small scar indicates the original entrance wound. Lead poisoning may occur due to absorption of lead from lead bullets remaining in a body. Synovial fluid is capable of dissolving lead.

(7) Fatalities with Blank Cartridges : A blank cartridge is one containing primer, gunpowder and wadding, but without a bullet or pellets. It contains ultrafast burning powder that explodes rather than burns. Distant shots with blank cartridge are harmless. Wounds can be caused from the gases or from the closing part in the end of the cartridge. Wadding or gunpowder may cause laceration and may produce death from shock by pressure on nerves or by damaging main vessels, when blank cartridge is discharged close to the body.

(8) Firearm going off by itself : The firearm can go off by itself without any one touching the trigger due to some defect in its mechanism. Country-made guns and old worn guns may discharge on being pushed or thrown, or by the butt being dropped against the ground.

(9) Frangible Bullets: They are designed to fragment upon impact, often to the point of disintegration. They are made mostly by lead or iron. Recovery and matching with a test bullet is difficult. If bone is penetrated, they are usually recovered in an eroded state. They do not ricochet.

(10) In some bullets, the component parts tend to separate on impact. Such bullets are partially jacketed at the base, with the tip remaining an uncovered lead core. The components separate on striking the body and create their own tracks.

(11) Duplex or Tandem Cartridge: This is used in military rifles, and contains two bullets which enter the target at different points, separated by as much as 30 cm. The base of the forward bullet is notched, into which the second bullet fits closely. The base of the 'follow' bullet is not quite at right angle to its long axis, due to which there is a difference in line of movement of both the bullets.

(12) Artefacts : (A) Surgical alteration or suturing of gunshot wounds create problems. The evaluation of the wound, whether it was an entrance or an exit wound becomes difficult **(Kennedy phenomenon).**

(13) Bullet Embolism : Bullets which enter a large blood vessel become emboli.

Concealed Firearm Wounds : If the body is covered with blood, the blood clots may obscure the injury. A wound in the mouth, nostril, ear, eye, or in any of the body orifices, e.g. rectum or vagina may not be detected, if due care is not taken.

SCENE OF CRIME : Before any items of evidence



Fig. (8–55) Tandem bullet in the chest.

are moved, photographs must be taken from different angles to show respective positions of various items of evidence, including the victim. Photographs should also be taken after various objects are moved, for cartridge cases or bullets may be found under the victim's body, or they may be hidden by pieces of furniture, etc. Bullet holes in the walls, floor, ceiling or in the furniture should be photographed. Before undressing, the body should be photographed. After the clothes are removed, entrance and exit bullet holes should be photographed with identifying labels and rulers. The bullets, pellets, and wadding found in the body should be phtographed. All areas likely to bear suspect's fingerprints, such as door knobs, glassware and the weapon should be examined for fingerprints.

EVIDENCE FROM SCENE : Collect : (1) The gun. (2) Fired bullets. (3) Empty cartridge cases, shells and wads. (4) Hairs, fibres and bloodstains. (5) Objects struck by or containing spent bullets, e.g. wood, cement, etc. (6) Glass shattered by bullets. (7) Areas showing fingerprints and footprints.

EVIDENCE FROM SUSPECT: Collect: (1) Clothes with trace evidence. (2) Victim's hair, clothing, fibres and blood. (3) Gunpowder and other evidence on the hands. (4) Gun used in the crime. (5) Unspent ammunition and empty cartridges or shells.

AUTOPSY: (A) CLOTHING: Remove clothing layer by layer. List them all, and note their condition and the extent of blood-staining. Record the number and location of bullet holes. It is sufficient to describe the number of bullet holes in the outer garment and adding that bullet holes in the remaining clothes correspond in location to the outer garment. The location of bullet holes in clothes should be described in relation to the distance from collar, seams, pockets, heel level, etc. A single bullet may produce several holes due to the presence of folds in the garment, and simulate more than one shot. The holes in the clothing should be connected to those in the body to determine the direction of fire. The clothes should be dried. If damp clothes are packed, shrinkage may occur and position of the hole of the entry of the missile altered. Examination of the clothes help: (1) to establish the range at which the firearm was discharged, from the deposition of soot or powder tattooing, (2) to establish which defects are wound of entry or exit, (3) to locate the bullet. If no exit wound is present, either the clothing did not cover the area of the exit, or the bullet may be in clothing or fell away. The size of the bullet hole and the extent of soot and powder distribution should be measured and the density of tattooing noted. Note whether the fibres of the clothing are turned inwards or outwards. Clothing may be forced into the tissues in shotgun wounds. Probes, fingers, etc. should not be introduced through the defects in the clothing, as the direction or distribution of fibres will be changed and cause confusion in deciding the entrance and exit of a bullet.

Number the entrance and exit wounds. In the case of multiple wounds, it is advisable to give a number to each wound, disregarding whether it was caused by entry or exit of a bullet. The clothes should be preserved carefully in clean brown paper or plastic bags and sent to Forensic Science Laboratory for testing. The powder grains adherent to clothing should be carefully removed with forceps and preserved in a glass vial, as they may be lost from the clothes due to rough handling. Clothes should be folded in such a way, that the area of bullet holes and gunpowder soiling are not disturbed or contaminated. Infrared photography can be used to find out soot deposit on dark coloured or black fabrics. Ordinary X-ray can be used to search for larger metallic fragments for elemental content.

(B) BULLET WOUNDS: (1) If there are multiple wounds, they should be numbered. On the body diagrams, the wounds should be drawn as they appear on the body including burning, blackening, tattooing, abrasion collar, etc. Photographs of the wounds should be taken. Bullet wounds must be described with care. (1) (a) the exact location of each wound should be noted in relation to its distance from the top of the head or the sole of the foot, (b) midline of the body, (c) a fixed anatomical landmark, e.g., "in the chest, 50 cm. below the top of the head, 8 cm. to the left of the midline and 2 cm. above the nipple". The size and the exact appearance of the wound is not preserved after excision due to the cutting of the elastic fibres.

(2) EXTERNAL WOUND: (a) The character of the perforation, its shape (stellate, round, slit-like or irregular), and size should be noted. The hole in the skin should be carefully measured first, and then the abrasion collar and the powder pattern surrounding the borders of the entrance wound. Difference in the width of the abrasion collar at different points should be noted as they indicate the angle at which the bullet struck the skin. A circular wound requires only one diameter of measurements, e.g., the skin perforation is round and measured 8 mm. in width. It is surrounded by a uniform rim abrasion 2 mm. in width. An elliptical wound is measured across its widest and narrowest diameters and variations in width of abrasion rcorded, e.g., the skin perforation measured 8 mm. by 3 mm. with the larger diameter running horizontally. A rim abrasion measuring from 0.5 mm. to 2 mm. surrounds the skin perforation with the wider rea

located at its lateral side. (b) The presence or absence of blackening and tattooing should be specifically noted, e.g., blackening and tattooing are absent, or the skin perforation is surrounded by an area of blackening and tattooing measuring 8 cm. by 5 cm. with the larger diameter running vertically. (c) Skin splits, contiguous and non-contiguous. (d) Muzzle imprint. (e) Soot deposit, including the corona. (f) Metal deposition. (3) Alteration by medical care personnel. A scaled photograph or a diagram showing the numbered wounds is useful. It is advisable to record the wound in the skin and the wound track through the body in one section. If the entrance wound is soiled with blood, it should be sponged carefully so that any tattooing of the skin may not be disturbed. After the wound has been examined, the skin around the entrance and exit wounds should be cut out including at least 2.5 cm. of the skin around and 5 mm. beneath the wound. They should be packed separately in rectified spirit, labelled and sent to the Forensic Science Laboratory.

If surgical wounds are made on a shotgun or stabbing victim, the surgeon should make adequate documentation of their location and nature in his records or on the hospital chart, so as to prevent confusion, if the patient dies and an autopsy is performed.

(C) TRACK TAKEN BY THE BULLET THROUGH THE BODY: Bullet tracks should be numbered and described individually. Probes should not be introduced through the track. The path taken by the bullet through the body should be carefully traced by dissection with the organs in situ. It is useful to measure the height of both entrance and exit wounds from the undersurface of the heel. This shows the direction of the track, and also the height above the ground at which the bullet entered and left the body, if the person was in standing position when struck. The bullet track should be described in relation to the planes of the body: (1) from front to back or from back to front, (2) from left to right or from right to left, (3) from above downwards (caudad), or from below upwards (cephalad). Angular estimtes, i.e., vertical, horizontal and sagittal planes of the body are also useful to complete the description. To release the bullet from the bone without actually handling it, the segment of bone containing the bullet should be excised, or sawed, followed by manual bending. Frequently, the track of the bullet is unpredictable due to its deflection by bone, and the bullet may be found in a most unexpected situation. This point is very important when the question arises in the case of two wounds being found on the body not corresponding to the likely line of the track, as to whether they have been produced by one or two bullets. The bullet may take a straight path through the body, and in this case the relative positions of the entrance and exit wounds will indicate the direction of fire. When a cavity has been penetrated and blood has collected, the bullet should be searched in the effused blood. A high velocity bullet is rarely deflected. To avoid prolonged search for the bullet in the body, X-ray examination should be done before autopsy. Rib borders or the border of other bones must be examined for roughening or fracture, which may explain the deflection of bullet. If there are multiple tracks, each should be followed from the point of entrance to termination. The track made by the bullet widens as it goes deeper. If a bullet grazes a bone, it may produce a gutter with or without fracturing it. If a bullet passes through the bone, a track of tiny radio-opaque metallic particles removed from the surface of the bullet will be seen in X-ray. The size of pellets is difficult to measure after the shot is fired as it becomes deformed. All bullets and recognisable parts of bullets in the victim must be recovered, and described as to where it was found, whether it is intact, deformed or fragmented, whether the bullet is lead or jacketed, etc. Next to bone, the skin offers the greatest resistance to the penetration of a bullet. A bullet passing through the body may come to rest just underneath the skin on the opposite side. The type of missile and the point of recovery should be noted. The location and character of exit wounds should be noted. All wadding in the body should be recovered to know gauge of the shotgun and the type of ammunition.

When a rapidly travelling object is slowed by passing from a thin to a dense medium, there is a release of kinetic energy which may be so violent as to fracture bones in the immediate vicinity of the track although the bones are not actually struck. Shock waves will also pass through the tissues causing injury remote from the actual wound. When a high velocity missile passes through soft tissue it is followed by cavitation due to the released energy. This primary cavity then collapses, and is followed by lesser secondary cavitation, which creates a negative pressure and debris is sucked into the track. When a high velocity missile strikes a relatively solid internal organ, such as the heart, this organ may disintegrate as if an explosive charge has been detonated in its substance.

Part of the energy of the bullet is spent by the tip to break the resistance of the tissues, and another part to push the tissues aside. This radial displacement producing transient cavitation is important with regard to the severity of tissue damage. If almost all the energy of the bullet is lost during penetration of the scalp, bone and dura, it passes through the brain without producing cavitation until it comes to rest. If

the bullet passes through the brain with great velocity, the wound track expands immediately after the bullet has passed through. This expansion develops in few microseconds, during which time brain is suddenly pressed against the inner table of the skull and other firm structures, and momentarily bulges out of entrance and exit wounds and collapses equally fast. The track of damage may be 4 to 5 times the diameter of the bullet, as seen by a track of haemorrhagic pulped brain tissue. . Prior to cavitation of brain, skull fractures result. If the fractures extend beyond the area of entrance and exit and the cavitation is very severe, the skull may burst by the pressure of the brain. A large portion of the brain may be thrown out of the bursting skull and found relatively intact. This is known as kronlein shot. If such explosion does not occur but cavitation is severe, the sudden pressure on the tissues surrounding the wound track causes immediate necrosis due to shearing forces acting on submicroscopic structures of cells. A ring of tissue haemorrhage is found outside the necrotic tissue. Contusions are also produced at a distance from the wound track due to displacement of portions of the brain due to the sudden space-occupying entrance of the bullet into the brain and by the associated cavitation. They occur independently of the location. Small calibre, low velocity bullets produce cavitation but fail to perforate the head on the opposite side. Sometimes, the entire track or one segment of it is found several times larger than the bullet. This occurs when the bullet passes sideways through the tissues or when it tumbles.

Bullets may be found in or on bodies either due to being fired from a gun or having been discharged from the cartridge as a result of heat-induced explosion. An exploded bullet does not show land-and-groove markings and the cartridge case is devoid of firing-pin impression. Intense fire causes melting of ammunition and leaves a pool of molten lead which may stick to the body as tiny droplets. X-ray shows "birdshot' pattern in films.

DESCRIPTION OF A FIREARM WOUND : There is a gunshot wound of entrance in the left anterior thoracic wall. This is just above and immediately medial to the left nipple and is situated 40 cm. below the top of the head and 10 cm. to the left of the midline. The wound is circular in shape, 0.8 cm. in diameter, and is made up of a 0.2 cm. radial dimensional abrasion ring concentrically placed about a 0.4 cm. diameter defect. There is no soot deposit or powder tattooing. A small amount of blood comes out from the defect upon manipulation of the body. A track is established from the gunshot wound of entrance on the left anterior thoracic wall, passes through the third interspace, through the lingua of the left lung, through seventh thoracic vertebra. Here, embedded in the bone, is recovered a full metal jacketed bullet of 0.25 calibre. This is marked 'X' and retained. A left haemothorax of 1200 ml. of fluid and clotted blood is present.

PRESERVATION, MARKING AND PACKING OF EXHIBITS : All bullets recovered from the body must be preserved with correct labelling of the relationship of each bullet to the corresponding wound. This is important in case of dacoity or rioting, to know which of the several weapons may have fired the fatal shot. It is important to state from which portion of the body, or from which internal organ it was removed. When more than one bullet or other foreign object has been removed from the body of a victim, or found in or about his clothing, each one should be labelled and placed in separate envelope. Care must be taken in removing a bullet from the body so that marks due to artefact, such as, scratches are not produced on the bullet. Such markings may make difficult subsequent identification of the bullet. It is preferable to remove the bullets with bare fingers. A forceps protected with rubber tubing may be used. The appearance of the bullet should be described accurately, e.g., intact, deformed, fragmented, lead or jacketed and the calibre if known. The bullet should be weighed, and if its base is not deformed, the diameter of the base should be measured. The recovered bullet should be dried and not washed, because washing removes the powder residue. In shotgun injury, the pathologist need not recover every pellet present. A few pellets should be recovered for the ballistic expert to determine the shot size and possibly type of ammunition.

(1) FIREARMS: Identifying initials should be scratched on to gun's frame, receiver, or slide and on the gun barrel.

(2) FIRED CARTRIDGE CASES : The identification mark should be scratched on the inside of the open end. They may be wrapped in cotton and packed in cardboard boxes.

(3) FIRED BULLETS : The identification marks should be scratched on the base, or just above the riflings on the ogive but not on the end of the nose for the nose may pick up trace evidence, e.g., the pattern of the weave of fabric through which it has passed. It is wrapped in cotton and packed in cardboard box. Each bullet should be packed separately.

(4) PELLETS, SLUGS, WADS, ETC : They may be packed in cardboard box with cotton after drying and the container labelled.

(5) CLOTHES : The area of the powder tattooing should be preserved by fastening a cellophane paper over it, and packed in a box.

MEDICO-LEGAL QUESTIONS

(1) Is the injury caused by discharge of firearm ?

Firearm wounds are recognised by the appearance of clothing and body entrance and exit wounds, the track of the bullet, and the presence of bullet or pellets and residual matter in the clothing or around entrance wound and in the tissues.

(2) What kind of weapon fired the shot?

The kind of firearm can be determined by the size, shape and composition of the bullet, and examination of cartridge, shots and wad left in the body or found at the scene of the crime and the appearances of wounds. With the shotgun, the appearances of the wound are characteristic. In muzzle-loading gun, the wad consists of plug of paper or cloth; in breech-loading gun of circular discs of felt or cardboard, from which the bore of the gun can be determined. Stains on the clothes or skin may show whether black or smokeless powder was used, and microscopic and chemical examination of the stain is helpful in finding out the particular brand of powder. Evidence of recent fire can be made out for a few days, by examination of the weapon for mercury vapour. Spent cartridge contains residues of primer and detonator.

In firearm examination, the primary principles of identification are (1) determination of calibre and type; (2) number of rifling grooves; (3) width of rifling grooves; (4) direction (left twist or right twist) of rifling grooves; (5) pitch (angle of the spiral) of rifling. These are used to determine whether a bullet could have been fired in a type or model of a gun or specific weapon. The bullets expand into the grooves in the rifling, sealing the barrel and prevent excess escape of gases ahead of the bullet. The firearms leave their signature on the cartridge case and on the bullet. With all rifled firearms, the bullet is slightly larger than the barrel, and as it passes through the barrel, its sides are marked by the rifling of the barrel **(primary markings; class characteristics)** and cause the bullet to rotate. Class characteristics (bar codes) are determined before manufacture of the gun and result from manufacturing specifications, design and dimensions. The class characteristics in fired bullet identifications would be (1) calibre, (2) number of lands and grooves, (3) direction of twist of the rifling, (4) rate of the twist of the rifling, and (5) width of the lands and grooves. They are most useful in identifying the make and model of gun involved. The surface of the bullet is also grooved by irregularities on the inner surface of the barrel itself **(secondary markings; individual or accidental characteristics),** which are specific for that particular weapon. These irregularities are produced by the sticking of the particles of the bullet to the bore when shots are fired and is known as **'metallic fouling'.** Individual characteristics also result accidentally during manufacturing process, are usually microscopic in nature, and have random distribution. They are useful to identify one specific gun to the exclusion of all others **"bullet fingerprinting".** The bullet found in the body known as **crime bullet or exhibit bullet** is compared under a comparison microscope, with one fired from the suspected weapon known as **test bullet.** The suspected weapon is fired, using the same brand and type of ammunition into a roll of wool, a bag of rags, a sand bag, oiled saw-dust, blocks of ice, water tanks, or against white blotting paper. Fresh pork skins, cleanly shaven are ideal for comparison with patterns on human skin. From the fired cartridge case, the calibre and type of cartridge and its manufacturer can be determined, from the shape and size of the case and the stampings on the case head. Fired cartridge cases also may be identified with the firearm from which they were discharged by a study of the marks left on them by the weapon. They are: (1) The firing imparts its general shape, size and tooling imperfections, striking the primer in centrefire cartridges, or case rim in rimfire cartridge. (2) The internal pressure of firing, forces the soft brass head of the cartridge case against the breech face, which produces markings. (3) The extractor usually marks the rim of a cartridge case while withdrawing the cartridge from the chamber, but may also make marks as it slips over and engages the cartridge rim or at the time of disengaging the rim. The end of the extractor hook may produce markings against the case wall next to the head. (4) The ejector strikes the rim or head of cartridge sharply during ejection and usually leaves a mark. (5) The magazine or clip may leave both class and individual markings. Lips of a magazine usually mark the rim edges of cartridges as they are stripped from it and chambered. (6) Imperfections at the edge or mouth of the chamber will also mark the cartridge case as it is moved into and out of the chamber in loading and extraction.

(3) From what distance and direction was the shot fired? The range of fire is determined by the presence or absence of the marks of smoke, flame, tattooing, etc., on or in the body of the victim. When the range is greater, it can be determined only approximately and with difficulty, from the nature of wounds and penetration. Test fire with suspect weapon using the same ammunition is useful for estimating the range.

The direction of the track depends upon the posture of the body at the time of impact. The direction of fire may be determined from the position of entrance and exit wounds and the track, bearing in mind the possibility of deflection of bullet and the different relationships of the parts of the body in movement.

Sections of the skin and subcutaneous tissues taken from transcutaneous portions of bullet wound may provide evidence as to direction and range of fire. Carbonisation, desiccation or recognition of particles of debris embedded in epidermis near the bullet hole may indicate close range of fire and may distinguish entrance and exit sites. The findings of debris along the track and in laceration adjacent to it suggest fire at contact range. The range from which the weapon was fired can be estimated by lead deposits around the entry wound. Swelling and homogenisation of dermal collagen caused by heat of bullet, is more marked at the entrance than the exit site.

(4) If multiple wounds of entrance and exit are present, could they have been produced by a single bullet? This has been already described.

(5) If multiple wounds are present, were they produced from the same or different weapons? This is determined by examination of the wound and of the bullet, cartridge, shots, wad, etc.

(6) When was the firearm discharged ? Tissue reaction to firearm injury is similar as for other types of injury. If black powder has been used, hydrogen sulphide may persist in the barrel for a few hours if breech is closed. The washing from barrels having discharged gunpowder are alkaline, contain nitrite, sulphate and thiosulphate. Smokeless powder leaves a dark grey deposit in the barrel of a recently discharged firearm. It forms a neutral solution with distilled water and contains nitrites and nitrates, but no sulphides. The mixture of gases of explosion has a peculiar smell, which can be noticed for several hours after the discharge of a gun.

(i) PARAFFIN TEST OR DERMAL NITRATE TEST: It detects gun powder residue (nitrates and nitries). Melted paraffin is brushed on the surface of the hand. The wax is removed and inner surface of wax cast is treated with diphenylamine or diphenylbenzidine reagent. A blue colour develops where residue is present. It is absolete.

(ii) HARRISON AND GILROY TEST : This test is not specific for firearm discharges residues, but only for certain elements or compounds to be found in such residues. This detects the presence of antimony, barium and lead. A cotton swab moistened with molar hydrochloric acid is used.

(iii) Neutron Activation Analysis: This chemical method is useful in identifying minute traces of elements present in the hair, nails, soil, glass pieces, paints, gunshot residues, drugs, etc. This is helpful when samples for comparison are available. It is based on the detection and measurement of characteristic radioisotopes formed by irradiation in a nuclear reactor. The atoms of the elements present in a specimen are bombarded with neutrons. Some of the nuclei of the atoms capture neutrons. Those atoms capturing neutrons become radioactive. The radioactivity is measured with a detector connected to an analyser. In firearm discharges, the residue from the suspect's hand is removed for testing either by the ordinary paraffin cast or simply by swabbing the back of the hand with cotton-tipped swabs, moistened with 5% nitric acid. Antimony, and copper from the primer are the components of residue which are detected.

(IV) ATOMIC ABSORPTION SPECTROSCOPY (AAS) AND FLAMELESS ATOMIC ABSORPTION SPECTROSCOPY (FAAS): They are suitable for measurement of antimony, barium and copper in gunshot residues. This analytical system utilises high temperatures to vapourise the metallic elements of the primer residues and to detect and quantitate them. NAA and AAS can aid in (1) identifying holes in clothing, tissues, wood, etc. as bullet holes, from the presence of lead, antimony, barium and copper, (2) determining range of fire from concentration pattern of antimony around the bullet hole, (3) determining common origin of bullet fragments of shotgun pellets found at different places, from the concentrations of lead, antimony, arsenic, copper and silver in these alloys, and (4) determining from the presence of lead, antimony and barium on hands whether or not a person has fired a gun.

(V) SCANNING ELECTRON MICROSCOPE-ENERGY DISPERSIVE X-RAY ANALYSIS (SEM-EDXA): It is the most sophisticated tool which can detect most minute traces of gunshot residue (GSR) found on the body of suspect. As a gun is fired, the GSR comprising chemical substances that burn and produce gases providing the velocity for the bullet, and metals such as antimony, barium, copper, etc. are also sprayed out and get deposited on the hands, clothes and even on the face of the person. The investigator can conclusively prove if the weapon was used by the suspect with a negligible margin of error.

(7) How long did the victim survive? It depends on the cause of death, i.e., whether from shock and haemorrhage, injury to a vital organ or septic complications.

(8) How much activity could the victim perform following the injury ? This varies considerably depending on the site of injury and the organ involved. If the bullet destroys motor area, brain stem or cervical cord or if a gaping laceration of the heart or aorta is produced, the victim becomes immediately incapacitated. Death is instantaneous if medulla is involved. Sometimes, through-and - through bullet wounds of the brain or heart do not cause immediate disability and the person may be able to carry out voluntary acts. Wounds of the auricles are most rapidly fatal; wounds of the right ventricle come next and the wounds of the left ventricle are the least rapidly fatal. The amount and rapidity of blood loss will also help to form an opinion about the extent of physical activity that would be possible. In any injury to other parts of the body, the victim may be able to walk about.

(9) Is it a case of homicide, suicide or accident? In deciding the status of a particular case, all the available information should be obtained, including autopsy findings, the investigation of scene of death and the circumstances of the shooting. Each case will present its own problems. (Table 8–13).

(A) The position and direction of the wound: These are very important. For suicide the sites of election are: (1) temple (about 60%), (2) centre of forehead, (3) roof of mouth, (4) midline behind the chin, and (5) left side or front of chest.

A suicide using a revolver or pistol, usually shoots himself in the right temple region, the bullet passing almost horizontally or upwards and backwards through the head, and making its exit in the left parietal region. If the individual is left-handed, left temporal region is selected. Occasionally, a right-handed person holds the weapon with the butt projecting backwards and the thumb on the trigger, in which case the direction may be different. Sometimes, the gun is put into the mouth, nose, ear or on the face or undersurface of the chin, and fired upward into the brain. In some cases, the suicide interposes his hand between the pistol and his head, shooting himself through the palm of the hand. Suicidal wounds of the chest and abdomen are less common. On the chest, the gun is fired into the cardiac region in a backward direction with a slight deviation upwards or downwards either to the right or left. On the abdomen, the gun is fired into the epigastric region and produces perforations and lacerations of the upper abdominal viscera.

In rifle and shotgun, the butt is usually supported by ground and the weapon is fired by hand into the head, or the butt is supported against a wall and fired into the chest or abdomen. The person may attach some string or cord to the trigger and tie the loose end to his foot, lie down and discharge the weapon through the roof of his mouth. He may tie the cord to some firm structure and pull the barrel towards him, thus firing the weapon or he may press the trigger with a stick or some similar object. The presence of blood splashes on the hand which held the gun or the presence of an impression of the trigger or trigger guard on one or more fingers indicates suicide.

In homicide, a great variety of wounds can occur depending upon the circumstances. If there is a scuffle, some of the wounds may be from close range and the bullets may strike the body in various places and at different angles. If the victim runs, most of the entrance wounds will be on the back. If victim rushes at the assailant, the entrance wounds are on front of the body. If the assailant is in a panic or under strong emotion, a number of shots may miss the target or graze the skin. Wounds about the sides and limbs are suggestive of murder, if accident is excluded, and wounds about the back, and back of head are strongly suggestive of murder. Premeditated, calculated homicide by shooting can occur in a variety of ways. In gang feuds, the victim may be surprised and shot by enemies at close range into the back of the head and neck, with the bullet tracks parallel or only slightly deviating.

(B) The nature of the entrance wounds: This is very important. A contact wound is possibly

suicidal, unless found on a part of the body which is not easily reached by the deceased, e.g., back of the chest or neck. Suicides usually pull the clothes aside to expose the skin before shooting themselves. Bullets vertically entering fatal parts like heart or head, indicate suicide. Several methods **(combined suicides)** or several attempts of committing suicide e.g., hanging and shooting indicate suicide. A close or distant shot is rarely suicidal.

(C) The number of wounds : Multiple wounds are usually homicidal especially if they occur on the back or sides of the body or involve different regions of the body or are non-contact shots. Occasionally, multiple suicidal wounds are seen involving a single region like the temple, chest or abdomen or a combination of two different regions. When multiple suicidal wounds are fired serially, the injuries by the first shot do not incapacitate the victim immediately, enabling him to fire other bullets into his body. A bullet passing through the brain, causes immediate unconsciousness, but if bone is involved without injury to the brain or a large vessel, the person may retain consciousness and power of motion. A bullet passing through the chest or abdominal viscera, in some cases does not incapacitate the victim and he may be able to shoot himself again.

(D) The position of the weapon : In suicide and accident, the weapon is usually found at the scene of the crime; the gun may bear the victim's fingerprints. The hand that fired the shot may show evidence of gunsmoke, powder deposits and traces of metals. With handguns, the effects of the components of a shot are commonly seen on the thumb, index finger and the area between them. Blood splashes may soil the gun and the hand, if the hand pulling the trigger is close to the wound. In cases of murder,weapon is usually not found near the victim. If the weapon is strongly grasped in the hand of the victim due to instantaneous rigor, it is a strong presumptive evidence of suicide. In some cases of homicide, the scene may be altered to simulate suicide in order to conceal the crime. The assailant after producing a contact wound on the anterior surface of the body or an accessible part of head, may place the weapon in the hand of the deceased. In such cases, the thumb is usually found under and not around the gun handle, and the weapon is not tightly grasped. If the gun has been tampered with after shooting, e.g., removing of empty cartridge and substituting of unexploded cartridge, causes confusion. The absence of weapon is not conclusive of murder, for it may be removed by a person passing the place or in some fatal cases, the deceased is able to walk considerable distance, leaving the weapon behind.

(E) Motive : When a person commits suicide, he usually has a motive, and he may leave a note or make preparations to kill himself.

(F) The Scene : Photographs of the scene, the body and the gun should be taken. Fingerprints on the weapon, doors, etc., are useful. If the gun is present at the scene, its position in relation to the body, its make, model, calibre, type of action and description of the ammunition should be noted. Any loose bullets found should be collected. If they are embedded in the walls, ceiling or furniture, they should be extracted and retained. The place in which the body is found, e.g., in a room in his own house, the state of the room, whether showing signs of struggle or not, the condition of the doors and

Table (8–13) Difference between suicidal, accidental and homicidal firearm wounds

Trait	Suicide	Accident	Homicide
(1) Site of entrance wound:	Head or heart.	Any area.	Any area.
(2) Shot distance:	Contact or very close range.	Close or very close range.	Any range.
(3) Direction:	Upward or backward.	Any direction.	Usually upward.
(4) Number of wounds:	Usually one.	One.	One to many.
(5) Hand pressing trigger:	Powder residue present.	Powder residue present.	Powder residue absent.
(6) Position of the weapon:	Found at the scene	Found at the scene.	Not found at the scene.
(7) Scene:	Usually in his own house.	In his house or while hunting, etc.	Any place.
(8) Sex:	Usually males.	Usually males.	Any sex.
(9) Motive:	Insanity, incurable illness, financial loss, etc.	Nil .	Gang feuds, robbery, revenge, etc.

windows whether locked or not, should be noted. If the body is found in the open, a search for footmarks and marks of struggle must be made.

(G) Sex: It is very rare for females to commit suicide by shooting, and rare for persons who are not used to firearms.

(H) Disposal of the body : If the body is removed from the place of shooting or if an attempt is made to dispose off the body, commission of crime becomes obvious.

Accidental Wounds : They are comparatively rare, and usually single. Most of the accidents are caused by carelessness or misuse. The technical reasons for accident are: (a) the construction of the weapon, (b) the absence of safety device, (c) the poor quality of the material used. Accidents occur when loaded weapons are handled carelessly or carried without safety catch, or when the victim slips while examining, loading or cleaning the weapon. The wounds are found on the front of the body, and frequently directed upwards. Sometimes, the victim is shot unintentionally by a person who is ignorant of firearms or careless in their use. Occasionally, when the police shoot on law breakers , an innocent bystander is struck by bullets.

Hunting accidents may result from (a) swinging the weapon through the line of hunters, (b) ricochetting bullets or pellets, (c) failing to unload the weapon when jumping over ditches, climbing fences or crawling through them, (d) failing to unload or uncock the weapon when it is carried or laid down. In case of a discharge caused by falling weapon, (a) the weapon or its muzzle will be some distance away from the body, and there cannot be a direct contact shot, (b) the weapon will show signs of force to allow a discharge, (c) marks will be found on the ground where the weapon dropped.

Faked Firearm Wounds : Firearm wounds are very rarely voluntarily inflicted for the purpose of attributing murder. They involve non-vital parts and are near wounds.

Identification by Flash : It is possible to distinguish features with the help of a discharge from a gun or pistol up to a distance of about 8 metres, but the details of features of clothing cannot be made out. Thus, if the assailant is well-known to the victim, identification is possible.

BOMB EXPLOSION WOUNDS

A bomb is a container filled with an explosive mixture and missiles, which is fired either by detonator or a fuse. Terrorist bombs often involve only 2 to 10 kg. of explosives. When an explosion occurs, the explosive material produces a large volume of gas, and releases a large amount of energy. Pressure of up to 1,000 tons per sq. inch. can be generated. A minimum pressure of about 700 kilopascals (100 lb/sq inch) is necessary for tissue damage in humans. A person can be injured by an explosion in a number of ways.

(1) Disruptive Effects : If the victim is almost in contact with a large bomb he may be blown to pieces, e.g., when the victim is carrying it. The pieces can be scattered over an area of 200 metres radius. Many parts of the body are never found, having been disrupted into tiny fragments and mixed with the masonry and other debris of the bomb site. A bomb exploding on the ground may cause severe damage or traumatic amputation of the lower legs. A bomb which explodes when the victim is bending over it, may cause severe damage to arms, face and front of the chest. When the victim is a few metres away or with smaller explosion, disruption is usually limited to mutilation of a localised area.

(2) Burns : The temperature of the explosive gases can exceed 2000°C., and the radiated heat can cause flash burns. It burns nearby objects and clothing. The flame causes extensive burns, which involve irregular area of skin to a different degree. Tight clothing protects, so that beneath collars, bras, waist bands, socks and shoes the skin may be quite normal.

(3) Air Blast : An explosion produces a 'shock wave' which spreads concentrically from the site of explosion at about the speed of sound. This wave of very high pressure is followed by a weak wave of negative pressure (below atmosphere), a suction which lasts about five times as long. A shock wave exceeding 700 kilopascals (100 lb/sq. inch) pressure is necessary to cause serious damage to a body. The shock wave can throw the victim against a wall or toss him through the air causing blunt force injuries. The clothes may be blown off by the blast. The clothing should be retained for chemical analysis. The shock wave passes through the body. The homogeneous tissues like liver and muscle are not damaged. Lungs show subpleural patchy haemorrhages, often in the line of ribs, rupture of alveolar septa, intrapulmonary haemorrhages and bullae at the margins. Desquamated alveolar and bronchial epithelium is seen lying free. This causes

reactive pulmonary oedema and blood-stained froth is found in the air-passages, and later bronchopneumonia. This specific pulmonary injury of air blast is called **'blast lung'.** The tympanic membrane may rupture with haemorrhage in the ear. Subperitoneal haemorrhage and laceration of abdominal organs may occur. Intracranial haemorrhage, contusion of the brain, injuries of heart and aorta, pneumothorax, ruptured stomach and bowel may occur. Death may occur from systemic air embolism, from air which enters the pulmonary veins after blast damage to the lungs. When the explosion is in the water, the pressure changes are called **underwater blast,** and when it forces a solid material such as the deck of a ship against the body, the effect is called **solid blast.** Gastrointestinal damage is more common than lung damage. In some cases, death may occur without any external injury.

(4) Flying Missiles : The blast may drive multiple fragments of bomb or pieces of nearby objects, e.g., gravel, glass, wood, brick, plaster, etc. through the air into the skin and cause bruises, abrasions and puncture lacerations intimately mixed on the skin. This triad of injury is diagnostic. Most of the bruises and abrasions are less than one cm. in diameter, although they tend to unite (fig. 8-56). The puncture-lacerations are also usually of this size. They are ragged, sometimes with soiled margins, and may contain foreign material, such as scraps of clothing, wood or metal. The skin can be darkened by an explosion which drives dust into the skin and causes fairly uniform tattooing. The force of the bomb explosion is extremely directional and the pattern of injury might indicate that the person was carrying the bomb or bending over it or sitting to one side of it.

(5) Falling Masonry : When a building is destroyed by a bomb blast, the persons inside sustain multiple injuries and die of traumatic asphyxia.

(6) Fumes : If a bomb explodes in a confined space, enough CO is produced to cause asphyxia. In a victim of bomb death, X-rays of tissues should be taken, as pieces of metal especially the detonating mechanism may be seen.

Incendiary bombs, e.g., napalm bombs primarily cause burns. A temperature of about 1000° C is produced. In incendiary bombs, usually phosphorus and magnesium are added. The **Molotov cocktail** is an incendiary bomb which is thrown by the hand.

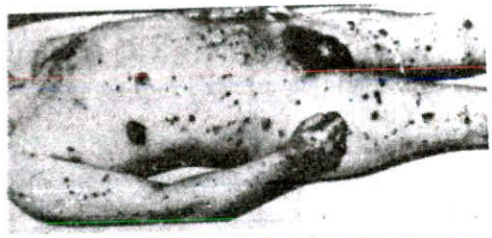


Fig. (8–56). Bomb explosion injuries showing contusions, abrasions, puncture lacerations and tattooing.

In a crude type of this bomb, a bottle is filled with gasoline and a rag to serve as a wick. The wick is lighted and thrown at the target. Various acids and chemicals are sometimes added to increase the destructive effects.

INVESTIGATION OF BOMBING INCIDENTS:

(1) Whether the explosion was caused by a bomb ?

Dispersed explosion usually occurs in houses when domestic gas leaks into the atmosphere and mixes with the air to form an explosive mixture that then catches fire. A dispersed explosion can blow off clothing and burn the exposed skin. All the exposed parts of the body are affected and all the burns are of same depth.

A bomb explosion (localised explosion) never causes extensive burns. Injuries by blast force, and the fragments are seen on the body. A part of a limb is blown off or a localised area of the body is mutilated. The triad of bruises, abrasions and puncture-lacerations with tattooing of part of the body also indicate bomb explosion.

(2) Identification of the dead.

(3) Number of dead persons.

(4) CAUSE OF DEATH : Death may result from multiple injuries, burns, shock or cerebral air embolism.

(5) CIRCUMSTANCES OF DEATH : To differentiate the terrorist from the peaceful citizen, the reconstruction of the scene and the circumstances of the deaths from the autopsy findings are helpful. The injuries on the victims, particularly their type, severity and distribution are of much help.

(a) EXPLOSIVE FORCE DECLINES RAPIDLY : Injury due to the explosive force itself is likely within a few metres of the bomb. With a terrorist bomb, when a person is blown to pieces he must have been in contact with it, i.e., either carrying the bomb, sitting with it or arming it. Persons can be injured by flying fragments and collapsing structures when at considerable distance from a terrorist bomb.

(b) EXPLOSIVE FORCE IS HIGHLY

DIRECTIONAL : The parts of the body directly exposed to explosive force only are injured. An explosion at ground level injures mainly lower legs and feet. When a person is in front of a bomb when it blows up, the face, chest, forearms, hands, inner thighs and the legs below the knees are injured, but the back of the body, the lower legs and face escape. If a person is bending down over the bomb, the face, chest, lower legs and hands are severely damaged. The legs may be blown off or the abdomen disrupted or the hands and arms torn away in the person who was planting the bomb. If the thigh, pelvic region and abdomen are damaged, the bomb may have been carried on the lap. If a parcel bomb is opened by a person sitting at a desk, his face, chest and hands are injured. If a bomb explodes behind a person sitting in a chair, injuries will be produced on the back of the legs below the knee and on the back of the trunk.

All bodies should be X-rayed before autopsy to identify any radio-opaque objects including those from the bomb mechanism, such as small springs or contacts from the timer or detonator.

DOMESTIC GAS EXPLOSIONS: They occur due to leaks in the gas supply. Natural gas explodes at concentrations in air between 5 to 15 percent. Ignition is usually caused by matches or candles, fires, sparking electric switches. The explosion is accompanied by a momentary flame that sets fire to furnishings. The air pressure usually rises to less than 10 psi, due to venting through broken windows and doors. Deaths are rare. The person can be thrown off their feet and injured. The damage is haphazard or diffuse. The flame singes hair and causes localised superficial burns of exposed surfaces. Most deaths occur when the building collapses and the victims are buried in the debris.

MECHANICAL EXPLOSION: Mechanical explosion occurs when a steam boiler bursts due to increased pressure. Heat and large volumes of gas are produced. The effects are similar to those of chemical explosion.

CHAPTER 9
REGIONAL INJURIES

HEAD INJURIES

Head injury is a morbid state, resulting from gross or subtle structural changes in the scalp, skull, and/or the contents of the skull, produced by mechanical forces. The application of blunt force to the head may result in injury to the contents of the skull, either alone or with a fracture of the skull. The extent and degree of an injury to the skull and its contents is not necessarily proportional to the amount of force applied to the head. Any type of craniocerebral injury can be caused by any kind of blow or any sort of head.

SCALP: The thickness of the scalp in the adult is variable, ranging from a few mm to about one cm. Most wounds are caused by blunt force to the head, e.g., from falls or blows, and such wounds are contusions or lacerations. Contusions may occur in the superficial fascia, in the temporalis muscles, or in the loose areolar tissue between the galea aponeurotica and the pericranium. Contusions in the superficial fascia appear as localised swellings and are limited in size because of the dense fibro-fatty tissue of the fascia. A haematoma of the scalp may be very extensive and spread beneath the galea over most of the skull. (subgaleal haemorrhage). A temporal bruise may later appear behind the ear, suggesting primary neck injury. Deeper bruising in relation to the fibrous galea beneath the skin becomes visible when the scalp has been dissected and reflected. When the vault of the skull is fractured, blood may extravasate into the scalp tissues from the ruptured diploic veins. Bruises of the scalp are associated with prominent oedema. The veins of the scalp and face are connected with the parasagittal, lateral and cavernous sinuses through emissary veins which pass through the foramina in the skull. Infected wounds of the scalp and face may be complicated by thrombophlebitis, which may extend to the intracranial sinuses. Bruising of the scalp is better felt than seen. Multiple contusions of the scalp may join together, and as such, it is often difficult to determine the number of blows inflicted. Its firm edge often feels like the edge of a depressed fracture of the skull. Lacerations of the scalp resemble incised wounds. An oblique blow usually causes a large wound and direct blow a small wound. If the scalp is lacerated by a blow, blood will be driven out of the vessels due to compression, and considerable bleeding will occur subsequently, but it will not be projected around the scene, If further blows are struck, blood will be projected about the scene under considerable pressure from the already lacerated vessels, and blood and tissue may adhere to the weapon and thrown in the direction of the uplift of the weapon. With repeated blows, blood will be splattered over the assailant, which will indicate the relative positions of the victim and assailant, and also whether they were inflicted with the right or left hand. Temporal arteries spurt freely, as they are firmly bound and unable to contract, and a fatal blood loss can occur from an extensive scalp laceration. Avulsion of a large area of scalp can occur in traffic accident, or if hair becomes entangled in machinery. Even after death, scalp injuries may bleed profusely, especially if the head is in a dependent position.

In case of homicide with head trauma, always retain a sample of victim's head hair, pulled from an area adjacent to the area of trauma.

FACE : EYES : Bleeding is more in facial wounds. A blow on the eye with a blunt weapon may cause a permanent injury to the cornea, iris, lens, or vitreous haemorrhage or detachment or rupture of the retina and traumatic cataract. The eyes may be gouged out with the fingers.

BLACK EYE (PERIORBITAL BRUISING) : It is caused by (1) direct blow in the front of the orbit, bruising lids, (2) injury to the forehead, the blood tracking down under the scalp, (3) fracture base of the skull in the anterior fossa, the blood leaking through cracked orbital plates.

NOSE : The nose may be bitten or cut off due to sexual jealousy or enemity. A blow on the head may cause nose-bleeding due to partial detachment of mucous membrane without any injury to the nose.

EARS : A blow over the ears may produce rupture of the tympanum and deafness. The labyrinth may also be injured.

FACIAL BONES : A blow with a blunt weapon or fist often fractures the nasal bones, and also ethmoid bone with radiating fractures into the supra-orbital plates, if the force is severe. A blow with a blunt weapon may cause fracture of the maxilla and malar bones. Pulping of the face may result from striking with a heavy stone. The mandible is fractured by a

blow from a fist, stick or by a fall from a height. A heavy blow on the jaw drives the condyles against the base of the skull, producing a fissured fracture. Very rarely, the condyles may be driven through the base of the skull.

TEETH : A fall or a blow with a blunt weapon may cause fracture or dislocation of teeth, with contusions or lacerations on the lips or gums and bleeding from the socket. X-ray of the jaw may show fracture of the alveolar margin at the site of dental injury.

SKULL : The outer table is twice the thickness of inner. In young males, the thickness of frontal and parietal bones is 6 to 10 mm. The temporal bone is thinnest, 4 mm. Skull is thicker in the midfrontal, midoccipital, parietosphenoid, and parietopetrous buttresses. Outer table is thicker than the brittle inner table.

Fractures : Fractures may be caused by direct or indirect violence. Direct injuries may be caused by: (1) Compression as by midwifery forceps or crushing of the head under the wheel of a vehicle. (2) An object in motion striking the head, e.g., bullets, bricks, masonry, machinery, dagger, etc. (3) Head in motion striking an object, as in falls and traffic injuries. Indirect injury of the skull occurs from a fall on the feet or buttocks. Fractures of the vault occur from direct violence.

Under experimental conditions, a force of 400 to 600 pounds per square inch is required to fracture a cadaver skull covered by an intact, hair-bearing scalp, but 25 inch-pounds energy is suffficient to fracture empty human skulls lacking normal soft tissue coverings. The fracture may be simple or compound, i.e., associated with the injury to the scalp or nasal sinuses. There is no relation between the damage to the brain and linear fractures of the skull. Skull fractures can occur without any significant or detectable brain injury or any impairment of consciousness. Conversely, severe or fatal brain injury may occur without fracture of skull. A thin skull with a fracture may produce less brain damage than a thick skull without it.

MECHANISM OF FRACTURE OF SKULL:

(1) FRACTURES DUE TO LOCAL DEFORMATION: If a small mass travelling at sufficiently great speed strikes the head squarely, it will drive inwards a piece of bone, shaped as cone-like indentation. At the apex of such a cone, the inner table will be stretched, but the outer table will be compressed, due to which the inner table fractures first. If the force continues to act, fracture of the outer table follows, and the completed fracture line runs from the central point radially. At the periphery of the indentation, the bone is bent in the opposite direction, and as the convexity of the bend is outwards, the outer table fractures first. The fracture lines tend to run circularly to enclose the base of the indentation. If the injuring force is not lost, a piece of bone fragmented by the radial fracture lines will be loosened and then depressed to form a comminuted fracture.

(2) FRACTURES DUE TO GENERAL DEFORMATION: Whenever the skull is compressed, e.g., laterally, there is a shortening in the line of pressure, while the vertical and longitudinal diameters are increased, due to which parts of the skull distant from the side of application of the injuring force are bulged and may fracture by bending. The head may be compressed: (1) Between two external objects, such as the ground and a wheel of a car. (2) Between an external object and spinal column. The latter method is common and is seen in motor car accidents when an occupant thrown from his seat, strikes the head against resistant surface. It is also seen when the body is at rest, and a heavy object falls on the top of the head, driving the skull downwards on to the condyles of the atlas.

Fractures due to local deformation are commonly associated with those due to general deformation.

Types of Fracture Skull: (1) Fissured Fractures: They are produced by general deformation of the skull. These are linear fractures involving the whole thickness of the bone or inner or outer table only. Fig (9-1&2). Majority of skull fractures are linear. The outer table is capable of rebounding to its normal shape, while the more brittle inner table fractures. It may be present alone or associated with

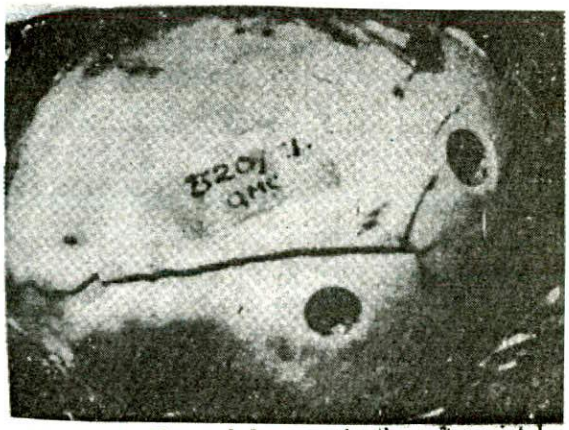


Fig. (9-1), Fissured fracture in the left parietal and occipital bones and two burr holes.

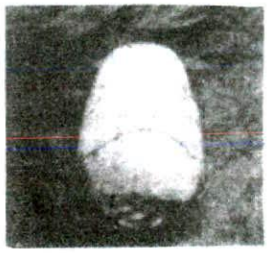


Fig. (9–2), Fissured fracture of vault of skull.

other types. They are likely to be caused by forcible contact with a broad resisting surface like the ground, blows with an agent having a relatively broad striking surface, or from a fall on the feet or buttocks. When a blow is struck on the side and the head is free to move, the fracture starts at the point of impact and runs parallel to the direction of the force. If the head is supported when struck, the fracture may start at the counter-pressure, e.g., in bilateral compression, the fracture often starts at the vertex or more commonly at the base. In both cases, the line of the fracture runs parallel with the axis of compression. A fall on the flat surface may show one or several fracture lines radiating from the point of impact, but the depression of bone fragments is not seen. An extensive haematoma of the scalp will indicate the site of impact. In general, an injury of the head sustained by a fall is mostly situated in the level of the margin of the hat, while an injury due to a blow is commonly situated above this level.

A fracture line tends to follow an irregular course and is usually no more than hair's breadth. Sometimes, a fracture line reaches a suture and follows its course for a while, and then starts again on its own way. The fracture line is often red, but usually there is no haemorrhage. At autopsy, blood can be allowed to run over the suspected fracture which is then washed. Blood will then mark out a fine fracture, which might otherwise be missed. Linear fractures do not tend to cross bony buttresses, such as glabella, frontal and parietal eminence, petrous temporal bone, and occipital protuberance. They tend to cross points of weakness, such as frontal sinuses, orbital roof, parietal and occipital squama. From the site and direction of the blow on the scalp, one can predict the likely fracture line. A blow over the glabella may cause shattering of the ethmoid sinuses and roof of the nose. In the case of the blow and subsequent fall, the fracture lines produced from a fall are arrested by those produced by the blow. If two blows are struck, the same results are seen and it may be possible to determine which of the blow was first struck.

Anterior blows to the skull produce linear fractures of the orbit, often bilateral and the fractures cross the middle through the cribriform plate, and pass posteriorly through the pituitary fossa into the basal portion of the occipital bone. Blows to the anterior parietal bone produce fracture of the anterior temporal area, and with severe violence, linear fractures running towards the vertex and the anterior and middle fossae. Interparietal blows produce linear fractures running towards the base, involving the lesser wing of the sphenoid and pituitary fossa. The pituitary and hypothalamus may be damaged (micro-haemorrhages, ischaemic necrosis, avulsion) by blows to the side of the head causing fracture of the middle fossa. With posterior parietal blows, the fracture lines often cross the petrous temporal bone and extend towards the inner ends of the middle fossa into the posterior fossa. Blows on the occipital region produce fractures which extend towards the foramen magnum and often tend to run forwards to the lateral side of the occipital buttress and the foramen itself and may cross the petrous temporal bone and middle fossa. There may be fragmentation of one or both of the orbital plates. Blows high on the vertex tend to produce fractures which cross the midline, sometimes following suture lines. About 20 per cent of linear fractures are not found on X-ray.

The human adult head weighs about 4.5 kg. Fractures of the dry skull (i.e. without coverings or contents) can be produced by 3.3 foot-pounds. In the cadaver head about 35 foot-pounds are required to produce a linear fracture of skull. A linear fracture of skull may be produced when the head is hit on the side by a stone weighing 90 to 120 g. and when a weight of 2.25 kg is dropped through a distances of one foot.

The first blow to the skull will weaken the structure and subsequent blows may cause a degree of damage out of proportion to the force used, e.g. a second blow on an aea of skull already fractured, may cause widespread collapse of the skull in that area, and the fragments of bone and the weapon itself may be driven far into the brain.

In persons having abnormally thin skull, mino violence may cause a fatal fracture.

(2) Depressed Fractures: They are produced by local deformation of the skull. In this, fractured bone is driven inwards into the skull cavity. The

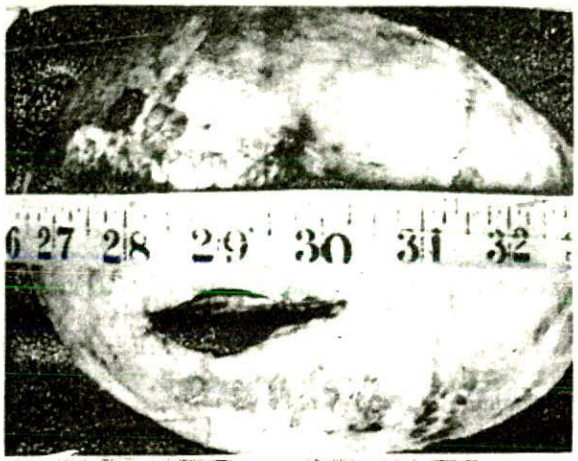


Fig. (9–3). Depressed fracture skull.

outer table is driven into the diploe, the inner table is fractured irregularly and to a greater extent and may be comminuted. They are also called "fractures a la signature" (signature fractures), as their pattern often resembles the weapon or agent which caused it. Localised depressed fractures are caused by blows from heavy weapon with a small striking surface, e.g., stone, stick, axe, chopper, hammer, etc. Depth varies according to the velocity with which the impact is delivered. Rarely only the inner table may be fractured under the site of impact, leaving intact the outer table. Sometimes, the depressed fracture may involve the outer table only. A violent blow with the full face of a weapon completely detaches almost the same diameter of the bone, which is driven inwards. A less violent blow, or an oblique blow, may produce a localised fracture with only partial depression of the bone. When a hammer is used, the fracture is circular or an arc of a circle, having the same diameter as the striking surface,

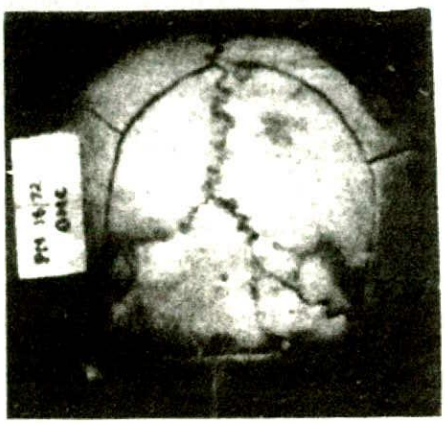


Fig. (9–4) Comminuted fracture skull.



Fig. (9–5) Extensive comminuted fracture of the skull.



Fig. (9–5) Pond fracture.

and usually there are no radiating linear fractures. The part of the skull which is first struck shows maximum depression. When the butt of a firearm strikes the skull full faced, the fracture is rectangular, but if only the corner of the butt strikes, the fracture is triangular. Depressed fracture caused by a stone may be irregular or roughly triangular. Ante-mortem fractures in case of homicide often appear as localised depression of the skull. They show a shelving inward of the outer table of the bone along the edges of the fracture.

(3) Comminuted Fractures: In a comminuted fracture there are two or more intersecting lines of fracture which divide the bone into three or more fragments. Fig(9-4). They are caused by vehicle accidents, fall from a height on a hard surface, and from blows by weapons with a large striking surface, e.g., heavy iron bar or poker, an axe, thick stick, etc. They may also result from a kick by an animal or by a bullet. It is often a complication of fissured or depressed fracture. When there is no displacement of the fragment, it resembles a spider's web or mosaic. Fissured fractures may radiate for varying distances from the area of comminution (fragmentation). When the force is great, the broken pieces of bone are displaced and some may enter the brain and others may be lost.

(4) Pond or Indented Fractures: This is a simple dent of the skull, which results from an obstetric forceps blade, a blow from a blunt object or forcible impact against some protruding object. Fig (9-5). They occur only in skulls of infants. The inner table is not fractured, but fissured fractures may occur in the outer table around the periphery of the dent. The dura mater is not torn and usually the brain is not damaged. In an infant a blow often produces only a dent like that seen in a Ping-Pong ball.

(5) Gutter Fractures: They are formed when

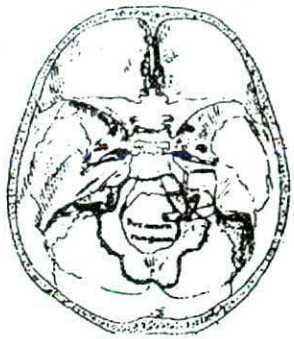


Fig. (9–6) Ring fracture.

part of the thickness of the bone is removed so as to form a gutter, e.g., in oblique bullet wounds. They are usually accompanied by irregular, depressed fracture of the inner table of the skull. The dura mater and brain may be torn.

(6) Ring or Foramen Fractures: It is fissured fracture which encircles the skull in such a manner that the anterior third is separated at its junction with the middle and posterior third. But usually the term is applied to a fracture, which runs at about 3 to 5 cm. outside the foramen magnum at the back and sides of the skull, and passes forwards through the middle ears and roof of the nose, due to which the skull is separated from the spine. Fig(9-6). They are rare and occur after falls from a height on to the feet or buttocks. In very severe cases, the spine may be driven into the skull cavity, and the vault of the skull may burst open (explosive type), giving the impression of an original massive fracture. A severe blow to the vertex may also cause a ring fracture, but in this case the fracture of the vault will be depressed. A forceful blow on the chin in traffic accident may produce ring fracture. There may be laceration of skin but mandible is not fractured in most cases. They are thought to be produced from forces transmitted into the back of the skull through the mandibular joints. They are also caused by a sudden violent turn of the head on the spine, shearing the vault from the base.

(7) Perforating Fractures: These are caused by firearms and pointed sharp weapons like daggers or knives and axe. The weapon passes through both tables of the skull leaving more or less a clean-cut opening, the size and shape of which corresponds to the cross-section of the weapon used.

(8) Diastatic or Sutural Fractures : Separation of the sutures occur only in young persons, due to a blow on head with blunt weapon. It may occur alone but often is associated with fracture.

FRACTURE BASE OF SKULL : Basal fractures may be produced by : (1) Force applied directly at the level of the base. (2) General deformation of the skull wherever the forces are applied. (3) Extension from the vault. (4) Force applied to the base through the spinal column or face. In the base, fracture patterns are strongly influenced by the petrous buttress. Fracture lines which approach it from the middle or posterior fossa are turned either towards its apex or base, according to the angle at which they strike it. The middle of the body of the petrous bone is fractured only when the force is very great. Most basal fractures tend to meet at and overrun the pituitary fossa. Isolated fracture of the base is often due to transmitted force, either from the point of the chin or through the spine, except following blows in the temporal region. Fracture lines usually open into the basal foramina. The sphenoidal fissure is most commonly affected. The foramen magnum is not spared inspite of its thickened margins.

Blows on the chin occasionally fracture the glenoid fossa. The force of a blow on the mandible, e.g., an upper cut in boxing, may be transmitted through the maxilla and its internal angular processes to the base of the skull and cause a fracture of the cribriform plate of the ethmoid. An oblique blow of great force applied to one side of the back of the head, will start a fracture in the underlying posterior fossa, which crosses the middle line to enter the middle fossa of the opposite side, and may end in the anterior fossa. Longitudinally or transversely directed forces always produce fractures in the corresponding axis. A fall on the back of the head or blow on the top of the head usually produces fractures of the roof of the orbits, especially in old people. These fractures may be comminuted and sometimes depressed. These fractures are supposed to be produced from the contrecoup of the orbital lobes of the brain on these paper-thin orbital plates. Sudden violent increase in internal pressure also produces fractures of roofs of orbit, especially in suicidal gunshot wounds of the skull. Fractures of the base of the skull may be : (1) Longitudinal, which divide the base into two halves. This may result from a blunt impact on the face and forehead, on the back of the head, or in front-to-back or back-to-front compression of the head, e.g., run over by a vehicle. (2) Transverse, which divide the base into a front and back half. This may result from an impact on either side of the head or side-to-side compression of the head as when run over by a vehicle. (3) Ring fractures. Basal fractures are usually associated with cranial nerve damage.

COMPLICATIONS: **(1) Fractures of anterior cranial fossa may involve the frontal, ethmoidal or sphenoidal air sinuses, with loss of blood from nose and mouth. (2) If the dura and nasal mucosa are torn, CSF and even brain tissue can leak into the nose. (3) Leptomeningitis may result due to bacteria passing upwards from the nose. (4) Fractures involving paranasal sinuses may cause cranial pneumatocele. (5) Fractures of the middle fossa passing through the basiocciput or sphenoid bone may communicate with mouth, from which blood will run. (6) A direct communication between the cavity and the airway via the sphenoid sinus is produced in fracture of base of the skull involving the sella turcica. Blood may pass into the bronchial tree. Foci of inhaled blood are commonly seen in the lungs which indicate that death was not instantaneous. (7) A fracture of the petrous temporal bone may involve the middle ear, which allows blood and CSF to escape from the ear. The blood may pass into the mouth through the Eustachian tube, and may be swallowed. Tear of the posterior branch of the middle meningeal artery as it crosses a fracture of the temporal bone produces severe bleeding from the ear. (8) In posterior fossa fractures, extravasation of blood is seen behind the mastoid process or a large haematoma in the soft tissues of the back of the neck. (9) If the fracture reaches foramen magnum, cerebellar contusions may result, and the subsequent oedema may herniate the cerebellar tonsils fatally through the foramen. Cranial nerves may be injured by stretching or bruising but they are usually not severed. (10) Damage to surrounding structures. (11) Shock. (12) Portal of entry for bacteria. (13) Fat and bone marrow embolism. (14) Depressed fractures of the skull pressing the brain may cause severe dysfunction, coma and death.**

Fracture of the skull occurring opposite to the site of force is known as **contrecoup fracture**. This usually occurs when the head is not supported. This is explained by the sudden disturbance in the fluid brain content which transmits the force received to the opposite side, where the thrust of violent motion impacts against the cranial wall, which is unable to absorb this degree of disturbance.

THE CIRCUMSTANCES OF FRACTURE OF THE SKULL: **Most of the fractures are due to an accident, e.g., a fall, or an injury by a motor vehicle. Multiple, localised and depressed fractures suggest homicide. Suicide by head injury is rare, because it is painful and cannot be produced easily. The victim is usually insane. It may be attempted by hitting the head against a wall or by driving a nail, etc., into the skull. Suicidal blunt impact injuries are grouped close to midline and directed to the top of the head.**

Age of Skull Injury : Healing occurs without the formation of visible callus. The periosteal blood vessels are damaged, delaying the development of external callus. The edges of fissured fracture stick together within a week. The edges are slightly eroded and the inner surface of the skull may show pitting or deposition of lime salts in 14 days. The edges become slightly smooth and bands of osseous tissue run across the fissure in 3 to 5 weeks. When the edges are not in apposition, they become quite smooth in three months, or partially healed if close together. If there is much loss of bone, the gap is filled only with fibrous tissue.

INJURIES OF BRAIN AND MENINGES: The intracerebral lesions are divided according to the state of dura. If the dura is lacerated, e.g., by a bullet or any other object, such as fragment of bone, it is called open injury because it is open to infection. The brain is also lacerated. If the dura remains intact, it is called a closed injury, whether the skull is fractured or not. Blunt force to the head with a non-penetrating object or from a fall, or the head striking a flat surface or a firm object produce closed injuries. The mechanical force which produces a brain injury, usually produces injury of the scalp, fractures of skull and intracranial injuries. However, a fatal brain injury may be caused without any damage to the scalp or skull. Contusions are produced in the brain.

THE OCCURRENCE OF BRAIN INJURY: **Brain injuries may be caused by: (1) Penetration by a foreign object, such as knife, bullet, etc. or fragments of skull in a depressed fracture. (2) By distortion of the skull. When a localised segment of the skull undergoes deformation, shear strains may develop in the brain tissue underlying the indentation, and a contusion may be produced in the surface layers of the brain tissue. If a fracture occurs, pieces of bone may penetrate the dura and lacerate the brain.**

(3)ACCELERATION/DECELERATION INJURIES: **Acceleration or deceleration injuries are due to sudden movement of the head due to injury, which produces intracranial pressure gradients and subject the brain to both shearing and tensile forces.**

An impacting force to the head can produce linear acceleration, rotational (angular) acceleration, or a combination of both. In linear acceleration, the force passes through the centre of the head, accelerating the head in a straight line. In rotational or angular

acceleration the force does not pass through the centre of the head and thus the head will rotate about its centre.

Impacts to the front and back of the head tend to produce linear acceleration while those to the side produce a combination of linear and angular. A combination of linear and angular acceleration of the head in the coronal plane, e.g. side impact is more injurious to the brain than similar acceleration in the sagittal plane (frontal impact). Subdural haemorrhage and diffuse axonal injury are produced.

The cranial cavity is divided into three compartments by the falx cerebri, and the tentorium cerebelli, which are relatively rigid structures and act in the same way as skull in resisting the movements of the brain.

MECHANISM OF CEREBRAL INJURY

Severe brain damage may be caused without actual blow or fall on the head, e.g. by shaking the infant as in child abuse may cause subdural haemorrhage. A blow to the head will cause linear change in velocity or a rotational change in velocity about same axis. The forces involved are linear acceleration (or deceleration) forces due to the change in linear velocity and centrifugal and rotational velocity. Linear acceleration forces, tend to produce compressional or rarefactional (sound waves) forces which start from this point and travel back and forth through the brain, but they do not cause any damage. The brain is easily distorted, but is incompressible. Change in velocity, either acceleration or deceleration, with a rotational element causes brain damage. In either deceleration or acceleration of the head, the initial sudden change in velocity would set the head in rotation and this rotation would be transmitted to the brain which would glide within the dura. The area of the skull beneath an impact becomes momentarily depressed even if it does not fracture, and may strike on the underlying brain causing compression, and typical cone-shaped contusions are produced on the cortex, with the base at the surface. Other areas of the skull bulge outward simultaneously to accommodate the deformation. Gliding or shear strains are produced by the angular rotation of the head, which move adjacent strata of tissue laterally. The sudden arrest of the moving skull causes the deceleration of the skull first, but the momentum of the brain will cause it to continue in motion. In either deceleration or acceleration, the skull and brain cannot change their velocities simultaneously, and the brain will slow down or speed up only due to the restraint by the falx and tentorium, causing damage to the base of the cerebrum, corpus callosum and brain stem. Impact against the wide wall of the skull may cause diffuse contusion of the cortex. The shearing is more severe where the gliding is prevented by bony prominences, especially those of the anterior and middle fossae. As the cerebellum is smaller and lighter than the cerebral hemispheres, it is less liable to damage from rotatory movements of the head.

A blow to the side of the head may damage the medial surface of cerebral hemisphere on that side, especially in the region of the cingulate gyrus, because the soft brain is pushed violently against the free edge of the falx. Shearing strains caused by unequal rotational gliding and twisting of hemisphere may produce multiple haemorrhages and much direct damage to the fibres of the corpus collosum. Holbourn postulates that brain tissue is injured when its constituent particles are pulled so far apart that they do not join up again properly when the blow is over. In the brain, the amount of this pulling apart is proportional to the shear strains.

Contusions of the Brain: Contusion and laceration of the brain are two degrees of the same process. When a localised segment of the skull undergoes deformation at the moment of impact, shear strains may develop in the brain tissue underlying the indentation, and a zone of contusion may be produced in the surface layers of the brain. When the head is rotated by an impact, the layers of brain tissue slide over each other at different depths in the cortex, which cause damage to the blood vessels. Contusion may occur on surface of cortex or deeper down. There is no actual tearing of the tissues. They may occur without injury to the skull, but often there is a fracture of the skull. The period of unconsciousness varies, but it usually lasts from 30 minutes to several days. Pia-arachnoid is intact over surface constusions, and is torn in lacerations.

CONTUSION HAEMORRHAGES : Cerebral contusions are circumscribed areas of brain tissue destruction, which are accompanied by extravasation of blood into the affected tissues. They are usually multiple and vary in size. They are produced by blunt force and are found in gray and white matter due to injury of blood vessels by mechanical stress. They present as streaks or groups of punctate haemorrhages. Contusions are most often found in the frontal and

temporal lobes of the brain, and less commonly on the lateral and ventral surfaces of the cerebral hemispheres. If the haemorrhage is close to the surface of the cortex, there may be overlying focal subarachnoid haemorrhage. Deeper structures, e.g., basal ganglia, midbrain and brain stem are contused especially from impacts to forehead and vertex. The medulla may be contused in association with fractures which extend into the foramen magnum or involve the atlas and axis. Most haemorrhages occur at the crest of convolutions facing the dura of falx and tentorium. The haemorrhages are densely arranged and often elongated, radially pointing towards the white matter. Haemorrhage is first seen in the perivascular spaces along the shrivelled and collapsed blood vessels. It spreads along the sulcus and often involves several convolutions. Rarely, contusion haemorrhages occur in the cortex facing a sulcus. Traumatic contusion haemorrhages at the crest are recognised by their columnar arrangement perpendicular to the surface of the convolution. A larger haematoma may be formed by their union especially in persons with hypertension or in alcoholics. Many contusions are only seen on section of brain.

Blows to the back of head, usually due to falling backwards result in little or no occipital contusion, although there may be posterior cerebellar contusion or subarachnoid haemorrhage. They usually produce subfrontal and temporal lobe contusion. Blows to the top of the head produce minimal coup contusion but prominent contrecoup subtemporal or uncal contusion and less commonly subcerebellar contusion or laceration, which even result in posterior fossa subdural haemorrhage. In serious impacts, corpus callosum is lacerated. Blows to the side of the head produce a lateral coup lesion and more prominent contrecoup contusion, or laceration of the lateral aspect of the opposite hemisphere. There may be contrecoup contusion in the deep cortex or sylvian fissure. Blows to the front of the head usually do not produce cerebral

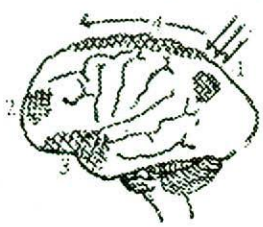


Fig. (9–7). Contusion and laceration of the brain. (1) Coup injury. (2) Contrecoup injury. (3) Dashboard injury in the region of the sphenoidal ridge. (4) Avulsion injury along the superior border of the hemisphere (the rotary vein).

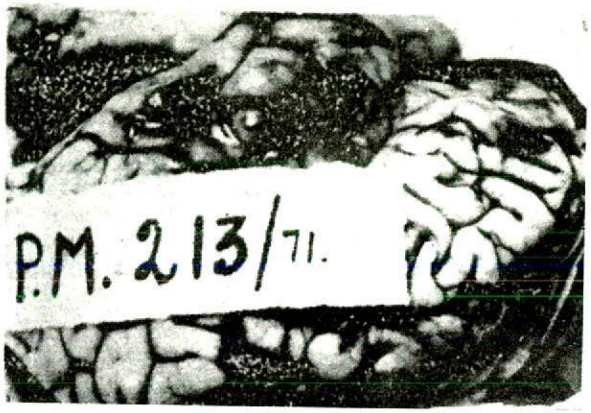


Fig. (9–8). Contusions of the brain.

contusion or laceration. There may be only diffuse subarachnoid haemorrhage, and contrecoup injuries to the occipital lobes are rare. In severe frontal injury, frontal coup laceration occurs.

AGE OF CONTUSIONS : Ischaemic changes occur in neurons in an hour. Capillary proliferation begins in 5 days and is maximum at 10 to 12 days. Macrophages containing fat are present in small number during the first two weeks. Astrocytic proliferation occurs in few weeks, and a scar is left in about two months, which may have pale or golden yellow colour and are depressed.

CONTUSION NECROSES : They are found at the crest of convolutions and form small clefts, irregularly shaped holes, or trenches with sharply outlined walls, and usually brown in colour. They communicate with subarachnoid space, and do not contain any blood vessels. On section they are triangular or wedge-shaped, the wedge pointing into the white matter. The margins are straight. The area of necrosis is usually delineated by haemorrhage. If the necrosis is purely ischaemic, without haemorrhage, the gross lesion is seen in 10 to 12 hours after the injury as an area of swollen gelatinous parenchyma. The age of the lesion can be made out by the cellular reaction in the marginal areas. The first layer of cortex is always destroyed over contusion necroses, except for short stumps. At the moment of impact, the crest of the convolution is flattened or depressed by striking against the dura. The deformation of the crest spreads from the point of impact to the periphery of the convolution. If the convolution is suddenly pushed against the stationary bone as in a fall, the same result will be seen. The laterally spreading deformation produces shearing forces within a wedge-like area. Necrosis is caused by the action of shearing forces on microscopic structures.

The combination of extensive contusion and an associated subdural haematoma is referred to as a

'burst' lobe, usually seen in forntal or temporal lobe and less commonly in a cerebellar hemisphere.

Lacerations : Cerebral lacerations are traumatic lesions in which there is loss of continuity (tearing) of the substance of brain. Surface lacerations are accompanied by ruptures of the pia mater and subarachnoid haemorrhages. They are usually surrounded by groups of contusions. When the parenchyma is completely disorganised, it is termed pulpefaction. These tears are caused by stretching and shearing forces within the tissues produced by blunt force. In infants up to five months of age they are common, as the skull is easily deformed and the brain very soft. Lacerations are usually seen underneath skull fractures. In depressed fractures, the bone fragments tear the brain surface and may be driven into it. All penetrating injuries produce lacerations of brain. Lacerations may also be produced without fracture of skull, when they are usually found in regions where the brain is in contact with projecting buttresses and ridges on the inner surface of the skull, e.g., the temporal poles and orbital surface of the frontal lobes. Blunt trauma to the head, without fracture of the skull lacerate the corpus callosum or septum pallucidum, especially in younger individnals.

In severe hyperextension of the head, as in motor vehicle accidents, lacerations may be produced in the pyramids at the junction of the medulla oblongata and pons or avulsion of the brain stem at the pontomedullary junction. These lesions are usually associated with fractures of the base of the skull and of the upper cervical vertebrae.These slit-like, sometimes irregularly shaped lacerations often contain very little blood and may be mistaken for artefacts. Adhesions may develop between the brain and dura mater due to healing of surface lacerations, which may cause secondary epilepsy. Healing of deep lacerations involving ventricles may produce large glial cysts filled with CSF (traumatic porencephalic cysts.)

CONTRECOUP LESIONS : Coup (blow, impact) means that the injury is located beneath the area of impact, and results directly by the impacting force. Contrecoup means that the lesion is present in an area opposite the side of impact. Holbourn (Oxford physicist) in 1943, demonstrated that contrecoup lesions are chiefly due to local distortion of the skull, and sudden rotation of the head resulting from blow, which cause shear strains due to the pulling apart of the constituent particles of the brain. Holbourn defines **shear strain** as "a strain produced by applied forces which cause or tend to cause adjoining parts of the body to slide relatively to each other in a direction parallel to their planes of contact". A certain amount of shear may occur below the point of impact, particularly if the skull is fractured, which accounts for the coup. A much greater shear strain develops as a result of the rotation of the skull, and because the changes in the rotational velocity are usually greater at the pole opposite to the point of impact, contrecoup injuries are more extensive. A line drawn between the centres of coup and contrecoup indicates the direction of impact relative to the head. Contrecoup injuries are not seen if the head is well fixed and cannot rotate.

Contrecoup injury is caused when the moving head is suddenly decelerated by hitting a firm surface, e.g., striking the head on the ground during a fall, usually seen in traffic accidents. Subdural or subarachnoid haemorrhage may be caused as a contrecoup lesion. The sudden arrest of the head results in the brain which is still in motion, striking the arrested skull. A blow to the head causes the skull to move forward, but the brain lags behind for a brief period and the skull strikes the brain (acceleration injury). Another factor responsible for contrecoup injury is formation of a cavity or vacuum in the cranial cavity on the opposite side of impact, as the brain lags behind the moving skull. The vacuum exerts a suction effect which damages the brain. Occipital injuries produce severe and extensive contrecoup lesions in the frontal region. The irregular bony prominences, particularly of the orbital and cribriform plates, and the lesser wings of the sphenoid, contuse or lacerate the base of the frontal lobes and the tips of the temporal lobes, sometimes with fracture of orbital plates. A blow at the front of the head may damage the inner and lower parts of the back of the brain by contact with the edges of the tentorium. This can also injure the brain stem and produce pontine haemorrhage. A fall on to the side of the head may cause a fracture of that side and contusion of the opposite side of the brain. Rarely, a contrecoup lesion may be seen on the opposite side of the same hemisphere, e.g., a blow on the left parietal area may cause contrecoup lesion on the medial side of the left cerebral hemisphere against the falx. A blow to the head produces coup contusions, while contrecoup contusions are either small or absent. A fall on the head produces

contrecoup contusions while coup contusions are small or absent. Contrecoup injuries are rare before the age of three years. Contrecoup injury is seen in skull, brain, liver, heart and lungs.

Lindenberg and Freytag introduced new names for the contusions in the brain which do not fit into coup or contrecoup. Contusions found in deeper structures of the brain, such as white matter, basal ganglia, corpus callosum and brain stem along the line of impact, i.e., between the coup and contrecoup points, are called INTERMEDIARY COUP CONTUSIONS. Contusions caused by fracture of the skull are called FRACTURE CONTUSIONS. Contusions in the cortex and white matter of the frontal and central convolutions near the upper margin of the hemispheres show no relationship to the area and direction of impact. They are called GLIDING CONTUSIONS and are caused by stretching and shearing forces occurring in the region of arachnoid granulations, during to and fro gliding of the brain within the skull in moderately severe impact. They are seen in falls and motor vehicle accidents. Contusions in the cerebellar tonsils and the medulla oblongata produced by momentary shifting of the brain toward the foramen magnum are called herniation contusions.

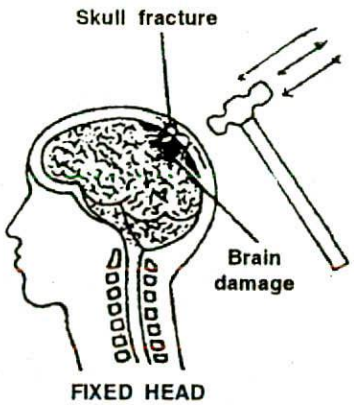


Fig. (9–9) Mechanism of coup injuries of the brain.

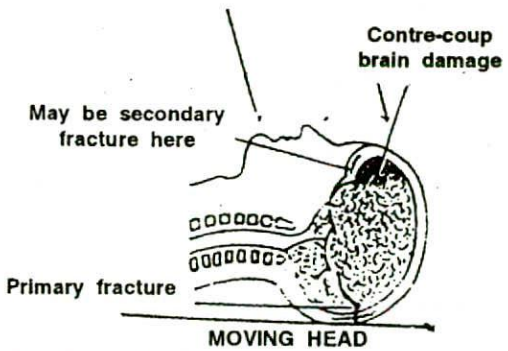
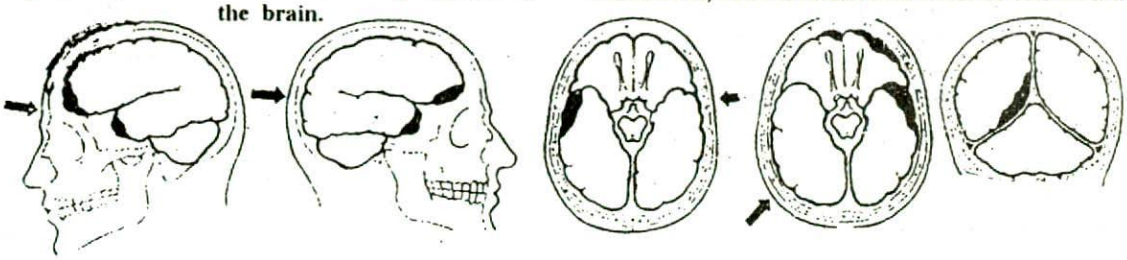


Fig. (9–10) Mechanism of contrecoup injuries of the brain.

CONCUSSION OF THE BRAIN

Concussion is a state of temporary unconsciousness (due to partial or complete paralysis of cerebral function), due to head injury, comes on immediately after injury, is always followed by amnesia, and tends to spontaneous recovery. Cerebral concussion occurs due to acceleration/deceleration of the head (the head freely moveable at some stage). The violent head movement causes shearing or stretching of the nerve fibres and axonal damage. Severe injuries occur in coronal head motion only. Sagittal head motion produces mild or moderate injury. At low levels of acceleration/deceleration, anatomical damage of the axons does not occur, but there is physiological dysfunction. The axon may recover completely or undergo degeneration. With increased physical force, there is immediate structural damage of axons, with immediate stoppage of all activities. In mild concussion, consciousness is not lost, and there is no confusion or disorientation, and amnesia may or may not be present. In severe concussion, there is amnesia and loss of consciousness



Coup contusions from falls on the frontal region. | **Contrecoup contusions from fall on the back of the head.** | **Contrecoup contusion from impact of head on opposite side.** | **Contrecoup injury from fall on the opposite side.** | **Diffuse contrecoup contusion from fall on parietal region.**

Fig. (9–11) Mechanism of coup and contrecoup injuries of the brain.

for less than six hours.

Diffuse axonal injury (DAI) is a clinical condition, in which there is diffuse injury of the axons with immediate loss of consciousness and coma of more than six hours. In mild DAI, there is coma for 6 to 24 hours. In moderate DAI, there is coma for more than 24 hours, but there are no clinical signs of brain stem dysfunction. In severe DAI, there is coma of more than 24 hours with brain stem signs. It occurs due to vehicle accidents in about 90% of cases, and due to falls and assaults in about 10% of cases.

During established concussion, the muscles are flaccid, pupils are dilated and unreacting, pulse is weak and slow, respiration is shallow. As consciousness finally returns, there is a period during which the person appears to be lucid and in touch with his surroundings , but in fact he is not. He behaves automatically and not rationally or responsibly. The patient has no recollection of accident or injury, although he can usually recall events up to or within a few minutes of the occurrence. A post-traumatic amnesia may range from minutes to days, and its duration is usually proportional to the severity of the injury. In some cases, amnesia after head injury may be permanent. If unconsciousness is prolonged, it is certainly due to obvious brain injury. Concussion can be ruled out if unconsciousness does not occur immediately after a blow, and if coma develops later, it is due to other pathology. Blows to the neck or cervicocranial junction produce brain stem concussion. With severe movement of the head, shearing stresses occur in the brain, which produce numerous small or punctate haemorrhages throughout the brain, "Commotio cerebri". There are many theories as to the cause of concussion: vasomotor disturbances, impaction of the brain into the foramen magnum or tentorial opening, but the most acceptable hypothesis is **"diffuse neuronal injury'**, a functional abnormality of the nerve cells and of their connections. Certainly, cerebral shift within the skull seems necessary to its production. Experimental evidence suggests that in concussion, direct damage occurs to the brain stem reticular formation. The nature of the interference with reticular formation function may cause damage to any part of the neurone; the cell body, axons or synapses and damage to synaptic junction between neurones. This damage may result from raised intracranial tension, brain stem deformation or shearing strain, but it commonly occurs with acceleration, or deceleration injuries than with injuries to the moveable head. Death may occur without the patient regaining consciousness, or he may recover partially and then die suddenly, from concussion of vital cerebral centres. In cases of recovery, post-concussion syndrome may follow with headache, dizziness, nausea, vomiting, insomnia and mental irritability. The victim may exhibit automatism. He may commit some violent or criminal act. Complete recovery takes place in less than ten days. Diffuse axonal injury occurs when head acceleration occurs over a long time period, as in traffic accidents and falls from a considerable height.

Autopsy may not show any change, but in some cases petechial haemorrhages may be found in the cortex, at the junction of the grey and white matter, in the roof of the fourth ventricle and under the pia mater of the upper segments of the cervical cord. Oedema, foci of myelin degeneration, etc., may be found. In mild DAI, some axons may be damaged. In severe DAI, there is shearing of axons in the white matter of the cerebral hemispheres, corpus callosum and upper brain stem, with focal haemorrhages in the corpus callosum and dorsolateral rostral brain stem. Microscopic examination does not show axonal injuries up to 12 hours after injury. After 12 hours, the axons first appear dilated, then club-shaped and finally appear as round balls known as **"retraction balls"**, which indicates transected axons. The number of retraction balls begins to decrease 2 to 3 weeks after injury, and clusters of microglial cells appear, followed by astrocytosis and demyelinisation.

FEATURES OF DIFFUSE AXONAL INJURY ARE: (1) A focal lesion in the corpus callosum and other midline structures involving the parasagittal white matter, the interventricular septum and the wall of the third ventricle with some intraventricular haemorrhage. (2) A focal lesion in one or both dorsolateral sectors of the rostral brainstem. (3) Microscopic evidence of numerous axonal swellings and axonal bulbs. In fatal head injury greater or lesser axonal injury is almost always found.

The interrogation of persons who have sustained head injuries: If a person becomes unconscious after head injury, he is likely to be mentally confused. He usually suffers from ammesia to a varying degree and may not have recollection of sustaining an injury. A person involved in a

traffic accident can walk around and talk to rescuers but may not, or only partially recollect the event. The police may have to interrogate such persons in regard to events preceding and following the injury. During interrogation the person should not be asked leading questions and no answer should be suggested. If such questions are asked, incorrect information may be obtained and the suggestions may become impressed on the mind of the person and he may have false memory in relation to the events. As such, the police should be advised to dealy interrogating an accused person or a complainant who has sustained a head injury, until he completely recovers from his initial confusion.

Amnesia Following Head Injuries: Amnesia following head injuries is quite common and is usually associated with concussion. The memory of distant events tends to return before the memory of more recent events. Permanent retrograde amnesia may vary from a period of seconds up to seven days. In a person recovering from concussion, events which occurred just before the injury are sometimes remembered indistinctly during the period of confusion, but there will be complete amnesia for these events after the return of complete consciousness. As such, the patient may make false accusations. Retrograde amnesia may also occur in injuries in which there is no loss of consciousness.

Post-traumatic Automatism: It is intimately associated with amnesia. After an accident, the patient may speak and act in purposive manner, but does not remember them afterwards.

Anterograde amnesia is characteristically seen in post-traumatic head injury.

Admission to hospital after head injury: A person who has sustained head injury, should be admitted in hospital for observation for at least 24 to 36 hours.

Head injury and acute alcoholic intoxication: A person may be confused and disorientated after a head injury which may simulate acute alcoholic intoxication. If an intoxicated person sustains a head injury, it may not be possible to assess to what degree his condition is due to head injury or to alcoholic intoxication. Such person should not be kept in police custody but should be admitted in a hospital for observation.

CASE : A slow moving car swerved into an intersection and over the curb onto the sidewalk. The right front door flew open, the driver was found sprawled across the seat in a pool of vomitus. The odour of alcohol was present and the driver was observed to be ataxic and incontinent, mumbling an incoherent story. A cursory examination resulted in a diagnosis of 'drunk', and he was sent to jail, where he was found dead the next morning. Autopsy revealed a cerebral haemorrhage, and no alcohol was present in the blood and only a mere trace in his urine. A chemical test of his breath or blood at the time he was examined would have indicated a low ethanol content, and other diagnoses might then have been considered.

Oedema of Brain and Swelling: In brain **Oedema of brain and swelling:** In brain swelling, oedema is mainly intracellular. The organ is enlarged and firm, but has a relatively dry cut surface. In oedema of brain, the fluid collection is interstitial. The organ is enlarged and soft and has a very watery cut surface. Swelling of the brain may occur following significant head injury, which may be focal, adjacent to an area of brain injury, diffuse involving both cerebral hemispheres, or involve one hemisphere only. Massive cerebral swelling can occur within 20 minutes following head injury. Swelling of one cerebral hemisphere is commonly seen in association with ipsilateral acute subdural haematoma. Increase in intravascular cerebral blood volume secondary to

Table (9–1). Difference between drunkenness and concussion.

	Trait	Drunk	Concussed
(1)	Face :	Suffused; flushed, warm.	Pale, clammy.
(2)	Pulse :	Fast, bounding.	Slow, feeble.
(3)	Pupils :	Contracted in coma, dilate on external stimuli and contract again. Reaction to light sluggish.	Contracted or unequal.
(4)	Breathing :	Sighs, puffs, eructates.	Shallow, irregular, slow.
(5)	Memory :	Confused.	Retrograde amnesia unrelieved by time.
(6)	Behaviour :	Un-co-operative, abusive, unresponsive, insolent, talkative.	Co-operative, quiet.

vasodilatation (congestive brain swelling) or an absolute increase in the water content of the brain tissue causes brain swelling. Cerebral oedema occurs due to increase in the water content of the brain. If brain swelling due to an increase in the intravascular cerebral blood volume is continued for some time, cerebral oedema will be produced due to inreased vascular permeability.

Brain swelling may not occur immediately after an injury, but may develop minutes to hours later. Delayed brain swelling of a significant degree is rare, which is usually diffuse and is seen in less severe forms of brain injury. With severe brain injury, diffuse brain swelling of a severe degree may occur immediately without the person regaining consciousness. It is asymmetrical if caused due to a mass in one side of the brain or subdural space, e.g. subdural haematoma or intracerebral haemorrhage.

Increased Intracranial Pressure: The causes are extradural and subdural haemorrhage, cerebral haemorrhage, infarction of brain, tumour or abscesses of the brain or its coverings, dural sinus thrombosis, leptomeningitis, diffuse cerebral oedema, etc.

CEREBRAL COMPRESSION : Any increase in the size of the brain, e.g., generalised swelling or space-occupying lesions within the cranial cavity, result in compression of the brain. As the brain is incompressible, the compression will diminish the amount of CSF in the subarachnoid space and in the ventricles. A continued rise in intracranial pressure leads to a progressive interference with the blood supply of the brain. If there is increase in intracranial pressure above the tentorium, the adjacent uncus or inner margin of the temporal lobe is squeezed down through the hiatus along the midbrain, either on one or both sides, due to which the midbrain is squeezed from side to side and lengthens anteroposteriorly. This stretches the paramedian and nigral vessels which rupture to produce haemorrhages in the midline and along the substantia nigra, which is fatal. Sometimes, there is haemorrhagic infarction of the medial cortex of one occipital lobe, due to the twisting of posterior cerebral artery around the edge of the tentorium by herniation. A rise of pressure below the tentorium forces portions of the cerebellar lobes and tonsils of the cerebrum through the foramen magnum, and the medulla oblongata is compressed, which causes progressive failure of respiration. DURET HAEMORRHAGES are secondary herniation haemorrhages of the midbrain and pons, ranging from small streaks to massive confluent haemorrhage in the midline. They commonly occur with asymmetrical herniation of the brain stem. Uncal grooving and foraminal indentation of the cerebellar tonsils are common post-mortem findings and must not be misinterpreted as evidence of uncinate herniation and cerebellar coning.

Unconsciousness occurring some time after the infliction of a head injury suggests cerebral compression. Immediate unconsciousness occurs due to concussion, then the person slowly recovers consciousness and again loses consciousness gradually due to compression. A so-called "LUCID INTERVAL" thus intervenes between the two stages of unconsciousness. In some cases, there is no lucid interval. In other cases, there is no initial gradual unconsciousness, but there is gradual unconsciousness from compression some time after the trauma. A large extradural haemorrhage can produce rapid compression within a few hours of the injury. A large subdural haemorrhage produces relative slow compression, because it takes several days for sufficient blood to accumulate in the subdural space to produce compression. Delayed death may occur after a head injury due to a chronic subdural haematoma.

The suggestive evidence of cerebral compression are : flattening of the gyri, narrowing of the sulci, apparent decrease of CSF; deep grooved marking around the uncus of a temporal lobe and cerebellar pressure cone.

When severe brain swelling (or a space-occupying lesion) causes raised intracranial pressure above the tentorium, compression of the midbrain against the free edge of the tentorium may occur. This may be unilateral causing grooving of a cerebral peduncle (called **Kernohan's notch**). When symmetrical the oedema forces the undersurface of the cerebrum against the tentorium so that the hippocampal gyrus is squeezed into the opening.

LOSS OF CONSCIOUSNESS : A mechanism exists in the reticular system of the brain stem, which stimulates arousal (consciousness), due to the incoming impulses derived from all parts of the body. Destruction of the reticular system leads to absence of arousal and to unconsciousness. Reduced afferent activity may interfere with consciousness. Normal sleep may result from reduction of stimuli and irresistible sleep due to exhaustion of enzyme system essential to maintain the function of reticular system. Similarly, toxic agent may affect the function.

BRAIN STEM : It may be injured by (1) Stretching of peduncles when the hemispheres shift. (2) Deceleration against basisphenoid and dorsum sellae. (3) Lateral shift of peduncle against tentorial margin.

(4) Stretch or avulsion from it of cranial nerves. (5) Traction on its vascular supply.

Spontaneous pontine haemorrhage is usually single, occupying from one third to half of substance of pons. Traumatic haemorrhage occurs in a number of separate foci in the pons, which may unite if the victim survives for a sufficient time. Both types can rupture into the fourth ventricle. Pinpoint pupils not reacting to light in case of head injury indicates pontine haemorrhage. Primary haemorrhages in the brain stem are usually small and are seen in relation to the walls of the third and fourth ventricles and of the aqueduct. Haemorrhages in the restral brain stem are usually more numerous and severe than those into the medulla in rapidly fatal injuries. In most persons who die after prolonged unconsciousness, brain stem injuries are seen.

Cause of Death in Head Injuries : Most deaths are due to damage to vital cerebral areas, located around the posterior hypothalamus, midbrain and medulla. Usually respiratory failure or paralysis is followed by permanent cardiac arrest. Vital centres may be compressed or concussed directly or they may be injured by secondary changes. Another mechanism is markedly raised intracranial pressure. Other causes of death are infections, hypostatic pneumonia, pulmonary embolism and renal infection.

INTRACRANIAL HAEMORRHAGE

If the bleeding is small and thin-layered, it is called haemorrhage. If it is large and space-occupying, it is called haematoma. The application of a moderate force to the head may cause a severe intracranial haemorrhage, while a greater force may not produce haemorrhage. Injuries to the meninges may cause extradural, subdural or subarachnoid haemorrhage.

(1) EXTRADURAL HAEMORRHAGE : The dura is a strong and grey-bluish connective tissue membrane and is firmly attached to the skull. It forms the sinuses which are the drainage channel for the venous blood of brain, dura and bone. Extracranial veins also interconnect with the sinuses via emissary veins. Extradural haemorrhage is caused almost exclusively due to trauma. At the moment of impact, the skull moves relative to the dura beneath it, and the dura is stripped from the bone. This produces an empty extradural space at the site of trauma. A blood vessel may be injured at the same time. The vessel injured depends upon the site of trauma. (1) A blow over the lateral convexity of the head may fracture squamous temporal bone and injure the middle meningeal artery, especially in its posterior course as it passes upwards and backwards across the temperoparietal region. Sometimes, the thin-walled meningeal veins which groove the bone are involved. Venous bleeding will not produce sufficient pressure to strip dura from bone. Less commonly, the posterior meningeal artery near the foramen magnum (parieto-occipital haematoma) or anterior meningeal artery near cribriform plate are injured. (fronto-temporal) (2) A blow over forehead involves the anterior ethmoidal artery. (3) A blow over the occiput, or low behind the ear, may tear the transverse sigmoid sinus and produce posterior fossa haematoma. (4) A blow on the vertex may cause haemorrhage from sagittal sinus. (5) Venous extradural haemorrhage accompanies fracture of skull (usually occipital), and is due to bleeding from the diploic veins. In some cases the bleeding is both arterial and venous.

It is the least common type of meningeal bleeding and is seen in one to three percent of cases of head injury. These haemorrhages are rare in the first two years of life due to the greater adherence of the dura to the skull, and the absence of a bony canal for the artery, but are common in adults between 20 to 40 years. Haemorrhages may occur

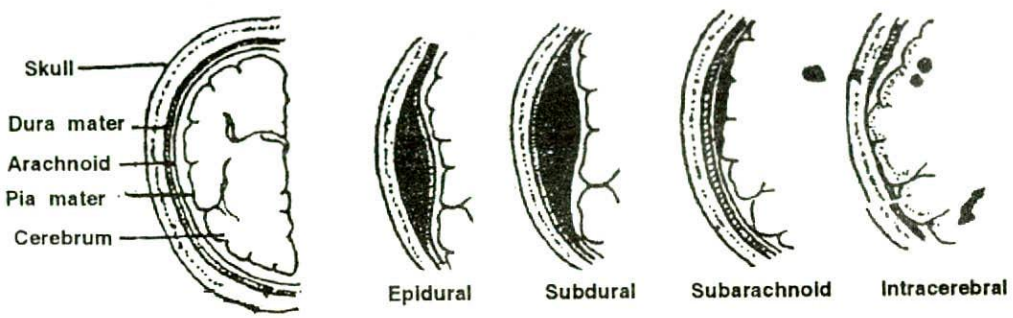


Fig. (9–12). Types of intracranial haemorrhage.

due to fall from a small height, or on being hit by a moving object, or after a minor accident. The initial deformity probably separates the dura from the skull, but as the haemorrhage continues, further stripping of dura occurs with more tearing of the communicating vessels between the skull and the dura and further bleeding. It shows typical limitation due to the dural attachments at the suture lines. Bleeding may continue for many hours or even a day after the injury. In 90% cases, the fracture is of fissured type, but sometimes is depressed. Rarely, the haemorrhage is found without any fracture of the skull or any external injury to the head. In almost all cases, the haematoma is directly under the site of surface injury. Bleeding from the main anterior branch of middle meningeal artery covers the motor area of the brain, and tends to run into the middle fossa. The clot is sharply defined, presses the dura inward and causes a localised concavity of the external surface of the brain. The clot is oval or circular, about 10 to 20 cm. in diameter, 2 to 6 cm. thick, weighs 30 to 300 g. and is adherent to the dura mater. The clot is usually in the temperoparietal area or in the fronto- temporal or parieto-occipital region. Occasionally, it is frontal or seen in the posterior fossa. The haematoma cannot be contrecoup unless the skull has been grossly deformed. If it is bilateral, then trauma has been bilateral or a middle structure, such as the sagittal sinus has been injured. Usually, 100 ml. is the minimum associated with fatalities.

The accumulation of blood is most rapid when coming from a torn vein, and intermediate when both arterial and venous. About 50 percent of cases have a second haemorrhage; subdural, subarachnoid or intracerebral. Extradural haemorrhage at the base of the skull is rare. Extradural haematoma may occasionally spontaneously become smaller due to escape of the blood through a fracture into the subcutaneous tissues, and form a haematoma of the scalp. Small haematoma may undergo slow resorption by phagocytes derived from the perivascular cells of the dura.

In a typical case, there is a history of head injury which starts the bleeding, and will usually cause temporary unconsciousness. This is followed by a period of normal consciousness, the **"lucid interval"** of few hours to a week. Lucid interval is seen only in 30 to 40% cases. Lucid interval is not seen if the injury to the brain is sufficiently great, because of the overlapping of unconsciousness due to the brain injury and due to the pressure of extradural haemorrhage. As the pressure on the brain increases, the patient first becomes confused and may appear to be drunk. With increasing pressure, sleep and coma occur. In about 25% of cases, there is no unconsciousness in the beginning. Increasing weakness occurs in the face or arms on the side opposite to the haemorrhage and spreads to the leg. Pupil is dilated and not reactive to light, usually on the side of haemorrhage. Worsening clinical symptoms are due to cerebral oedema or diffuse neuronal injury. Later there is bilateral dilatation and fixation of the pupils, decerebrate rigidity and death. The usual cause of death is respiratory failure due to compression of the brain stem. There may be cerebral oedema and secondary haemorrhages in the pons. Tentorial herniation occurs largely from the pressure of the blood clot, and also due to the brain swelling beneath the haematoma. 20 to 50 percent cases are fatal. At autopsy, gentle removal of blood may show the break in the vessels and fissured fracture of the nearby skull, sometimes confined to inner table.

Chronic extradural haematomas are rare and may or may not be associated with fractures of the skull. They are commonly seen in older children and young adults. In these cases symptoms are noted 2 to 3 days after injury. Sudden death may occur after several days.

(2) SUBDURAL HAEMORRHAGE: The arachnoid is a thin, vascular meshwork and is intimately applied to the inner surface of the dura. Subdural space is very narrow and contains a small amount of fluid permitting the thin and tough arachnoid to move relative to the dura. The cerebral veins (bridging veins) cross this space to reach the sinuses. The arachnoid is attached to the dura by venous sinuses and arachnoidal granulations. This is commoner than extradural haemorrhage. It is common in childhood or old age. This occurs in the subdural space between the dura mater and the arachnoid due to: (1) Most cases are due to rupture of parasagittal bridging or communicating veins near sagittal sinus. These veins are commonly ruptured when the brain moves across the face of the dura. (2) Rupture of inferior cerebral veins entering the sinuses at the base of the skull. (3) Rupture of dural venous sinuses. (4) Injury to cortical veins which are torn by sliding motion of the brain after the head

has been arrested. (5) Lacerations or contusions of the brain and dura. (6) Laceration of the dura and middle meningeal artery so that the vessel bleeds into the subdural instead of the epidural space. (7) Re-injury of old adhesion between the brain and the dura. (8) Rupture of an aneurysm or a superficial blood vessel malformation in the brain through the arachnoid into the subdural space. (9) Secondary to disease, e.g., cerebral tumour, cerebral aneurysms, or blood disorders. (10) Berry aneurysms may rarely bleed directly into the subdural space or a subarachnoid haematoma can rupture into the subdural space. (11) Drugs such as dicoumarol, warfarin and heparin can produce subdural haematoma usually, but sometimes without a history of trauma.

Subdural haemorrhage may occur from relatively slight trauma, often insufficient to cause unconsciousness and usually not producing fractures of the skull, and may be associated with contrecoup contusions. About 70% occur due to falls and assaults and 25% due to vehicle accidents, and are especially likely to be found in alcoholics, old persons and in children. It may occur in the absence of fracture of the skull or cerebral contusions or other visible brain injury. It occurs after the head impacts a hard surface and the brain is accelerated, which causes tearing of the parasagittal bridging veins.

Death may occur, if the haemorrhage is about 100 to 150 ml. Rapid development of a subdural haematoma will cause compression of the brain stem and secondary brain haemorrhage. The cerebral convolutions retain their normal contours, because the blood presses both the crests and depths of the gyri. The haematoma causes displacement of the cerebral hemispheres with flattening of the convolutions of the opposite hemisphere as they are pressed against the dura and bone. It is commonly seen over the upper lateral surface of the cerebral hemispheres but it may occur anywhere and may cover the entire side of the brain or even be bilateral. It is most commonly supratentorial. It usually appears as thick layers of blood over the superior surface of the brain, which drain down under gravity and cover the whole hemisphere, with a large accumulation in the middle and anterior fossae. The haemorrhage may remain fluid or may clot into a firm mass. It is essentially venous or capillary and not arterial. The volume of the blood varies from a few drops or a thin-layered effusion to 150 ml. or more. Fatal subdural haemorrhages are usually associated with contusions or lacerations of the brain and fractures of the skull. Often they accumulate gradually. With slow bleeding, a considerably large subdural haematoma can be tolerated without symptoms or serious side-effects. Usually, the vessels torn are so small that no main bleeding point can be discovered, either at operation or post-mortem. Small amounts of subdural haemorrhage are usually spontaneously absorbed.

It is divided into three types according to the time of onset of symptoms after the injury. (1) In the acute type, haemorrhage occurs immediately and very rapidly after the trauma. (2) In the subacute type, the symptoms develop from several days to 2 to 3 weeks after injury, due to the pressure of the haematoma. The clot contains some dark fluid with formation of a thin peripheral membrane. (3) Chronic type results from slight trauma in which symptoms appear some weeks or months later.

(1) ACUTE SUBDURAL HAEMORRHAGE : It arises mostly from rupture of large bridging veins, rupture of one of the cortical arteries, or cerebral lacerations. Blood from the torn vessels spreads freely in the subdural space, though it is greater near the injured vessels. In severe head injuries, it may be bilateral. The blood is usually liquid or semi-liquid. It may vary from a thin layer of one mm. to 2 to 3 cm. thickness, but in fatal cases a layer half to one cm. covers a large area of one or both hemispheres. The commonly affected areas are those in which cortical damage is commonest, i.e., the frontotemperoparietal regions. Fresh subdural haematoma (liquid blood) over the top of the brain can be easily washed off, in contrast to subarachnoid haemorrhage. Tears of sinuses produce clots in unusual positions, such as the posterior fossa over the occipital lobes or between the hemispheres over the corpus collosum. The sinuses are usually torn by penetrating wounds or depressed fractures.

In the acute type, the clinical picture closely resembles extradural haemorrhage, but the symptoms are delayed for 24 to 48 hours, instead of 2 to 4 hours. When there is a lucid interval, this may be longer than the average 4 hours of extradural haemorrhage. Subdural haemorrhage is almost always of traumatic origin. In the early period after the formation of haematoma cerebral compression is not produced, but secondary changes in the haematoma may increase the size considerably. Death is very common due to secondary pressure upon the brain stem. Subdural

haematoma produces some degree of cerebral shift and herniation, notably transtentorial.

If infarction is due to subdural haematoma, it will be underneath it and is of more recent origin than the oldest portion of the haematoma. If infarction is due to stroke, there will be proximal disease of the cerebral arteries, such as severe atheroma in cervical carotid arteries, coronary atheroma, scarring of the heart muscle or disease of the valve. The infarction does not necessarily underlie the haematoma and will be as old as the oldest portion of the haematoma.

(B) SUBACUTE TYPE : This occurs when the bleeding is from smaller bridging veins. In this, the brain may or may not be damaged. The blood may be thin, watery due to haemolysis or dilution with CSF, but it may appear like that of the chronic type.

(C) CHRONIC TYPE : They are usually seen over the parietal lobe and near the midline and may be bilateral, often spread over the temporal or frontal lobe, and may extend to the base. It may be fairly localised and deep, or it may occur as a widespread surface film. It presents usually 3 to 6 weeks after the injury. The fluid in the haematoma is at first reddish-brown, often with fibrin clots. Later it has a darker colour, and after several weeks changes to mildly brownish colour. A small haematoma may be replaced by fibrous tissue. The original haemorrhage gets rapidly sealed off. As the original clot becomes absorbed, chemical changes which occur in it may cause a further haemorrhage to occur in it spontaneously. Further trauma also causes haemorrhage. This second haemorrhage then becomes sealed off. Successive haemorrhages increase the volume and cause unconsciousness or death. This type of haemorrhage known as pachymeningitis haemorrhagica is usually seen in old persons. The brain atrophies in old age, and more space is produced in the skull for blood to accumulate. The haematoma will become gradually encapsulated by cells from the dura. The capsule is attached to the dura as the arachnoid does not take part in this encapsulation. This sac of blood will press on the gyri, flattening them and deforming the underlying surface of the brain. There is no shifting of the hemisphere toward the other side.

A layer of fibrin is deposited on the dura beneath the haematoma after 24 hours. Fibroblastic activity at the junction of the clot and the dura by 36 hours, and a 2 to 5 cells thick layer of fibroblasts is seen after 4 to 5 days. and only fibrin is seen on the arachnoid side. After 4 days, red cells begin to lose their shape. The blood may begin to appear brownish after five days, but this is not reliable. Haematoma is invaded by capillaries and fibroblasts in 5 to 10 days. Haemosiderin-laden macrophages are seen. A membrane 12 to 14 cells thick is present by 8 days. In about 14 days, the membrane enclosing the arachnoid surface of the haematoma begins to form, and the dural membrane attains one-third to one-half the thickness of the dura. In 3 to 4 weeks, the haematoma is covered by a fibrous membrane that grows inward from the edges of the clot. In 4 to 5 weeks, the arachnoid membrane has half the thickness of the dura, with the dural surface equal in thickness to the dura. The clot is liquefied completely and haemosiderin-laden macrophages are present in the membranes. The membrane is hyalinised on both its inner and outer sides in one to three months, with large capillaries invading the clot. This continues till complete resorption, leaving a gold-coloured membrane adherent to the dura.

SUBDURAL HYGROMA : When the arachnoid is torn, CSF may pass from the subarachnoid space into the subdural space. A large collection of fluid may accumulate and cause cerebral compression. This is called cerebral hygroma.

Persons suffering from subdural haematoma may be involved in arguments or fights, during which they collapse and die shortly.

(3) SUBARACHNOID HAEMORRHAGE: The space between the arachnoid and the very thin pia is genuine, and is called subarachnoid space. It contains the blood vessels of the brain, portions of its cranial nerves and a network of connective tissue fibres. It is filled with cerebrospinal fluid produced by the choroid plexuses of the lateral and fourth ventricles. The pia follows the surface of the convolutions along the sulci. This is the most common form of traumatic intracranial haemorrhage. In all cases of significant brain injury, some degree of subarachnoid haemorrhage is found. It may be the only complication of head injury but is often seen in association with other intracranial haemorrhages, with brain injuries and with skull fractures. This may be immediate or delayed until initial contraction and retraction of vessels has subsided. The latter is a reactionary haemorrhage (delayed post-traumatic subarachnoid haemorrhage).

Causes : (1) Rupture of bridging veins near the sagittal sinus. Focal haemorrhages result from force applied to the head, usually accompanied by shaking of the brain and its coverings within the skull. (2) Lacerations and contusions of the brain and the pia-arachnoid. (3) Rupture of a saccular Berry aneurysm, located usually at the bifurcation of one of the

vessels of the circle of Willis, or of one of its major branches. Size varies from few mm. to several cm. (usual 3 to 8 mm). Berry aneurysms are rare in children, and increase in frequency with age. Blood accumulates rapidly on the undersurface of the brain. With continued bleeding, blood passes along the fissures into the major cisterns and into the fourth ventricle. It may occur due to sudden rise of blood pressure due to emotional stress, such as assault, sudden exercise, sexual intercourse, etc. Alcohol also results in aggressive behaviour and a fight and fall, resulting in ruptured aneurysm. When an aneurysm ruptures, the victim seldom falls to strike his head. In most cases, the aneurysmal rupture is probably due to or consequent upon the fall and resultant head injury. The aneurysm should be examined at autopsy before fixation in formalin. To demonstrate the aneurysms, a constant stream of water should be poured over the base of the brain, while blunt dissection is made with the handle of a forceps or scalpel. Sometimes, the aneurysm is embedded in the brain surface. Intracranial aneurysms cause more than 50% cases of spontaneous subarachnoid haemorrhage. Berry aneurysms ae most often found (about 90%) at the bifurcation of the middle cerebral, anterior cerebral (more common) and posterior communicating arteries. They are least common in vertebral artery. (4) Angiomas and arteriovenous malformations cause spontaneous subarachnoid bleeding. (5) Asphyxia. (6) Blood dyscrasias, leukaemias, etc. Minimal trauma may precipitate haemorrhage.. (7) Tears of the ventricular ependyma. (8) Rupture of an intracerebral haemorrhage of non-traumatic origin (apoplectic haemorrhage or stroke) into the subarachnoid space. (9) A kick or a heavy blow on the side of the neck especially beneath the ear, can cause bleeding from the vertebrobasilar vessels in the posterior cranial fossa. (10) Sickle cell disease is a rare cause.

Traumatic subarachnoid haemorrhage over the base of the brain can be caused by lacerations of the internal carotid, vertebral, or basal arteries. These injuries can be immediately fatal. The most common causes of vertebral artery trauma are blows to the neck, motor vehicle accidents, falls, and cervical spine manipulation.

The vertebral artery is injured in the upper neck, usually as it passes through the foramen transversarium. The blood tracks along the vessels and bursts into the spinal canal where pressure may

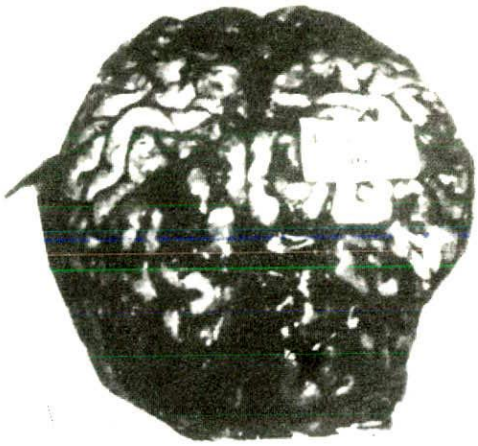


Fig (9–13). Subarachnoid haemorrhage.

be negative in the erect position. The violence causing the rupture may not cause any visible external injury, or the external injury may be so superficial as to escape notice. The lesion can be demonstrated by angiography and also by deep dissection of the upper cervical spine. The vertebral artery may rupture from sudden stretching due to partial dislocation of the upper cervical spine or atloido-occipital joint, or a fracture of the tip of the transverse process of the atlas. A partial dislocation and vertebral artery injury commonly occurs, when the normal neuromuscular control of the neck muscle is lowered, leading to a slow and inadequate response to the blow. Surprise, disease, old age, arthritis and hypnotic drugs are some of the factors likely to influence this response.

In acute alcoholism, traumatic subarachnoid haemorrhage is more common due to loss of muscular co-ordination, resulting in exessive rotational forces within the head, and also possibly due to increased bleeding from congested vessels which do not contract down, and the bounding pulse of the drunken man.

It is mostly venous. In the mild form, the subarachnoid haemorrhage is present as splashes of haemorrhage over the areas of contusion. In most cases it is diffuse overlying the cerebral haemispheres. Mild or moderate subarachnoid haemorrhage does not produce any significant damage. Rarely, it causes scarring within the subarachnoid space, especially over the brain stem and in the basal cisterns. A slightly yellow discolouration of the leptomeninges is seen as the subarachnoid haemorrhage becomes older. It can be unilateral or

bilateral, localised or diffuse. It is usually found over the orbital surface of the frontal lobes, parietal lobes, and the anterior third of the temporal lobes. In most severe head injuries, it is present over the greater part of both hemispheres and is often accompanied by subdural bleeding. It spreads into the basal cisterns. The blood mixes with C.S.F. and may be distributed over the whole of the brain. The occurrence of bleeding is suggested by headache, stiff neck and photophobia, often with deterioration of consciousness. Subarachnoid haemorrhage may be produced as an artefact at autopsy during removal of the brain due to damage to the cerebral veins, and the arachnoid. It may also be produced post-mortem due to decomposition with lysis of blood cells, loss of vascular integrity, and leakage of blood into the subarachnoid space.

(4) INTRACEREBRAL HAEMORRHAGE : This may be found on the surface or in the substance of the brain. Haemorrhage into the brain due to trauma usually occurs near the surface. Relatively large, deep-seated traumatic haemorrhages are usually accompanied by other types of brain injury, e.g., cortical contusions.

Causes : (1) Capillary haemorrhages are found in softening due to anoxia or arterial thrombosis or sinus thrombosis, in blood dyscrasias, in fat embolism and in asphyxial states. (2) Spontaneous haemorrhages in the region of the basal ganglia by rupture of lenticulostriate artery is common in middle-aged and elderly persons. (3) Angioma or malignant tumour of the brain. (4) Hypertensive cerebral vascular disease. (5) Laceration of the brain. (6) Blow on the head, with or without fracture of the skull. They result from coup-contrecoup mechanism. (7) Intraventricular haemorrhages occasionally occur in the case of puerperal toxaemia in which there have been no fits.

Traumatic intracerebral haemorrhage results from coup-contrecoup mechanism. It is mostly seen in the fronto-temporal region and is often associated with fracture of skull. Acute massive traumatic intracerebral haemorrhage is associated with extensive and widespread injury to the brain. The patient becomes deeply unconscious from the moment of injury. Large haemorrhage may occur in the inferior part of the head of the caudate nucleus and pallidum from tears of small branches of the anterior choroid artery. They usually occur due to severe blows on the vertex thrusting the brain down on to the base of the skull. Most traumatic haemorrhages occur at the time of the accident, but bleeding is often slow and in the nature of oozing from venules or capillaries. The continued slow oozing may be due to damage to further small vessels in the expanding haematoma. Clinical signs and symptoms may appear many hours after the injury. In rare cases, intracerebral haemorrhage may occur several weeks or even months after trauma, due to softening following the trauma, with damage to a blood vessel which ultimately ruptures. Isolate haemorrhages in the frontal or occipital lobes are more likely to be due to trauma. Small intraventricular haemorrhages occur in all kinds of craniocerebral injuries. Rarely, subarachnoid haemorrhage enters the ventricles through the foramen of Magendi. True traumatic ventricular haemorrhage as a sole finding occurs due to the head striking a firm object, e.g., a fall or a

Table (9–2). Difference between post-traumatic intracerebral haemorrhage and apoplexy

Trait	Post-traumatic haemorrhage	Apoplexy
(1) Cause :	Head injury.	Hypertension, atherosclerosis, aneurysm.
(2) Age :	Young individuals.	Adults past middle age.
(3 Onset :	Distinct interval (few minutes to several hours) between violence and symptoms.	Sudden.
(4) Position of head :	In motion.	Any position.
(5) Region :	White matter of tempero-occipital or frontal region.	Ganglionic region.
(6) Contrecoup haemorrhage:	May be present.	Not present.
(7) Concussion :	May be seen; may become conscious before clinical effects appear.	Not present.
(8) Coma :	Spontaneous variation.	Deep unconsciousness.

fall-like injury. The bleeding may be from the choroid plexuses or from one of the veins of septum pallucidum. It may also be caused by rupture of an arteriovenous fistula. Intraventricular haemorrhages usually arise from the extension of non-traumatic intracerebral haemorrhage through the ventricle. Death may be rapid or delayed for several days.

Violence or Disease : Intracranial haemorrhage may be due to violence, disease or the effects of injury upon disease. When disease is present, sudden rise of blood pressure due to physical exercise or excitement, e.g., alcohol, scuffle, assault, etc., may rupture vessels and produce haemorrhage. The commonly found diseases are cerebral aneurysm, degeneration of cerebral arteries, syphilis, and cerebral tumours, especially angiomata, and evidence of hypertension. If such person falls and sustains a scalp wound before death, the haemorrhage may appear to be due to trauma. In such case, the age of the person, the site and extent of the haemorrhage, the presence of vascular lesions in the cerebral vessels and signs of cardiac hypertrophy and generalised arteriosclerosis are helpful in differentiating from traumatic haemorrhages. The usual source of haemorrhage is rupture of a lenticulostriate branch of the middle cerebral artery, with bleeding into the basal ganglia and adjacent structures. Rarely, bleeding occurs in the pons or cerebellum. At autopsy, the scalp and skull should be carefully examined for the presence of injuries and the entire cerebral vascular system must be examined for evidence of disease. Intracranial haemorrhage due to violence can occur without any fracture of the skull or wound of the scalp. Extradural haemorrhage is always caused by mechanical violence. Subdural haemorrhage is almost always traumatic in origin, but may be caused by local inflammation. Subarachnoid haemorrhage often occurs spontaneously from rupture of congenital aneurysms of blood vessels in the circle of Willis. A single deep-seated haemorrhage in the brain is usually due to some disease.

Age of Effusion of Blood : Recent effusion is bright red in colour, which becomes chocolate or brown after some days, and pale brownish-yellow in 12 to 25 days. As the time progresses, the coagulum becomes firmer and laminated. Microscopy is useful in forming an opinion on the relationship.

Comminution of the skull or extensive laceration of the brain usually produce immediate unconsciousness. Extradural and cerebral haemorrhage may not produce unconsciousness for several hours. Injury to the frontal lobes is less likely to cause rapid death than injuries to other parts of the brain. Skull fractures themselves are harmless. In about 20% of fatal head injuries, there is no skull fracture.

In subdural haemorrhage due to head injury, if the person lives for some days, alcohol found in subdural haematoma at autopsy roughly correspond to the alcohol level of the deceased at the time of head injury.

NECK: Fractures of the hyoid bone or thyroid cartilage may occur due to falls injuring the neck, or when the neck comes in forcible contact with the handlebar of a cycle or the dashboard of a motor car. They can also occur in a fall against the side of a brick wall. A blow on the front of the neck may cause unconsciousness or even death due to vagal inhibition or by fracture of the larynx, usually involving the thyroid and cricoid cartilages and resultant suffocation from haemorrhage or oedema of the larynx. The mucous membrane of the trachea or larynx may be torn producing surgical emphysema and cause death by asphyxia. Non-penetrating laryngeal and cervical tracheal injuries can be produced by kicks with shod foot, judo, karate, and chop blows with the heel of the hand and by vehicular accidents due to striking the dashboard.

Suicidal incised wounds are more common than homicidal, but punctured wounds are usually homicidal. In wounds of the trachea and of the larynx below the vocal cords, speech is not possible. Wounds of the larynx and trachea are not fatal, if the large blood vessels are not damaged. Wounds of the sympathetic and vagus nerves may be fatal, and those of the recurrent laryngeal nerves cause aphonia. An incision of the trachea may give rise to surgical emphysema in the cervical tissues.

Homicidal stab wounds may occur in any region of the neck, but are often found in the lower part. The wound is usually directed backwards, medially and downwards. It may injure the subclavian vessels or may extend through the apical pleura and penetrate the lung. Air may be aspirated into a cut cervical vein and produce pulmonary air embolism. The trachea or oesophagus may be cut, when the stab wound is directed medially. If large blood vessels, such as carotid arteries or jugular veins are cut, death occurs almost immediately.

CAUSE OF DEATH IN HEAD INJURY: Raised intracranial pressure due to haematoma, contusions, lacerations, infarction or swelling is the common cause

of death. (2) DAI. (3) Ischaemic brain damange. (4) Fat embolism. In case of homicide with head trauma, always retain a sample of the victim's head hair, pulled from an area adjacent to the area of trauma.

VERTEBRAL COLUMN: Fractures of the spine are caused by: (1) direct violence, and (2) indirect violence, as by forcible bending of the body or by a fall on buttocks or feet. Hyper extension is the most common mechanism of fracture of spine. Falling from a height, diving and being thrown from automobile are the common causes. The common sites of fracture are upper and lower cervical regions and junction of thoracic and lumbar segments. Fracture-dislocations and fracture of the laminae can damage the spinal cord.

FRACTURE OF TRANSVERSE PROCESSES : They are common in the regions of the lumbar spine, where the quadratus lumborum muscle is attached. Any extreme unguarded, sudden muscular contraction will produce a direct pull on the transverse process and may fracture one or more of these. Attempts to resist falling to one side may also cause strong contraction and fracture of one or more transverse processes. Rarely, if a person falls backward and strikes a projecting object, transverse process may be fractured.

FRACTURES OF SPINOUS PROCESSES : Spinous processes may be fractured by direct trauma, e.g., an object falling from a height and striking the fixed spine of a person, or from indirect violence by muscular pull, when the last cervical or first thoracic vertebra is usually fractured.

FRACTURE OF LAMINAE: They are usually caused by direct trauma, e.g.,a person falling against an object or receiving a blow usually in the neck region.

FRACTURE OF PEDICLES AND ARTICULAR PROCESSES: They result from a direct blow or severe fall upon the edge of some object. Articular processes may also fracture due to severe strain of the lumbar spine.

FRACTURE OF VERTEBRAL BODIES : Compression type (wedging) of the vertebral body occurs commonly in the lower dorsal and upper lumbar zone, particularly T12 and L1. It may occur in a fall from a height. A person falling from a height and landing on his feet may fracture one or both calcaneum(s) and the force may be secondarily transmitted to the spine, flexing it so violently that one or more vertebral bodies are crushed. If the flexion force is a general one, gradual compression occurs and the spongy bone of the vertebral body is compressed between the superior and inferior plates. If the fall is accompanied by tilts or torsion, pedicles or laminae may be fractured. If a heavy weight falls upon the flexed back or neck, wedge-compression lesions of the vertebral bodies occur with fracture-dislocation of the articular parts of the vertebrae. This may cause compression, laceration or crushing of the cord and paralysis of the body below the seat of injury. If fracture occurs above the fourth cervical vertebra, spinal injuries are immediately fatal, due to damage to the phrenic nerves. Firearm injury to the spine often breaks one or two vertebrae into several pieces.

Hyperextension fractures of the lumbar or dorsolumbar junction are rare. Hyperextension may cause crushing of the posterior portions of the vertebral bodies and the vertebral arches. Hyperflexion may cause comminution of one or more vertebral bodies with crushing of their anterior portions. In both types of injury spinal cord and meninges may be injured due to the displacement of portions of vertebral bodies or arches into the spinal canal. Hyperflexion can produce rupture of the posterior and hyperextension rupture of the anterior longitudinal spinal ligaments without fracture, which produce instability of the spinal column. Fracture-dislocation of vertebra results from a violent force, e.g., when a passenger is thrown out of the car in an accident. Such injuries are of two types. In the first type, there may be pure anterior dislocation of one vertebra upon the one below. In the second type, the pedicle is fractured with forward or backward displacement of the body of this vertebra upon the one beneath. Injury to the cord or cauda equina may be present.

Injuries to the atlas and axis are more serious than similar lesions in the lower cervical vertebrae, because of the possible paralysis of the respiratory centre. Forward dislocation of the odontoid process is usually fatal due to crushing of the cord.

SPINAL CORD: Fractures of the spine need not injure the cord, but the cord is rarely injured without associated fractures of vertebral column "**Whiplash injury**" is an exception to this general rule. This is the injury sustained commonly by the occupants of the front seat. When the vehicle comes to a sudden stop, the forward thrust produces a state of acute hyperflexion, but this is converted into acute hyperextension as the forehead strikes the windscreen which causes injury to the cervical column. In such cases, and also due to a sharp blow against the spinous process of an upper cervical vertebra (rabbit punch), fatal contusion or laceration of the spinal cord may occur without fracture of spine.

Spinal cord can be injured by penetrating wounds, by fracture and fracture-dislocation of vertebrae. A puncture wound between first to third cervical vertebrae (pithing) may cause instantaneous death due to injury to the medullary centres or upper part of spinal cord. Even a light blade may enter the cord between the laminae, as when the victim is bending when struck or the blade directed from below upwards. In the cervical region, the laminae are narrower and a horizontal thrust can penetrate the cord. Quadriplegia occurs due to injury to the cord above the level of emergence of roots serving the brachial plexus (fourth cervical). Paraplegia occurs due to injury to cord at the level of the first or second thoracic segment. The common sites of injury to the cord in order of frequency are: the lower cervical region, the thoracolumbar junction and the upper cervical region. Falls on the buttocks, and the lifting of heavy weights may cause protrusion of an intervertebral disc, and may compress adjacent spinal nerve root.

CONTUSIONS : Contusions of the spinal cord may result from a direct blow on the spine, indirect violence or penetrating wounds. Spinal contusions may occur without any fracture or dislocation of the vertebrae. The haemorrhages usually extend in the axis of the cord. Bleeding may occur either into the spinal meninges (HAEMATORRHACHIS) or into the substance of the spinal cord (HAEMATOMYELIA). In some cases, bleeding occurs in the central grey matter of the cervical region, and may extend upwards and downwards. If the patient survives long enough, the haemorrhagic tissue breaks down to form a long fusiform cystic cavity filled with yellow fluid and surrounded by breaking down cord tissue.

Laceration of the cord with rupture of pia mater occurs with severe injuries. In rare cases, the entire cord may be completely severed.

COMPRESSION of spinal cord rarely occurs from effusion of blood from a fall. The cord is rarely penetrated in its upper part by sharp-pointed instruments. Firearm wounds may cause cord injury, even when missile has not entered the cord.

Concussion of the Spinal Cord : It may be caused due to some momentary collision of the cord against the wall of the canal or a transient deformity in the profile of the canal due to violent acceleration/ deceleration or rotational strains. This commonly occurs in railway and motor car collision, and is known as **'railway spine'**. It also occurs from severe blows to the back, compression from dislocation or fracture of the vertebrae, damage by effusion of blood, fall from a height or a bullet injury. It produces temporary paralysis, affecting the arms and hands or bladder, rectum, or lower extremities. The symptoms may appear immediately or after some hours. These are headache, giddiness, restlessness, sleeplessness, neurasthenia, weakness in the limbs, amnesia, loss of sexual power and derangement of the special senses. The paralysis is temporary and recovery occurs in about 48 hours.

Momentary dislocation of the spine may occur especially in the cervical region, causing crushing of the cord, followed by self-reduction. Autopsy may show areas of haemorrhagic discolouration on the surface or in the substance of the cord, or there may be subthecal effusions of blood. Blows on the spine without fracture or dislocation, may be followed by oedema, venous thrombosis and softening of the cord.

CHEST : Injuries of the chest may be: (1) non-penetrating or 'closed', i.e, they do not open up any part of the thoracic cavity, and (2) penetrating or 'open'. Most closed chest injuries are caused by blunt force. Blunt force applied to the chest may cause abrasions and contusions of chest wall and injuries to the lungs, heart, large blood vessels or the oesophagus which may or may not be accompanied by external wounds of the chest wall or fracture of the ribs or the sternum. Severe blows on the chest wall may produce concussion of the chest, shock and death even when the viscera are not injured. Traumatic rupture of intrathoracic trachea and bronchus usually occurs due to compressive injury of the chest. The most frequent site is within 2.5 cm. of the carina, especially in the main bronchi.

A stab wound through the heart may pass down into the liver. Stab wounds through the lower part of the pleura may cross the costophrenic space without touching the lung and pass into the stomach producing pneumothorax and entry of stomach contents into the chest.

A penetrating injury of the chest, which damages only the parietal pleura may produce an open pneumothorax, communicating directly with the external air. The jagged end of a broken rib mass also causes this. This may be followed by an irritative pleural effusion and empyema. If visceral pleura is also damaged, air enters from the lung, even after the chest wound is closed. In this type, large volumes of air may accumulate under positive pressure. The other complications are: interstitial emphysema of the chest wall, lung, and mediastinum, haemothorax or haemopneumothorax.

RIBS: If compression is front to back, lateral rib fractures may occur, and if back to front, the ribs tend to fracture near the spine. If compression is from side to side, the ribs may fracture near the spine and sternum. The middle ribs from 4th to 8th are usually fractured. In fractures due to direct violence, the fragments are often driven inwards and lacerate the underlying structures. In a fall from a height, or when run over by a motor car or bullock cart, or compression of the chest by the knees or elbows, the ribs are fractured symmetrically on both sides, in front near the costal cartilages and at the back near the angles. Fracture of the ribs from muscular contraction during violent coughing, sneezing, etc., is very rare. In falls on to the side, rib fractures are often seen in the anterior or posterior axillary lines. Fractures of first three ribs usually produce severe injuries to the tracheobronchial airway and great vessels of the upper anterior chest. Fractures of 10 to 12 ribs can cause injury to the diaphragm, liver and spleen. Multiple unilateral or bilateral rib fractures give rise to a 'flail' or 'stove-in' chest, with consequent paradoxical respiration and interferes with respiratory exchange and also with blood return to the right atrium. In an adult, blood loss in multiple rib fractures may be one to two litres, due to injury to intercostal vessels which arise directly from the aorta.

STERNUM: Fractures of the sternum are not common. They may be caused by direct violence and usually occur transversely or by indirect violence as a result of forcible flexion or extension of the body. In artificial respiration, sternal fractures may occur at the level of third or fourth intercostal space.

LUNGS : Compression of the chest or blows from a blunt weapon produce contusions or lacerations. Pulmonary damage from compression may be gross or microscopic, focal or diffuse, and located centrally, peripherally or both. If the impact is severe, there may be internal laceration of the tissue either as multiple small areas of damage, or there may be a large irregular cavity, 'traumatic cavitation'. Lobes or parts of a lobe may be detached. The hilum may tear. Extensive parenchymal lacerations can occur beneath an intact pleura. Lacerations are usually produced by the penetration of the lung tissue by fractured rib ends. After severe head injuries, where victim has been maintained for some time in a respirator, areas of collapse and haemorrhage with the formation of hyaline membrane is seen, 'RESPIRATOR LUNG'. Haemorrhagic pneumonitis may also occur.

The presence of blood in pulmonary alveoli is not pathognomonic of haemorrhage by rhexis. In many types of anoxic death (natural and traumatic), alveolar capillary permeability is so increased by endothelial hypoxia that profuse haemorrhage occurs by diapedesis. Pulmonary parenchymal haemorrhage may also occur in severe passive pulmonary congestion, probably due to diapedesis and capillary rupture. Blood in air-passages and alveoli may be due to aspiration of blood from nasal and oral injuries.

Rapid accumulation of blood and air in thoracic cavity due to perforation of a lung or large bronchus can result in lethal mediastinal shift to the contralateral side. Minimal bleeding into thoracic cavity with tension pneumothorax can cause death several hours after injury. Sudden death following minor pulmonary injury can result from cardiac inhibition due to mechanical injury to the pleura. A wound of the lung causes frothiness of blood, which issues from the mouth and nose or in coughing. High explosive blast produces numerous haemorrhages into the lung tissue, intense capillary congestion, distension and rupture of vesicles. Contusions caused by direct violence occur on the surface of the lung or deep to the site of impact, and appear as single or multiple irregular haemorrhagic zones, or as horizontal bruises corresponding to the rib markings on the lateral surface of the lungs. Sudden compression of the chest may produce CONTRECOUP CONTUSIONS due to violent displacement of air in the lungs to the posterior surfaces near the angles of the ribs. They may extend laterally or forwards into the substance of the lungs. A pure pulmonary contusion heals rapidly and completely without residual scarring. Thin-walled blebs on the surfaces of the lungs indicate rupture of alveolar sacs from pressure on the chest. Pincer forces operating in V-shaped spaces in the chest produce wedge-shaped phrenico-costal contusion of the lungs. Stab wounds of the lungs are usually not fatal, unless a major pulmonary blood vessel has been severed. Pneumothorax is common. Spontaneous pneumothorax may occur following rupture of an emphysematous bulla. Iatrogenic pneumothorax may occur by external cardiac massage, percutaneously introduced subclavian catheters and continuous ventilatory support.In decompression multiple thin-walled, small emphysematous bullae appear on the surface of the lung.

HEART : Contusions and lacerations of the heart may be caused by direct violence on the chest or by compression of thorax, or when a driver is forcibly thrown against the steering wheel. This may or may not be associated with external injury or fracture of the ribs or sternum. Cardiac contusions are usually seen on the anterior surface of either ventricle or the interventricular septum. Recent cardiac contusions are dark-red, haemorrhagic areas which are usually subepicardial. Cardiac contusions may simulate

myocardial infarction, but are usually haemorrhagic, and transition from normal to haemorrhagic tissue is more abrupt upon microscopic examination. Antemortem contusions show localised interstitial haemorrhage and rupture of myocardial fibres. They usually heal completely, but rarely may undergo necrosis with rupture into pericardial sac. Large ones may be transmural. The commonest pincer lesion is a contusion of the right atrium at the entrance of the inferior vena cava. This is seen in compression injuries. It is produced by an impact between the liver and the heart across the right pericardio-phrenic angle. Some injury to the related part of the liver is common. It may cause sudden death several days after the injury. CONTRECOUP CONTUSIONS of the heart are seen over the posterior wall of the left ventricle. They are seen in traffic accidents in which the driver is thrown forward against the steering wheel and the heart is compressed against the vertebrae. Contusions may cause sudden death from ventricular fibrillation, or they may cause progressive circulatory failure and death after few hours or days. Foreign bodies, e.g., bullet, may remain embedded in the myocardium for years without producing any symptoms.

CARDIAC CONCUSSION: Cardiac concussion can occur after a sudden forceful impact directed at the mid-anterior chest wall in the region of the heart, i.e. COMMOTIO CORDIS. Death occurs due to ventricular fibrillation or asystole. The functional disturbance may be caused by reflex coronary vasoconstriction and myocardial ischaemia or by abnormal autonomic responses.

Lacerations of the pericardium and the heart may be caused by fracture of the ribs or the sternum. If the wound is small and oblique, the victim may live for some hours or days. Death may occur from shock and haemorrhage, haemothorax or cardiac tamponade.



Fig (9–14). Ruptured myocardial infarct.

In falls from heights and in traffic accidents where the wheels of the vehicle passed over the trunk, sudden displacement of blood occurs into the thorax from the abdomen and the lower limbs leading to a rapid increase in intracardiac pressure. This may be sufficiently powerful to rupture the heart. Myocardial lacerations occur more frequently if the heart is filled with blood at the moment of impact, due to bursting force within the chambers. The heart is commonly torn due to compression, because the blood which it contains is incompressible. Auricular wounds are more dangerous than the ventricular. The heart may be ruptured by compression or from a blow or a fall, usually on its right side and towards its base. It may or may not be accompanied by marks of external violence or fracture. The common sites of traumatic cardiac rupture in order of diminishing frequency are: right auricle, right ventricle, left auricle, ventricular septum and valves. The only natural cause of rupture of the heart is softening or thinning by infarction, which invariably occurs in the left ventricle. Rupture of unhealthy muscle can be precipitated by any conditions associated with increased blood pressure and extra strain. Lacerations of valve cusps, leaflets, chordae tendinae or papillary muscles usually cause rapid cardiac failure.

Stab wounds of the heart are dangerous. If the left ventricle is pierced, the thickness of the muscle wall may restrict the bleeding, allowing time for surgical treatment. A stab of the right ventricle is more rapidly fatal, blood escaping through the wound to cause haemopericardium and cardiac tamponade. 200 to 300 ml. of blood can cause death by increasing intrapericardial pressure and producing progressive external compression of the heart, with subsequent inadequate filling of chambers and interference with ventricular contractility. The right ventricle is more likely to be wounded, as it exposes its widest areas on the front of the chest.

In gross crushing injuries, if coronary artery is bruised, it may cause a traumatic coronary thrombosis within a few hours. Direct injury to the coronary arteries usually does not cause death. Separation of the heart from its attachments is possible due to traction force on the organs away from the neck.

Rarely, left anterior descending branch is injured due to blunt trauma to the anterior chest-wall. Atherosclerotic vessels are more susceptible to trauma. Diffuse inflammatory changes in heart muscle, i.e., traumatic myocarditis, is seen in head injuries resulting in death after a few days in a coma, or injuries affecting respiration with resulting hypoxia.

BLOOD VESSELS: Wounds of the aorta or the

pulmonary artery are rapidly fatal. Wounds of the large veins produce air embolism. Most ruptures of the aorta are due to indirect force and may be complete or partial involving only intima. The rupture of the aorta commonly occurs at the junction of the arch and the descending parts, just beyond the origin of the le subclavian artery and is due to violent compression of the chest. It is common in traffic accidents and from falls from a height. Spontaneous rupture of the aorta may occur from local disease. The inferior vena cava may be torn transversely by severe trauma.

DIAPHRAGM: Wounds of the diaphragm may result from bullets or weapons which penetrate the cavity of the chest or abdomen, or by a severe blunt trauma to the anterior chest, severe, sudden blunt abdominal trauma with increased intra-abdominal pressure and upward displacement of abdominal viscera, or a fall from a height or from compression of the trunk. Lacerations may be unilateral or bilateral, anterior or posterior. It usually occurs near the central tendon on the left side and may be associated with visceral injury. It is frequently associated with fractures of the ribs and thoracoabdominal injuries. Death due to herniation of the abdominal contents into the chest (usually stomach, spleen, intestines and omentum in lacerations of left side), may be delayed for months or even years.

OESOPHAGUS: It is rarely ruptured near the lower end due to severe violence or vomiting. The ruptures are usually single, longitudinal splits on the lateral or posterior wall. Laceration can involve only mucosa or complete wall with perforation. Perforation of the oesophagus can result from the passage of instruments. A punctured wound of the oesophagus may produce mediastinal and cervical surgical emphysema and fatal mediastinitis.

COMPLICATIONS OF CHEST INJURIES: (1) Pneumothorax. (2) Haemothorax. (3) Chylothorax. (4) Interstitial emphysema. (5) Arterial air embolism. (6) Cardiac tamponade. (7) Air embolism. (8) Intraparenchymal haemorrhage. (9) Infection.

ABDOMEN: Injuries of the abdomen like those of the chest may be (1) non-penetrating or closed, and (2) penetrating or open. "Closed" abdominal injuries are caused by blunt force and occur in falls, in traffic accidents and in assault by blunt weapons. Abrasions, contusions and lacerations of the abdominal muscles occur due to blunt force. Profuse subcutaneous or deep-seated bleeding of the abdominal wall may track along the muscular and fascial plane to become more diffuse, and may cover a large area of abdominal wall, especially in the lower segment. Blood may track down the inguinal canal and appear in the scrotum or labia. Apparently trivial injuries may rupture abdominal viscera. In order of frequency, the structures most likely to be damaged in blunt abdominal trauma are: liver, spleen, kidney, intestine, abdominal wall, mesentery, pancreas and diaphragm. Injury to the stomach is caused by localised blunt force applied to the epigastric or left upper quadrant, e.g. kick or a blow with a fist. This crushes the stomach between the anterior abdominal wall and vertebral column. There is usually a circular defect with ragged ecchymotic edges. Severe or fatal internal haemorrhage may occur without any sign of injury on the abdominal wall, especially if clothing overlies the area, probably due to compressive, absorbable nature of abdominal wall. If the victim anticipates the blow and tightens the abdominal muscles, this will disperse the force of impact and reduce the probability of internal injuries, e.g. in boxers. Sometimes, the skin of the abdomen and chest is not damaged, but the underlying muscles are torn by kicks, blunt weapons or street accidents causing protrusion of a portion of a viscus behind the skin. The firmer and denser a viscus, the greater is its friability. Solid organs are more readily lacerated by blows than hollow organs. Readily moveable or displaceable organs have considerable capacity to absorb the force of blow without serious injury, because of their ability to "ride with the punch". The more sudden and forceful a blow to the abdomen, the more likely is the trauma to be serious and to involve solid viscera.

Penetrating wounds may be produced by a cutting or stabbing instrument, by a firearm, by the horns or claws of an animal or by a fall on a sharp projecting point. A single wound may result in injuries to more than one organ. Penetrating wounds of the lower chest may extend through the diaphragm and injure liver, spleen, stomach or intestines. When these wounds heal, the viscera protrude in the scar. A ventral hernia may take place. Penetrating wounds of the liver are relatively common than the spleen. The abdominal muscles may be torn by violent muscular action, e.g., convulsions of tetanus or in trying to escape a blow.

STOMACH AND INTESTINES: The jejunal portion of small intestine mostly occupies the umbilical and left iliac region; the ileum chiefly occupies the umbilical, hypogastric, right iliac, and pelvic regions. Injuries of the stomach and intestines may be caused by (1) forces of compression or 'crushing' forces, (2) traction or 'tearing forces', and (3) forces of disruption or 'bursting' forces. Compression forces produce contusions or lacerations. Contusion may occur in any of the layers of the bowel wall. Large contusion may form sloughs. Lacerations may be complete or incomplete. Small intestine is more commonly injured

by forces of compression than the stomach and the large intestine. Transverse colon is usually involved. Large contusions may form sloughs, which can cause delayed perforation. Mesenteric blood vessels may be damaged and thrombosis in these vessels may produce infarcts of the intestine. Traction forces cause displacement of the stomach and intestines, but may stretch and rupture the attachment of the stomach or intestines, e.g., the mesentery may be torn near its intestinal attachment as a result of displacement of the small intestine. Traction force applied to the intestine may rupture the junction of the fixed and mobile parts, e.g., at the duodenojejunal junction. Dueodenum is the widest and most fixed portion of small intestine. A blow to the central abdomen, especially in children may crush the duodenum against the front of the spinal column, sometimes transecting it clearly so as to simulate a cut. Forces of disruption, e.g., a blow over the abdomen cause contusions or ruptures. The ruptures are often multiple and occur along the length of the antimesenteric border of the bowel. The jejunum is the commonest site of rupture, followed by the ilium, duodenum, caecum, and large intestine. In crushing injuries there may be complete severance of the bowel. Less violent injuries produce a series of semicircular splits. Spontaneous rupture of the intestines may occur from chronic ulceration or from very slight force, if they are diseased or distended. Sometimes, paralytic ileus may occur due to injury. Thrombosis or haemorrhage may follow if the mesentery is contused or torn. The intestines and mesentery may be injured through the uterus by perforating instruments during a criminal abortion. Blast injuries may produce lacerations.

Compression may result in partial rupture of the stomach wall, with longitudinal mucosal tears, parallel to the lesser curvature. Similar injuries may be seen after forcible vomiting, e.g., in poisoning. More severe injury may cause laceration of the full thickness of the wall and rarely the stomach may be completely transected. The stomach distended with food or diseased from ulcer or cancer is easily ruptured by blunt violence, usually at the pyloric end and the lesser curvature. Traumatic rupture of the stomach may occur due to overdistention, as when an anaesthetic tube enters the gullet by mistake. Spontaneous rupture of the stomach may occur when there is an ulcer or even in the absence of disease. The stomach or small intestine may rupture due to an unexpected forceful blow, due to the relaxed abdominal wall musculature. The abdominal skin and the abdominal wall usually do not show any injury. Damage to the mesentery or mesenteric attachment of the intestine may be fatal because of the haemorrhage rather than inflammation.

Laceration of a single large mesenteric artery can cause death due to massive intraperitoneal haemorrhage. Colonic injuries are not common but midportion of transverse colon may be injured due to crushing between anterior abdominal wall and vertebral column.

The rectum is injured from wounds through the perineum, e.g., insertion of a foreign body through the anus for torture, or in sexual perversion, or from falling on a projecting point. A column of air under pressure rushing from the nozzle of a compressed air pipe, which is a little away from the anus may enter the bowel through the anus and cause fatal injury. Rupture of rectum or pelvic colon can occur by the tip of an enema syringe or a sigmoidoscope. The rectum of infants is sometimes torn by end of a thermometer.

In stab wounds of the abdomen, the small intestine is injured more commonly than the large intestine, and the stomach often escapes. The intestinal wound may be situated at some distance from the external wound due to the compression and mobility of the intestines, and the depth of the wound is greater than the length of the penetrating object.

PANCREAS : It is located retroperitoneally. The tail crosses the upper pole of the left kidney. Wounds of the pancreas are very rare. The pancreas may be injured by compression forces usually where the neck and body overly the second lumbar vertebra, when the viscera are crushed against the spinal column when the stomach is empty. A kick or a punch in the upper abdomen may injure the pancreas and cause death within a few days from inflammation. When the stomach is empty, the pancreas alone may be ruptured vertically by being pressed against the spinal column by the object struck.

Penetrating wounds of the pancreas are not common. Laceration of the pancreas may produce profuse intraperitoneal haemorrhage. Retroperitoneal fat necrosis, mesenteric fat necrosis or chemical peritonitis may result from the escape of pancreatic juice.Trauma to the pancreas can result in residual pseudocysts.

LIVER : It is the most frequently damaged abdominal organ and is second only to the brain in overall visceral susceptibility. Most injuries occur on convex surfaces. The liver is commonly ruptured by a blow, kick, crushing motor accidents, fall or by a sudden contraction of the abdominal muscles. They usually occur in association with other injuries, such as fracture of ribs, rupture of diaphragm, etc. Signs of external injury may or may not be present. Liver contusions may be difficult to recognise. Blunt force to the abdomen may produce the following types of

hepatic lacerations: (1) Transcapsular lacerations over the convex surface of the liver under the site of impact. The lacerations are often shallow splits, branching out in a cobweb-like pattern across the surface of the organ. (2) Subcapsular lacerations over the convex surface of the liver under the site of impact. (3) Non-communicating or central lacerations in the substance of the liver. (4) Coronal lacerations over the superior surface due to distortion. (5) Lacerations of the inferior surface due to distortion. (6) CONTRECOUP LACERATION involving the posterior surface of the right lobe, at the point where it rests against the vertebral column.

If the force impacting the front of the liver is directed upward, it may lacerate the undersurface of the liver. If the force is directed downward and backward, superior surface is lacerated. If the force is directed straight at the liver along its anterior margin, lacerations occur both in the concave and convex surfaces. Transcapsular lacerations may cause rapid death from haemorrhage and shock. A subcapsular haematoma may rupture several hours or days after the injury and cause fatal delayed intraperitoneal haemorrhage. The right lobe is five times more commonly affected than the left. They usually involve the convex surface and inferior border but may only involve the deep substance of the organ, consisting of blood fissures. They are usually directed anteroposteriorly or obliquely. An impact on the right upper part of the abdomen may cause tearing of the liver close to its ligaments. Tangential or glancing blunt force to the right upper quadrant of the abdomen may separate the liver capsule from the underlying tissue with subcapsular haemorrhage. Rarely one lobe of the liver may be detached.

Mild degree of external violence may rupture the liver if it is diseased, e.g., fatty metamorphosis, abscess formation, malaria, bilharziasis. In newborn child, there may be a laceration in the liver or a large subcapsular haematoma which may not rupture for several days. This results from sudden pressure to the chest during delivery. In some cases, liver tissue emboli may produce fatal pulmonary embolism. In cases of central or subcapsular rupture, blood may pass through the bile duct into gastrointestinal tract. Penetrating wounds of the liver are relatively more common and may cause death by haemorrhage and shock.

Injuries of the GALL BLADDER and the extrahepatic bile ducts are rare, and are associated with injuries to other abdominal viscera. A gall bladder distended with stone may rupture spontaneously. The extravasation of bile into the peritoneal sac may cause peritoneal irritation and infection.

SPLEEN : The spleen lies in the left upper quadrant of the abdomen extending to the epigastric region, largely protected by the lower ribs. Penetrating wounds of the spleen are less common than those of liver, but bleeding is more profuse. The spleen may be injured by forces of compression or traction forces. Compression forces produce lacerations. Traction forces may tear the spleen from its pedicle. The spleen is ruptured usually in its concave surface, and is usually associated with injuries to other organs and rib fractures. Lacerations are usually transcapsular and may occur at the hilar or convex surfaces. They are often multiple and may simulate the alphabetical figures, Y, H, or L. Death from rupture of spleen is usually rapid, due to profuse haemorrhage. A relatively mild trauma or even the contraction of the abdominal muscle may predispose the spleen to rupture when it is diseased and enlarged,e.g., malaria, Kala Azar, and leukaemia. Spontaneous rupture of the spleen can occur in malaria, typhoid fever, haemophilia, leukaemia and infectious mononucleosis. A single blow may produce more than one rupture. Sometimes, the splenic substance may rupture without damage to the capsule. In such a case, death may be delayed for some days, as the capsule limits the rupture or prevents severe bleeding. The effused blood under the capsule clots, presses on the rupture and prevents further bleeding. The clot may be disturbed due to sudden muscular excitement, with further bleeding and death.

COMPLICATIONS OF ABDOMINAL INJURIES: Laceration of the spleen produces rapid and copious haemorrhage. Laceration of the liver produces slow bleeding due to the relatively low pressure in the hepatic sinusoids but considerable bleeding occurs over a period of time. Peritonitis is more common in ruptures of the large intestine than ruptures of the small intestine due to the presence of pathogenic organisms in the colon. Chemical peritonitis is caused by leakage of gastric contents or pancreatic juice into the peritoneal cavity. Multiple contusions of the intestines may produce paralytic ileus.

UROGENITAL TRACT : KIDNEY : Injuries to the kidneys are not common as they are situated in relatively well-protected part of the body. Contusions and lacerations usually result from blunt force applied directly to the posterior or lateral aspect of the kidneys as from blows to loins. When the kidney is diseased, it may be injured from slight external violence. Contusions may be localised, or generalised and may appear as horizontal bruises corresponding to the rib markings over the posterior surface. Contusion about the upper pole of the right kidney is caused by its crushing against the lower ribs by force transmitted

through liver.

Lacerations of the kidneys may be transcapsular, subcapsular and transrenal (tear extending from the capsule to the renal pelvis). In transcapsular and transrenal lacerations of the kidney, the capsule ruptures at multiple points often along lines which radiate from the hilum towards the convex border. These may cause haemorrhage into the perinephric fat and form a large perirenal haematoma. An extensive retroperitoneal haemorrhage usually occurs due to some secondary trauma, e.g., lacerations of the liver or the kidney, but it may occur as a primary traumatic lesion and cause death from shock. The kidneys may be ruptured when a person is run over by a vehicle, or falls from a height or is crushed. In transrenal lacerations, haemorrhage may occur as a primary traumatic lesion and cause death from shock. Blunt force applied to the loin may cause contusion or laceration of the renal pedicle. If the renal artery is torn, death may occur rapidly from haemorrhage. A partial tear of the artery may be followed by thrombosis and renal infarction. Contusion of pedicle may produce renal spasm and infarction. In falls from height, renal artery may be torn. Injuries of the ureters are rare.

The kidney is encapsulated and filled with blood and urine due to which a severe blow initiates forces which act according to PASCAL'S LAW, which states that the "force exerted upon any part of enclosed fluid is transmitted equally in all directions". Violent blows to a kidney can cause bursting injuries with fragmentation or multiple bisections.

Penetrating wounds are produced by bullets or pointed weapons usually through the loin, and other viscera are also injured, with retroperitoneal haemorrhage. Infarction of the kidney occurs due to direct injury to the renal artery, or a large intrarenal vessel. The complications may be sepsis, and the extravasation of urine into the surrounding tissues with the development of urinary fistula.

ADRENALS : The adrenal gland may be injured by the same force which damages the kidney and may be lacerated or crushed. Haemorrhage (adrenal apoplexy) may be rarely seen associated with other injuries.

BLADDER : In children, the anterior surface of the bladder is in contact with the lower part of the abdominal wall. Beginning at puberty, it slowly begins to descend in the pelvis. The bladder may be lacerated from a fall, a kick or a blow on the abdomen. When the bladder is distended, the peritoneum over the upper surface is stretched and tears, which often extends through the bladder wall and urine is extravasated into the peritoneal cavity. When the bladder is only partially distended, blunt force to the lower abdomen may cause an extraperitoneal rupture. Rupture of the bladder usually occurs in association with fracture of the pelvis. In extraperitoneal ruptures, the urine may extravasate upwards to the level of the kidneys or downwards along the spermatic cord into the scrotum, which may produce cellulitis and death. Spontaneous rupture of the normal bladder is rare, but may occur when it is ulcerated or diseased (tuberculosis, carcinoma, cystitis, etc.), when there is obstruction in the urethra. During parturition, the bladder may rupture due to the pressure of the child's head. It may rupture by a cystoscope, catheter or other body introduced through the urethra.

Stab wounds of the lower abdomen may penetrate bladder and may cause rapid death from haemorrhage. There may be extraperitoneal extravasation of urine. A high velocity bullet penetrating the distended bladder causes an explosive effect on the urine, which may produce extensive laceration of the bladder.

The male urethra may be ruptured usually under the pubic arch by a kick in the perineum, by a fall on a projecting substance, by fracture of pubic bone or by a foreign body. Forcible catheterisation or cystoscopy, especially in the presence of some obstruction can cause rupture of urethra from within. The tear in the urethra may be complete or partial, and the wound may communicate with the skin. Usually damage occurs in the bulbous or membranous urethra. Violent displacement of a full bladder, as in crushing injuries of the pelvis may rupture the posterior urethra. The female urethra may be ruptured by an act of rape.

FEMALE GENITAL ORGANS : Contusions and lacerations of the vulva and vagina may be due to kicks during assaults, or falls on a projecting substance. Wounds of vulva caused by a blunt weapon may resemble incised wounds. Lacerated wounds of the vulva may bleed profusely. The vaginal wall may be lacerated during delivery, which may extend into the bladder or rectum.

The uterus, ovaries or the Fallopian tubes may be contused or lacerated in severe compression injuries of the pelvis. The non-gravid uterus is not usually injured. The gravid uterus may be ruptured by a blow, kick, trampling on the abdominal wall, or by instrumental criminal abortion, or in obstructed labour. Placenta may separate from uterus and cause death of foetus. Penetrating wounds are not common.

MALE GENITAL ORGANS : The penis may be injured by a squeeze or crush, and the engorged erected penis may be completely avulsed from the pubes by forceful pull. Self-inflicted injuries may be

seen in insane persons. Accidental injuries are rare, but they may be injured or amputated from motives of revenge. Penile strangulation may occur due to placement of a constricting apparatus around the penis. The testicles are contused from blows, kicks and squeezes. Compression or crushing of the testis may cause sudden death from cardiac inhibition.

LIMBS : Abrasions, contusions and deep lacerated wounds involving the muscles and extending up to the bone are common in traffic and industrial accidents. Deep-seated limb injuries affecting the muscles, vessels, nerves or bone may occur without any external evidence of injury.

FALLS FROM HEIGHT: In a typical case of primary impact with the feet, the bones of the feet are fractured (usually comminuted and often open) and the tibias may be driven through the soles of the feet; fracture-dislocation of the ankles; comminuted or oblique fractures of the tibiae and fibulae; fractures of the femurs (shafts, condyles or neck); pelvic fractures; fractures and dislocations of the vertebral column; ring or comminuted fractures of the base of the skull and injuries to the brainstem and inferior surface of the brain. Fractures in the lower limbs could be unilateral or bilateral. There may be lacerations of the liver and spleen. The aorta usually ruptures at the junction of the aortic arch with the descending thoracic aorta. Intimal tears are often seen along the thoracic or abdominal aorta. Ruptures or lacerations of the pulmonary trunk, arteries and veins and of the superior and inferior vena cavae are less common. This full range of injuries does not occur in every case.

In primary head impact, open comminuted or depressed skull fractures with brain lacerations and partial or complete extrusion of the brain may occur. This may also be seen with primary impact of the feet and subsequent secondary head impact.

Imprint abrasions and contusions are common when the body lands on hard patterned surfaces. Bruising of the palms and fingers may be seen if the victim clings into the edge of the building or parapet before releasing the grip. Perineal bruising due to relative movement of the pubic-perineal region against the clothing may be seen in vertical feet-first impacts, which may be misinterpreted as resulting from sexual assault. All sorts of lacerations may be sustained. Bodies may be transected or decapitated as they hit other objects. Usually, the greater the height of the fall, the more severe are the injuries. In most cases, the posture or orientation on impact determines the regional preponderance of injuries. The body can bounce off the surface as it impacts and then fall some short distance away.

The severity of injuries is not directly related to the distance of the fall of the person. Some persons may die after falling from a standing position on to the back of the head, but some may survive a fall of many metres. In old people, falls can cause fracture of the neck of the femur, ribs, arms, and pelvis. When a person falls or jumps from a height, the trajectory is downwards and outwards, and the distance the body strikes the ground is variable. Much depends on whether the victim fell passively from near the wall or projected himself outwards at the top. The body turns and twists in an unpredictable manner during fall. A person falling 15 metres attains a speed of 17m/s. When falling from a high building, the displaced air tends to act as a cushion which drives the body from the wall. A simple fall can result in a body impact some distance from the foot of the building, which is not an evidence of a push or of a deliberate jump. The site of primary impact shows most severe injury. Sometimes, two areas may strike the ground simultaneously, e.g. head and shoulder, or the person may bounce and two or more major impacts occur. If the person falls on to the head, both vault and base can fracture, with extrusion of brain. When the person falls to the side of the body, fractures of multiple ribs, shoulder girdle, arms, and contusions and lacerations of back, buttocks or limbs, and severe abdominal and thoracic injuries can occur. The lungs are most sensitive to deceleration; pulmonary haemorrhages and haemothorax are common. The liver is most commonly injured followed by heart and aorta. The aorta tears usually either at its origin or at the end of the thoracic arch.

AORTA: Rupture of the thoracic aorta may occur in traffic accidents; the driver's chest striking the steering wheel, or the front seat passenger's chest striking the dashboard, or impact of the occupant's chest against the pavement following ejection, or pedestrian sustaining a violent impact to the chest or been run over. Falls from height and crushing chest injuries also cause rupture of aorta. A transverse tear of the aorta immediately above the cusps of the aortic valve may occur when a violent force compresses the heart and intrapericardial portion of the ascending aorta. This is associated with fractures of sternum and upper ribs. Lacerations are usually transverse and may be associated with rib and/or vertebral fractures. Most common site is in the descending thoracic aorta, just distal to the origin of the left subclavian artery. Rarely, a periaortic haematoma due to laceration of aorta may produce a false aneurysm. Traumatic rupture of abdominal aorta may occur in automobile accidents.

INJURIES OF ARTERIES : A direct injury of a

large artery of the limb, e.g., the brachial or femoral artery, may produce contusion or partial or complete rupture of the arterial wall which can cause immediate localised arterial spasm (traumatic segmentary arteriospasm). Arteriospasm is commonly seen in fracture of the limb bone, and occurs when the bone ends or fragments contuse or lacerate an artery.

Contusions of the arteries are usually found in the intima, and are usually associated with tears of the intima and thrombus formation. Arterial contusions are common in crush injuries of the limbs. When an artery is lacerated or punctured, e.g., by a bone fragment or by a bullet, a perivascular haematoma may form in the surrounding tissues. The blood at the periphery of the haematoma may coagulate and organise, and if the central portion of the haematoma remains fluid, a direct communication may be maintained between the artery and the haematoma forming a false aneurysm. This aneurysm may gradually increase in size and rupture resulting in profuse haemorrhage and death. True traumatic aneurysms are rare. It may occur when outer coat of a large artery is injured, e.g., a tangential bullet injury of a vessel. A penetrating wound of a limb, e.g. from a pointed weapon or bullet may pass through an artery and the accompanying vein producing an arteriovenous fistula.

A penetrating wound of a limb, e.g., from a bullet or a stab may extend through an artery and the accompanying vein, and produce an arteriovenous fistula. In a large arteriovenous fistula situated in proximal part of a large limb artery, considerable amount of blood may leak into the related vein. Muscle necrosis and gangrene may occur due to impairment in the blood supply to the limb. Progressive cardiac decompensation and death may occur due to fall in arterial pressure and the rise in venous pressure.

Arterial thrombosis and embolism, traumatic segmentary arteriospasm, true and false traumatic aneurysms and arteriovenous fistulas may impair the circulation in a limb. Sensory and motor nerve endings are highly susceptibe to ischaemia. Muscle tissue becomes necrosed. Ischaemia persists for six to 8 hours, and skin becomes necrosed in 24 hours. Fibrosis of necrosed muscles cause shortening of the muscles and produce deformities and contractures (Volkman's ischaemic contracture). Most commonly occlusion of the brachial artery results in deformities and contractures of the forearm and hand.

VEINS : Minor injuries may damage the veins. Contusion of the wall or tearing of the intima may occur in trauma. A thrombus may form at the site of damage to the intima which may become dislodged and give rise to pulmonary embolism. Pulmonary air embolism may occur in laceration of a large vein in an open wound to a limb. Fat embolism may result from laceration of vein in regions of extensive damage to adipose tissue.

PERIPHERAL NERVES : Incised or puncture wounds may divide peripheral nerves. They may be lacerated in certain types of fractures, e.g., the musculospiral nerve may be lacerated in fractures of the shaft of the humerus. Compression or crushing injuries may cause contusion or concussion of nerves. Traction forces may injure the nerves, e.g., the ulnar nerve may be stretched in fracture-dislocations of the elbow joint. Ischaemia of limb due to arterial injury may result in paralysis and anaesthesia, even when the nerves are not directly injured.

BONES : FRACTURES OF THE EXTREMITIES: (1) DIRECT FORCE: (1) PENETRATING FRACTURES. (2) FOCAL FRACTURES occur when a small force is applied to a small area resulting in a transverse fracture. Overlying soft tissue injury is relatively minor. (3) CRUSH FRACTURES occur when a large force is applied over a large area. Overlying soft tissue is extensively injured, and often the fracture is comminuted. In severe impact injuries of the legs, the fracture may be transverse, oblique, spiral, segmental, comminuted, longitudinal split, tension wedge or compression wedge type.

(II) INDIRECT FORCE: (1) TRACTION FRACTURE: In this the bone is pulled apart by

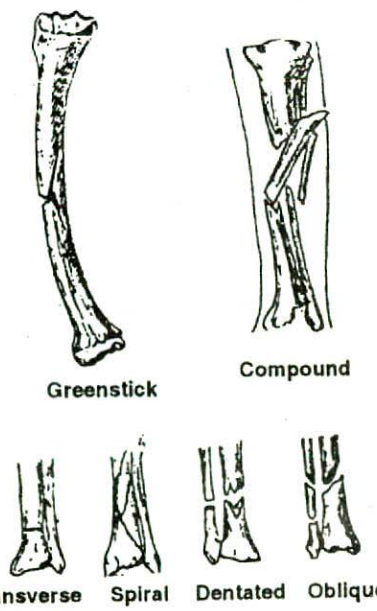


Fig. (9–15) Various types of fractures.

traction, e.g. transverse fracture of patella due to violent contraction of the quadriceps muscle. (2) ROTATIONAL FRACTURE: The bone is twisted and a spiral fracture is produced. It occurs only when the bone is subjected to torsional force. (3) VERTICAL COMPRESSION FRACTURES produce an oblique fracture of the body of long bones with the hard shaft driven into the cancellous end. (4) IN ANGULATION AND COMPRESSION fractures, the fracture line is curved. (5) ANGULATION, ROTATION AND COMPRESSION fractures are oblique.

Contusion of bone and of its periosteum occurs due to a blow or a fall. Fractures may occur from falls, blows or the action of the muscles. In direct violence, fracture occurs at the site of impact, and some injury to the overlying soft tissue is always present. They may be compound or comminuted. If the violence is indirect, a simple fracture occurs in a region distant from the site of impact of the force, e.g., a fracture of the head of the radius or of the lower end of the humerus caused by a fall on the extended palm. In a simple or closed fracture, there is no communication between the bone and the air. A fall on the outstretched hand will cause Colle's fracture (fracture of the distal end of radius). A compound fracture from indirect violence may occur when a fragment of bone pierces the skin from the inside. The sharp ends of the fractured bone produce injuries of the soft tissues. In a compound or open fracture, there is a communication between the bone and the air through a wound. Clavicle is commonly fractured at the junction of middle and lateral one-third. The sudden contraction of the muscle during an unexpected movement may fracture a bone, e.g., the olecranon or the patella may be fractured by the sudden contraction of the triceps or the quadriceps muscles respectively. Most spontaneous fractures occur due to disease, e.g., osteoporosis, osteomalacia, Paget's disease, etc. In children, green-stick fractures may be more common. In childhood, slipping of an epiphysis is common, e.g., in distal end of the radius, internal epicondyle of the humerus, capitulum, and distal end of the tibia. In simple closed fractures of the tibia and fibula half to one litre of blood is usually lost and in fracture shaft of the femur about two litres.

When a car dashes against a tree or a wall, injury to spine and fractures of the limbs may result from indirect violence. In an accident, fracture occurs at the weakest part of the bone, is usually spiral or oblique without a bruise or wound. Fractures of metacarpal bones occur usually by striking the closed hand (first) against a firm surface (BOXER FRACTURES).

At autopsy a fracture may be suspected when there is extensive swelling and discolouration of the skin, or when there is abnormal mobility, or crepitus is found. The tissues surrounding suspect fracture should be dissected to determine injuries to the soft parts.

DISTINCTION BETWEEN ANTE-MORTEM AND POST-MORTEM FRACTURES : Fractures caused some hours before death show signs of effusion of blood, laceration of muscles and oedema, which are not seen in a fracture produced after death. A fracture produced shortly before, or shortly after death has same characters, except that in the former there may be more effused blood, which penetrates further into the adjacent tissues.

AGE OF FRACTURE : Haemorrhage occurs at the time of injury, and the haematoma formed around the fracture is usually clotted within 12 to 24 hours. A fibrin network is formed within hours. Histologically, some necrosis of the bone is seen in two days. Acute inflammatory changes are seen within hours of injury and last for several days. After partial organisation of blood clot, the fracture gap may be filled with condensed highly eosinophilic fibrin. Within one to two days, there may be migration of polymorphs into the necrotic tissue and later macrophages. Osteogenic granulation tissue is formed accompanied by hyperaemia and oedema in four days. New vessels are formed and numerous fibroblasts are seen in seven days. Small areas of bone are laid down around blood vessels in an irregular interwoven manner within a week. This woven bone is formed equally by periosteal, endosteal, and marrow reticulum cells. The fibroblasts lay down reticulin and then collagen, which is usually well marked by ten days. A haemotoma, resulting from a fracture usually gets absorbed in 10 to 14 days. Cellular elements give rise to osteoblasts and chondroblasts which produce a matrix of collagen and polysaccharides impregnated with calcium, called callus in two weeks. Much of the damaged marrow is invaded by vascular fibrocellular tissue by 15 to 20 days. By X-ray examination, callus is not readily visible for three weeks. The periosteal callus gap is usually obliterated by 30 days. Callus is formed into hard bone in about two months, although the bone still undergoes reconstructive modelling. After healing is complete, an approximate estimation of the age can be given from the extent of remodelling, the smoothness of edges, and the form of trabeculae passing through the line of fracture. The last thing to occur in fracture healing is regaining of full joint motion.

If a fracture is inadequately immobilised, repeated movements of the limb cause tearing of the granulation tissue with recurring hyperaemia, continued resorption of bone and delayed union.

JOINTS: Injuries may be caused by blows or fall or by dislocations. Any injury to the synovial membrane, the intra-articular cartilages, the ligaments, or the capsule of a joint may be accompanied by a transudation of serous fluid into the joint cavity. Laceration of the synovial membrane or the joint cartilages, or fracture-dislocations cause haemorrhage into joint cavity. Serous effusions are absorbed, but blood is organised with intra-articular adhesions. Dislocations may occur due to direct violence like falls, blows, or muscular action, or spontaneously when the joints are diseased. They are not dangerous unless they occur between the vertebrae, or are compound. Punctured or incised wounds of the joints are likely to become infected. To determine the range of movement at a joint, graduated protractor is used.

The estimation of age of dislocation can be made by the colour change if bruise is present. In older cases, the amount of new fibrous tissue or the formation of false joints may give some idea of the age of the injury.

TRAFFIC ACCIDENTS

A large variety of injuries are sustained by persons involved in traffic accidents.

(1) INJURIES OF PEDESTRIANS: Three patterns of injury are often seen. (1) Primary impact injuries (the first part struck). (2) Secondary impact injuries (further injuries caused by the vehicle). (3) Secondary injuries, sometimes called tertiary injuries, (injuries caused by the victim's striking objects, such as the ground). Diagrams indicating the location, nature and extent of injuries are of value to establish injury patterns.

In primary impact injuries, the part of the body involved depends upon the position of the person in relation to the vehicle when struck, i.e., whether crossing the road from one side to the other or walking with or against the traffic. The injuries also depend upon the relative heights of various parts of the vehicle, i.e., bumper, fenders, radiator, door handles, etc. If a person is struck from behind, the back of the legs are first struck. If the foot is fixed, a fracture results, and the buttocks and back will come in contact with the car and then pushed forward. He may sustain a fracture-dislocation of the lumbar or thoracic spine, sometimes associated with the fractures of adjacent ribs. The detached portion of the vertebral column may move forward transecting the cord and thoracic aorta. If the feet slide forward, the whole body will fall backward with a secondary impact of the head against the windshield, or he may be thrown into the air, or to one side and strike the ground. If the person falls on the hood, the tangential force directed by the hood to the buttock and thigh may cause separation of the skin and subcutaneous tissues from the muscle, producing a pocket in the upper thigh and buttock. Large amount of blood may collect in this pocket, which is often not seen externally.

In the typical case, the victim is struck by the front of the vehicle, and sustains so-called bumper injuries on legs. These injuries may be severe with fractures and extensive soft tissue damage. They may also be minimal or absent externally. Usually the tibia is fractured, with forward displacement of the bone fragment, which was struck. The fracture is usually spiral or wedge-shaped. The base of the triangular fragment of bone indicates the site of impact, and the apex points in the direction in which the vehicle was travelling. If the leg is lifted, the fracture is often transverse. If the vehicle was braking violently at the moment of impact, the legs are injured at a lower level. Frequently, bumper injuries are at different levels on the two legs or absent on one leg, which suggest that the victim was walking or running when struck. In children, the bumper usually produces fracture of the femur. When he is thrown clear of the vehicle soon after the impact, he may sustain secondary injuries which vary greatly in severity. They may be abrasions,

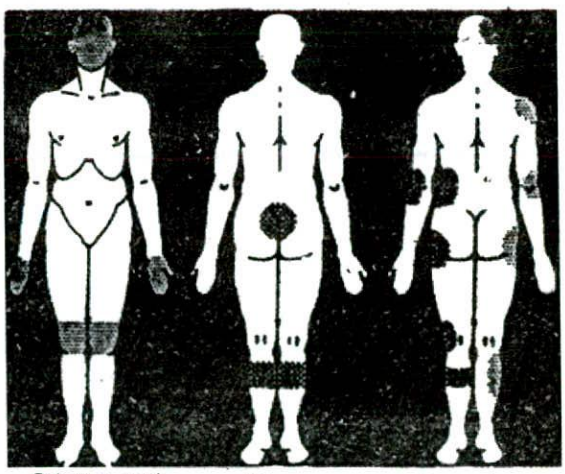


Fig. (9–16). Impact injuries; primary (cross-hatched); secondary (dotted).

contusions, fractures of various bones including the skull.

An impact against a mudguard or headlamp may result in fracture of the pelvis or fracture-dislocations of the sacroiliac joints. The pubic ramus opposite to the buttock or hip involved is usually fractured. The margins of the lacerations show contusions in primary impact injuries, and abrasions in secondary impact injuries. If the person is struck from behind, the buttocks and back come in contact with the vehicle before being pushed forward. If the person is facing the vehicle, he may sustain intra-abdominal injuries, and injuries to the chest wall and the thoracic contents. Direct impact to the thorax may cause rupture of the aorta below the arch at the level of the ligamentum arteriosum, due to the sudden increase of intravascular pressure. There may be bruising of the right atrium and sometimes laceration or rupture of the heart. Splenic lacerations cause slow oozing. Injury to the intestinal tract is less common. If the victim is struck from behind, linear superficial tears of the abdomen or the inguinal regions are seen due to overstretching of the skin which appear dry, yellow and bloodless. If the vehicle travels at high speed, deep lacerations may be produced due to the same mechanism. If the victim is run over by the vehicle, similar injuries may be produced due to overstretching of the skin by the passing wheel, and the crushing weight of the vehicle on the body. Many impacts are on front corner of the car and pedestrian may be knocked diagonally out of the path of the car. If thrown into the centre of the roadway the person can be run over by another vehicle. When a pedestrian walks into the side of a vehicle, pressure abrasions or tears on the side or front of the face are produced which are sometimes patterned. A hand, forearm or elbow may intervene between vehicle and either the face or trunk. The chest or loin may be injured sometimes with fractured ribs and rupture of lungs, diaphragm, spleen or liver. Tearing wounds may be caused by protruding objects, such as door handles, or with cuts from broken glass. If the person falls and is run over by the back wheels, crushing injuries of the head, trunk and limbs are seen.

At a speed of 20 kmph, the person can be thrown away. At a speed of 40 to 50 kmph., if the vehicle strikes a person below the centre of gravity of his body, he is thrown upward and slides on to



ig. (9–17). Linear superficial tears of the inguinal region and lower abdomen in a pedestrian struck from the automobile from behind.

the hood(bonnet) of the vehicle (scooping-up) and may sustain superficial abrasions of his elbows, shoulders and head, or severe injuries from striking the bonnet or protruding objects and especially the windscreen or windscreen pillars at either side. Impact with the windscreen and secondary injuries cause fractures of ribs, arms, pelvis and injuries to the abdomen. Often the injuries are found more on one side, usually on the opposite side to the point of primary impact. Subsequently, the person slides sideways off the hood and sustains a final impact with the road's surface, usually by the head and occasionally with the buttocks. Fractures of the sacroiliac joints and fractures and dislocations of cervical spine due to sudden unexpected hyperextension of the neck following initial impact are common. Atlanto-occipital dislocation and partial disruption of intervertebral disc are common. When the vehicle travels at a greater speed, the pedestrian may be thrown higher and falls on the vehicle roof sometimes somersaulting so that the head strikes the roof. He can then slide or thrown over the back of the car on the road behind the vehicle. In general, the higher the speed more severe the injuries.

If a fast moving bus, truck or van having a straight and high front end hits an adult, the impact is naturally much higher, at hip or even shoulder level. Even low speeds can cause severe injuries. If the body is hit at hip level, the victim will be pushed directly forwards in front of the vehicle. If the body is hit high up, the upper part of the body will be pushed down, or he may be carried on the radiator grille for some distance and fall in front

of the vehicle as it slows down or comes to a halt, and the victim may be run over. If clothing or part of the body becomes engaged under the bumper or in the wheel arches during the initial impact, the person will be dragged underneath, instead of being thrown upwards. Protruding objects may cause serious injuries. In children, impact with the front of a vehicle is above the centre of gravity. The victim is pushed down and run over. If prior to the impact, the vehicle is braking hard, the front of the vehicle dips below the child's centre of gravity throwing the child forward.

It is always not possible to associate injuries sustained by pedestrians with specific features of external automobile design, especially when the victims are children or the accident is of the low speed type.

Second collision refers to impact of the person against an object inside or outside the vehicle after the vehicle itself has hit a relatively stationary barrier. The most extensive fractures are produced by the head striking a flat surface. In pedestrians, the head is injured by direct impact with the vehicle and by striking the road. Both impacts produce fractures involving almost all parts of the skull, especially the base. Fractures may extend transversely across the base of the skull through the middle fossa to the lower parietal bone on each side. Extensive injuries of the brain are caused under such distortion and with added severe acceleration or deceleration. Extensive surface laceration with much subarachnoid and often subdural bleeding may occur. Haemorrhage into the basal ganglia and severe laceration of the corpus callosum may occur. Bleeding into the uncus and brain stem are rare.

If the person is run over, there may be (1) Tyre marks, often spread a little due to yielding and flattening of the body from pressure. (2) Grazes. (3) Avulsion. The rotatory effect against a fixed limb may strip off almost all tissues down to the bone. The avulsed wound may be segmental or circumferential, the latter completely encircling arm or leg. If scrotum or abdominal wall are injured, testes or intestines are extruded. (4) If the brakes of the vehicle are applied, the wheels will lock and long lacerations are produced due to the shearing action of tangential force. Complete amputation of a limb or decapitation may occur. (5) Burning of the skin due to the heat produced by sliding of the body. (6) Deep crushing of the internal organs and flattening deformities of the head, chest, or pelvis. Decapitation or complete amputation of an extremity may occur. When a child is run over by a vehicle, fractures may not be produced, because of the elasticity of partly cartilaginous skeleton. If a tyre passes over the head of the victim, avulsion of an ear usually occurs, because it is pulled off the moving wheel. If the vehicle travels from back to front in relation to the victim, the tear is produced behind the ear. If the vehicle travels in the opposite direction, the tear is produced in front of the ear.

Rolling Injuries: Rolling injuries are produced when a vehicle with a low chassis, "rolled" the victim along the roadway as it passed over him. They are usually located on two or more body surfaces and run circumferentially. They are mostly abrasions, grease soiling and burns from the exhaust system. There may be fractures of the skull, neck, shoulder, shoulder-girdle, ribs, vertebrae or pelvis. Patterned imprints may be present caused by parts on the undersurface of the chassis. The elasticity of skin and tissues tends to keep the body in one piece, though broken. At very high speeds (130 km/ph), impacts result in hemisection of the victim.

(2) CYCLISTS AND MOTOR CYCLISTS: This includes motor cycles, motor scooters, mopeds and pedal cycles. Accidents to cyclists and motor cyclists are commonly caused by turning in front of a vehicle from one side or the other. The cycle is hit, and the person is thrown violently to the ground or into some other object or on to the car. In being thrown, they may injure the groins or legs by protruding objects, such as handlebars or projection levers or mirrors, long tears being common. The handlebar may tear the liver or the spleen and cause massive internal bleeding. The head or shoulder may hit some object first, or they may be thrown beneath a vehicle and crushed like any pedestrian. Primary impact injuries may be to the leg or to the chest if the victim runs into a stationary vehicle, often causing a ruptured heart or aorta. Injuries to legs may occur in relatively low speed accidents due to the machine falling heavily on them. Falling from the vehicle, especially at speed can cause visceral damage, especially rupture of the liver and spleen. Pillion riders falling off the backs of motor cycles have a laceration of the back of the head, a fracture of the posterior fossa, contrecoup contusions of the frontal lobes and abrasions of the back and elbows. Crash helmet reduces friction of the head against

the ground, and makes deceleration less drastic by allowing the protected head to skid across the ground. When a crash helmet is worn, the crown may be protected, but the whole head may be "eggshelled" on to the base or cervical spine with fracture of the spine. Occasionally, when the helmet is pinned or crushed, the chin strap may be drawn upwards and cause strangulation. Although helmets do decrease fatalities, their greatest value is protection at lower speed or tangential impacts. A helmet which shows only glancing linear abrasions indicates the likelihood of a less severe brain injury than one in which there is direct impact imprint. About 50% of helmetless motor cyclists sustain head injury. The person falls head-on into some hard surface, or slides and tears the unprotected brow, face or ears with flattened or depressed fracture of the skull. The brain is crushed or torn and bleeds from multiple sites. Impact on the crown of the head may produce ring fracture around the foramen magnum. In about 25% of cases cervical spine fractures occur. Severe brain damage may occur even when a helmet is worn. Cortical contusions and lacerations are common. Brain tissue may extrude through compound fractures of the skull.

The classical fatal injury in both motor cyclists and pillion passengers is fracture of the skull, usually from secondary impact with ground. Temperoparietal fractures are common often with contrecoup brain injury. In many types of road accidents or due to a very heavy impact on one side of the head, a fracture is produced across the floor of each middle fossa passing through the pituitary fossa, often associated with fissured fractures passing upwards to the temporal bones. At autopsy, the base of the skull is seen divided into two halves, each moving independently of each other like a hinge, the so-called **motor cyclists fracture.**

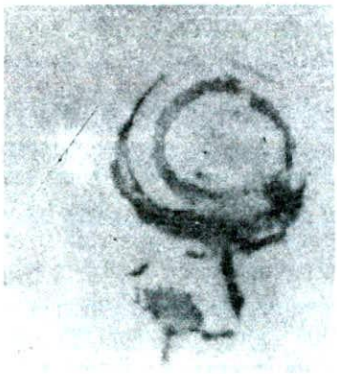


Fig. (9–18). Steering wheel impact injury.

Bicycle spoke injury: It occurs when a rider or passenger on a bicycle slips from the seat and the foot or leg passes through the spokes of the wheel. In this soft tissue is crushed, with internal avulsion or damage.

The motor cyclist may drive into the back of a large vehicle, e.g., truck, known as **"under-running" or "tail-gating".** This may occur due to the sudden and unexpected stoppage of the truck or when the motor cyclist is at high speed in darkness. In such cases, the motor cyclist's head and shoulders are smashed against the tail-board. In extreme cases decapitation may occur.

OCCUPANTS OF THE VEHICLES : Most of the vehicular accidents are frontal (two vehicles colloiding head-on or vehicle striking a fixed object occurs in about 80% of all vechicular accidents) causing violent deceleration. The occupants of the vehicle will continue to move forwards, even though the vehicle has stopped. The unrestrained driver first slides forwards so that his legs strike the fascia parcel shelf area and his abdomen or lower chest strikes the lower edge of the steering wheel. This is called **"secondary accident".** Then the body flexes across the steering wheel and begins to rise. The heavy head goes forward and there is flexion of cervical and thoracic spines. The head strikes the windscreen, the upper windscreen rim or the side pillar. The windscreen may break and the person may be ejected through the broken glass on the bonnet or on the road. The front seat passengers will sustain the same impact except on the steering-wheel. The back seat passengers will impact on the back of the front seat. The driver may be thrown forward and strike the chest on the steering-wheel or the upper abdomen on wheel-rim. The horn boss may strike the sternum and produce a transverse fracture and crush the heart or split the aorta. The wheel-rim may crush the liver, spleen or kidneys. In some cases, lungs are contused or lacerated, due to fractured ribs penetrating the lung tissue. Transection of the aorta usually occurs immediately distal to the origin of the left subclavian artery. There may be transverse lacerations of the intima of the aorta. The heart may show contusions, laceration of the pericardial sac, rupture of right and left atrium, right ventricle and interatrial septum. The throat may be crushed across the horn-ring or

the top of the steering-wheel. These are often known as **'the steering-wheel impact type of injury'.**

If the driver appreciates that the accident is unavoidable, he may put some of the force on his hands on the steering-wheel and avoid full impact of his chest against it, or severe impact of the head against the windscreen, and may sustain injuries to the wrists or forearms. Transmitted fracture of the femur or pelvis occurs if attempt is made to apply the brakes. The driver and front passengers may split the head or face on a shattering windscreen frame, or may sustain a fracture-dislocation of the cervical spine due to hyperflexion of the head or being thrown up as well as forward. Dislocation of atlanto-occipital joint is found in about one–third of all fatal motor vehicle accidents.

Whiplash injury is due to a violent acceleration or deceleration force applied to the passenger, usually front seat occupant. When the vehicle comes to a sudden stop due to head-on obstruction, the heavy head continues to move forward (acute hyperflexion). When the body comes to rest, or the head strikes an obstruction in front, there is then a reactionary hyperextension as the head is thrown backwards, this double movement being known as the 'whiplash'. If a vehicle is hit violently from behind by another vehicle, then the head is thrown violently backwards into hyperextension and then comes forwards after body stops movement. In either case, this violent extension-flexion movement can cause dislocation of the upper cervical spine, or less commonly a fracture-dislocation in the lower part of the spine at about C 5 and 6. Fatal contusion or laceration of the spinal cord may occur without fracture of spine. Fracture or dislocation can also occur in the upper dorsal spine, often around T 5 to 7.

The head strikes the upper part of the window glass, frame and adjacent roof; upper limbs and thorax strike the window-sill and adjacent door structures; and hip and abdomen strike the arm-rest area. Head injuries are caused mainly by contact with portions of the vehicle interior located at head level, e.g.,upper part of the side door and window-frame and glass, windshield, sun visor and its hinges and rear vision mirror. When the forehead strikes the windshield, shallow penetration produces U-shaped, L-shaped, right angle or linear cuts or abrasions, and deeper penetrations lacerations. Multiple punctate lacerations of the face are produced due to the shattering of the windscreen glass into multiple small cubical fragments with relatively blunt edges (**"sparrow foot"** marks). They are relatively superficial. Thin pieces of windscreen glass may be embedded in the wounds or may be found loose in the clothing. There may be partial avulsion of the skin. These wounds may bleed profusely. Impaction of the head with the windscreen can cause fracture of the base of the skull, closed head injury and fractures of the cervical vertebrae. Basal fractures tend to run along the petrous ridges through the sella turcica (hinge fractures).

In a head-on crash, the front passengers move forward in a seated position, the knee striking the rigid metal lower panel, chest and abdomen striking the upper portion of the panel and the head is thrown against the windshield. Abrasions and lacerations are produced on the shins and knees, or one or both patellae are fractured, or knee joint is crushed. Abrasions and lacerations on the dorsum of the foot and on the toes, sometimes with separation of great toe-nail, may be caused by hyperflexion of the toe at the time of impact. Such injuries are probably caused by the foot slipping off the depressed brake pedal and flexing against the metal frame in front. The femur or the pelvis may be fractured by being thrown against the dashboard or shelf-edge. In forward crashes, the floor of the vehicle frequently bends inward, twisting the foot on the ankle and causing fracture-dislocations. Sometimes, the foot may be trapped and the shaft of the tibia or fibula fractured, as the occupant's body moves forward.

In front-to-side collisions, injuries to the face due to shattering of the glass can be seen on either or both sides of a driver. In these, doors on the side opposite to impact are opened. If the impact is from the right side, the driver will sustain external injuries mostly on the right side of the body. Injuries are usually more severe than those sustained in frontal collision and usually involve the chest and abdomen. The right arm or leg may be fractured. Rib fractures occur mostly on the right side. Heart may rupture. Liver and right kidney may be lacerated. Fractures of the base of the skull and neck may occur. If the impact is from the left side, the injuries tend to be more severe on the left side.

During violent deceleration, unrestrained back seat occupants are projected forwards and strike the back of the front seats including head rests and suffer from fewer facial, neck, pelvic and arm

injuries but more abdominal trauma. They may be thrown over the seats and fall on the front seat passengers or sometimes ejected through the windscreen.

Aorta is usually ruptured with deceleration. It may be associated with a severe whiplash effect on the thoracic spine. The aortic rupture is circular and clean-cut, and appears as sharp as if it has been transected with a knife. Sometimes multiple transverse intimal tears are seen adjacent to the main rupture, the so-called **"ladder tears"**. Contusion of the pericardium and myocardium may be seen even without fractures of ribs. The posterior surface of the heart may be damaged from impact against the spine. In high speed impacts, the heart may be completely avulsed from its base and may be found lying loose in the chest cavity. The auricles and ventricles may be lacerated. The heart may be lacerated directly from fractured ribs, sternum or external objects. Blunt impact often produces a line of bruising down the posterior part of the lung where it lies in the paravertebral gutter. Air bullae or blood blisters may be seen under the pleura overlying the bruised areas. Haemothorax or pneumothorax may be present. The interior of the lung may be pulped due to transmitted force or large variations in intrathoracic pressure during the impact. The liver may rupture in any part. Usually there is central laceration of the upper surface, which may extend deeply and may transect the organ. There may be shallow, multiple, parallel tears in the upper surface of the right lobe. In subcapsular tears a haematoma is formed which can rupture later. Sometimes there is gross pulping and disruption or internal laceration without surface injuries. Shallow tears are produced in the spleen usually around the hilum. Rarely the spleen may be avulsed from the pedicle. There is often contusions of mesentery and omentum.

Sometimes, the door may spring open and the person may fall out (occupant ejection). Occupants thrown out of the car can sustain injuries to any area of the body, and multiple injuries to the chest and abdomen are common with multiple limb fractures. When the car overturns, the occupants may be pinned and crushed, showing classical traumatic asphyxia, or the chest and abdomen may be crushed with serious injury to the heart, lungs, liver, spleen or kidneys. Depending on the circumstances of the accident, the occupants are burned, asphyxiated or drowned while trapped in the interior of a car. Tail-gating occasionally occurs with cars driving into the back of large trucks. In such case, the windscreen and front of the passenger compartment are smashed with severe injuries to the occupants of the front seat.

Seat Belt Injuries: Seat belts are of the lap strap and shoulder diagonal type, which are of the "inertia-reel" type, which allow slow movement but jam at a sudden tug. A seat-belt acts in the following ways: (1) It restrains the body against severe deceleration, keeping it away from windscreen, steering wheel and other obstructions. (2) It spreads the decelration force over a considerable area of broad strap against the body surface. (3) The strap stretches during deceleration and reduces G-forces by slightly extending the time of deceleration. (4) It prevents ejection from the vehicle into the road. It reduces risk of death by about 40%. Small intestine avulsions, intestinal, omental and mesenteric lacerations and intestinal contusions and perforations occur, known as **"seat belt syndrome"**, due to acute flexion over a lap strap. Rupture of the spleen, liver, pancreas, caecum and bladder occur due to compression between belt and vertebrae. Abrasions, contusions and haematomas of the lower abdomen can occur as a direct result of seat belt trauma. The abdominal aorta can be crushed. Transverse fractures of lumbar vertebrae, usually at the second or third segment of interspace can occur due to acute trunk hyperflexion over the belt fulcrum. The posterior arch, pedicle or transverse processes may be fractured.

Triage is the sorting out and classification of casualties of war or other disaster, to determine priority of need and proper place of treatment.

INVESTIGATION OF AUTOMOBILE ACCIDENTS : Investigation of traffic accident should include: (1) study of the accident scene, (2) mechanical and engineering examination of the involved vehicles and of each component whose failure could affect safety in motion, (3) complete autopsy of all dead victims, (4) physical and psychiatric examination of surviving drivers, (5) personal and social histories of surviving persons.

EXAMINATION OF THE SCENE: The investigation of the scene of accident consists of a search for traces left by the vehicle, and traces left by the victim of the accident, who may have been struck by the vehicle or thrown from it.

(A) TRACES LEFT BY VEHICLES : (1) SKID MARKS: The location, direction and length of skid marks are a reliable guide as to the speed of the vehicle

at the time the brakes were applied. They are important for the reconstruction of an accident and evaluation of the statements made by the driver. Skid marks should be measured and sketched. Their shape and location should be recorded by photographs.

(2) DIRT FROM THE UNDERSURFACE OF THE VEHICLE: The location of such dirt dropped on the highway due to impact of a vehicle with some resisting substance may be of value in accident reconstruction. It may provide evidence as to which vehicle was violating traffic rules.

(3) TYRE MARKS: Tracks leading to and from the scene indicate direction of travel, speed and attempts to stop or avoid collision. Tyre marks are usually caused by the skin being forced into the grooves of the tyre tread, the edge of the raised rubber tracing out the pattern. They are helpful in the identity of the vehicle as each tyre has individual character caused by skid, cuts by glass, nails, sharp stones, etc.

(4) MATERIAL FROM THE VEHICLES : This may consist of glass from broken headlight, windshield or window mirrors, parking lights. metal parts from the grille, door handles, etc., fragments of paint or enamel from the vehicle. Puddles of oil, water or antifreeze may give evidence of damage to the vehicle if the vehicle was driven away or removed after the accident. These can be matched with the suspect's car found later.

(B) TRACES LEFT BY VICTIM: Blood, fragments of tissue, hair, and fragments of clothes may be found at the scene due to injury to the victim. The location of such evidence should be sketched and the material preserved and sent to the laboratory for examination.

THE CASE VEHICLE : The vehicles involved in accidents are photographed from various angles to record the type and amount of damage. Measurements of the vehicles are made to determine the amount of deformation and collapse of various structures. Photographs of the interior of the vehicle should be taken, especially areas of deformation and of impact points, i.e., steering wheel, the instrument panel, bent knobs, broken windshields, interior of the doors, etc. The investigators should look for items of evidential and correlative value, such as human tissue, blood, hair, cloth fibres, etc. at the possible points of impact. The undersurface of the chassis should be examined for blood, hair and fibres from clothes. The location of vehicles, the type of road and its surface in the vicinity of the accident, the location and position of victims on the road, distance and direction of the victims from the impact, and size and direction of blood on road should be noted, photographed and drawings made showing various details of the accident.

THE ACCIDENT VICTIMS: In a fatal accident, when the victim is still at the scene, photographs of the victim should be taken to show his position relative to vehicle if he was ejected.

THE AUTOPSY: The main objects are: (1) To construct the accident to determine its cause. (2) To find out the cause of death. (3) To find out the manner of death. (4) To establish identity of the victims. (5) To identify the driver and reconstruct the position of the victims prior to the accident. (6) To differentiate injuries caused by being hit from those caused by being run over. (7) To identify the automobile in cases of hit-and-run. (8) To determine the significance of previous injuries in case of delayed death.

(A) CLOTHES: The clothes should be examined while still on body to determine any evidence to indicate what part of the vehicle struck the victim. Paint, smudge marks, grease and oil marks and dirt on the garment must be noted. The clothes may sometimes show clear imprints of tyre. Pieces of glass or metals may be found in or on clothing.

(B) THE BODY : The description of nature, size, extent and location of the injuries may identify some wounds on the body with some object or part of the vehicle in question. Primary impact injuries vary in extent and severity in different cases depending on the position of the pedestrian, and on the speed of the vehicle, and the part of the vehicle hitting the person, etc. The distance of wounds from feet should be recorded, because the position of these wounds will indicate which part of the vehicle struck the person, and will serve to corroborate or contradict the evidence of witnesses. The secondary impact injuries should also be described. Drag marks may indicate that the victim had crawled from the vehicle and was not ejected. They show typical brush burning without significant haemorrhage in the skin and subcutaneous tissues. Valuable information may be obtained from the victim's injuries, regarding the mechanism of the accident. Every injury cannot be interpreted and that patterned injuries should be primarily considered. It is useful to make a diagrammatic representation of injuries on a body outline.

CAUSE OF DEATH: When a person is found in a crashed vehicle, it should be determined whether he died from injuries sustained in the accident or due to a natural cause.

(A) DISEASE AND ACCIDENTS: In the causation of accidents, diseases do not contribute very much. Natural disease can cause accident and death of driver, the commonest cause being coronary artery disease. A person suffering a heart attack while driving, usually slows down or stops the vehi time to avoid

collision, and no major injuries are sustained by the occupants or the pedestrian hit. Diabetics who take insulin, rarely experience hypoglycaemic attacks while driving, which may cause an accident. An attack of angina, valvular heart disease and migrainous headaches may cause an accident. Minor vision defects, colour blindness, and defective hearing do not contribute much for an accident. Epilepsy is a well known risk. Physical fatigue usually does not affect the accident, but mental or psychological fatigue, 'tiredness' may affect. Driver falling asleep is a major cause of accidents.

(B) DRUGS AND ACCIDENTS : DRUNKEN DRIVING is a major cause of accidents. A serious lack of control is produced by an overdose or a normal dose of insulin not covered by sufficient intake of food. Drowsiness and accidents may be caused by morphine, pethidine, cannabis, cocaine, benzodiazipines, tranquilisers, amphetamine, antihistamines and some hypotensive drugs.

(C) PSYCHOLOGY AND ACCIDENT : Disorganisation of skilled reactions may occur in persons who are emotionally upset or suffer from anxieties.

Severe blunt force injuries alone or in combination with minor natural disease will point to a traumatic cause of death. The pattern of injuries and their circumstances of death will indicate whether death is accidental, suicidal or homicidal. Severe natural disease alone or in combination with insignificant trauma will point to a natural manner of death. Minor trauma may be sufficient to cause death either by affecting diseased organs or by affecting vital areas, e.g. nervous centres of the brain or the conduction system of the heart.

Deep injuries of the back of the neck, such as:(1) haemorrhage in the atlanto-occipital joints, (2) fractures of atlanto-occipital joints, (3) extensive muscular haemorrhages, (4) extradural or subdural spinal cord haemorrhages , (5) contusions and lacerations of upper cervical cord, may be caused due to vehicular accident. These may be missed in the usual autopsy and cause of death may be given as natural. Car exhaust gas may seep into the vehicle and precipitate a fatal accident or an apparent natural death.

In the case of pedestrians, cardiovascular or cerebrovascular disease, poor vision, impaired hearing and reduced mobility may lead to accident. A dead body lying in a street may be run over by a vehicle. In such case, large amount of blood may be found in the body cavities and also some degree of haemorrhagic infiltration of tissues at the site of gross traumatic lesions.

Death may occur a long time after the person sustains injuries in a traffic accident. A continuous chain of symptoms from the accident to death (bridging symptoms) and morphologic proof that the changes of natural disease complicating the trauma can be traced to the time of the accident will prove causal relationship in delayed traffic deaths.

Wrong determination of the cause of death may result from (1) faulty reconstruction of accident, (2) incomplete gross and microscopic autopsy, (3) misinterpretation of pattern of injury, (4) improper ageing of injuries, (5) incomplete toxicologic examination.

MURDER BY MOTOR VEHICLE: A crime may be committed with the help of an automobile in the following ways. (1) Premeditated murder of a pedestrian with an automobile.

(2) HIT AND RUN: The driver of the vehicle kills or injures a person accidentally and leaves the scene to avoid blame. The vehicle and driver will have to be identified. Examination of the scene, of the body, and collection of gross and trace evidence, e.g., tearing of the clothes, grease marks, oil, road dust, broken lamp or windscreen glass, metallic or paint fragments from the vehicle, blood, urine, hair, etc., on the clothes and on the body is helpful. The clothes may show a tyre tread pattern. Matching studies of blood type, hair, dirt, grease, paint, oil, rust, etc. found on the victim and the vehicle in question can lead to the identification of the vehicle involved. Measurement of the leg fractures from the heels of the feet, and the height of other body injuries sustained from the protruding part's of the vehicle, may aid in the identification of the suspect vehicle. The pattern of injuries and other findings may help to determine the position of the victim at the time of first vehicle contact.

RUN OVER INJURIES : If a person is run over by a vehicle and dragged, impact injuries may not be found, but the clothes are torn and avulsion of skin and compression injuries of internal organs are present. The body or clothing of the victim may show oil and rust from the low parts of the vehicle and the vehicle may show fibres of clothing, hair, and blood from the victim on some of its low parts. Tyre tread patterns on the clothes or skin is valuable evidence.

(3) ACCIDENT FAKED TO CONCEAL CRIME : Rarely, a person may be killed by other means, and the body placed in a vehicle is pushed off the road to make the scene look like an accident. In such a case, careful evaluation of all the injuries is necessary. Sometimes, a murder victim is burnt in a vehicle to conceal the crime. Sometimes, putrefaction indicates

the body to have been dead prior to the fire.

SUICIDE BY MOTOR VEHICLE: Some vehicular accidents are suicidal. Suspicion may arise from the circumstances preceding the accident, e.g. family quarrel, financial crises, threats of suicide, previous suicide attempts, history of depression, suicide note, etc. The characteristic of a suicidal death by a driver is a head-on collision with a roadside object, pole or bridge support at a high rate of speed, without evidence of an effort to apply the brakes or to evade striking the object. The accelerator pedal imprint is an additional evidence of the deliberate nature of the crash. Rarely, premeditated homicide may be disguised as an accident. Suspicion should arise when the accident cannot be reconstructed.

IDENTIFICATION OF DRIVER : The driver can be identified by: (1) The presence of steering-wheel impact type of injuries. (2) Finding of paint or glass from the door on the driver's side on the victim's body or clothes. (3) Finding of cloth fibres, hair and tissue from the suspected driver on the driver panel, door or glass on the drivers side or on the steering wheel and columns. (4) The brake or accelerator pedal design is imprinted on the sole of the driver's shoe. (5) Matching of the blood group of the driver with that of blood of the driver panel, driver door and steering assembly.

AIRCRAFT INJURIES: Most of the aircraft accidents occur on landing (55%), or take off (22%). The sudden deceleration on crashing causes breakup of the aircraft and injuries to the occupants.

CRASH ACCIDENT : Both on landing and take-off the passengers are usually secured to their seats with lap type safety belts, which are fixed to the back of the base of the seats, and pass over the hips to tie across the abdomen just above the symphysis pubis. Many types of injuries are seen, in the relatively intact bodies. Fracture spine, especially thoracic spine are very common. These are usually hyperflexion injuries due to rapid deceleration when the aircraft strikes the ground. The forward momentum throws the head, on to the back of the front seat causing facial injuries. There may be fracture of the base of the skull, especially ring fracture. Fracture of the lower legs due to seat displacement is typical of the landing time accident, while fracture of the femur and internal injuries due to vertical deceleration, indicate a deep stall type of accident. Intrathoracic injuries occur due to squeezing of the chest by pressure against the sternum, which may rupture or detach the heart. Ruptures of the liver, spleen, kidneys and abdominal aorta occur. Death may be due to burning or CO poisoning. The seats may break loose due to which almost any type of injury may result. In about 20% of fatal crashes fire occurs.

FLIGHT ACCIDENTS : The cabins are pressurised to prevent anoxia while in flight. If a door or window breaks, cabin pressure falls and anoxia may produce death. The rush of air out of the cabin is sufficient to blow a standing or even seated man out with it. The injuries vary from total disintegration of the body to relatively minor injuries. When a plane breaks up at a relatively high altitude, fragmented bodies may be distributed over wide area.

AUTOPSY: (1) Identification and autopsy of the pilots for natural disease and toxicological analysis. (2) Examination of clothing of all fatal casualties. (3) Determination of cause of death of all casualties. (4) Preservation of blood specimens from all casualties for COHb and alcohol estimation and lactic acid estimation. Brain lactic acid levels exceeding 200 mg% indicate hypoxia. (5) Collection of specimens of lung from all casualties for assessment of agonal period.

BOXING INJURIES: A wide range of injuries are produced, but the head is frequently injured. Fracture of skull is rare, but subdural haemorrhage, usually within one of the middle cranial fossae, due to tearing of dural emissary veins, is the most common cause of death due to prolonged repetitive punching occurs in about one-third of fatal cases. Some victims may show pontine haemorrhage, the so-called **"boxers haemorrhage"**. Retinal detachment, choroidal tears and vitreous haemorrhages may occur. Deterioration in speed and coordination is the chief symptom of the onset of punchdrunk (traumatic encephalopathy or dementia pugilistica) condition, which may arise years after the last injury. **Punchdrunk syndrome** in its final form is recognised by slurred speech, defective memory, slow thought process, stiff limbs, ataxia, broad-based gait, outbursts of violence, Parkinsonian-like facial appearance and dementia. The intracranial findings in boxers are: subdural, subarachnoid and intracerebral haemorrhage, diffuse axonal injury, focal ischaemic lesions, dissecting aneurysms, cortical atrophy, slight hydrocephalus, thinning or tears of corpus collosum, scars or patches of gliosis, loss of neurones from the cerebellum and substantia nigra, and contusions of the brain. The changes are broadly similar to that in Alzheimers disease. These injuries can cause cerebral oedema, herniation of the brain, secondary brain stem injuries, and hypoxic injuries of the brain. The mechanism is not known, but probably repeated blows to the head produce small

haemorrhages and degenerative changes in brain, especially in the region of corpus striatum, thalamus, deep temporal gray matter, septal regions and on certain neurons along the cerebellar pathways. There is enlargement of the ventricles due to loss of white matter and a perforation of the septum pallucidum.

RAILWAY INJURIES : Accidental Injuries : When the person walks on the track, primary injuries are seen on the front or the back, due to contact with the protruding parts of the engine. Secondary injuries are produced due to being thrown or run over which are soiled with oil and dust. When the person is crossing the line, primary injuries are seen on the side of the head and shoulders. If the person is bending down, the primary injuries are seen on the back and the buttocks, and secondary injuries on the face and front of the body. When the person walks at the side of the line, the shoulder or head may be hit by some protruding part of the train. When a person falls from the train, multiple injuries are produced. When a person leans out of the window, head injuries are produced due to railway fixtures, bridge abutments or tunnel sides, etc., and the person may be found in the carriage or on the side of the track. A person trying to board a fast moving train may be thrown off or may get crushed between train and the platform. A shunter may be squeezed between the buffers during uncoupling rolling stock. When a person is run over by a train, very severe mutilation of the body may occur and the body may be severed into many pieces and soiled by axle grease and dirt from the wheels and track. Wheel marks, dirt and grease contamination may be seen on the body. In collisions, and derailment, fractures of the legs and spine are produced. Head injuries, contusions, cuts and burns are also produced. Most injuries are produced due to crushing and trapping within distorted carriages.

Suicidal Injuries : The injuries are extensive and due to primary impact. If a person lies down on the track, extrusion of organs, traumatic amputations of the limbs or trunk or decapitation may occur. Wheel marks and dirt and grease contamination may be found on the body.

Sometimes, a person is killed and the body placed on the railway track to simulate suicide or accident. Examination of the scene of the crime and the body for marks of violence will often solve the problem.

CHAPTER 10
MEDICO-LEGAL ASPECTS OF WOUNDS

Homicide : Homicide is killing of a human being by another human being.

Types : (1) Lawful : (I) Justifiable. (2) Excusable. **(II) Unlawful :** (1) Murder (S.300, I.P.C.). (2) Culpable homicide (S.299, I.P.C.). (a) amounting to murder, (b) not amounting to murder (S.304, I.P.C.). (3) Rash or negligent homicide (S. 304 A, I.P.C.).

Justifiable Homicide : This is the homicide which is justified in the circumstances which led to the killing of a person. This may occur (1) in the administration of justice, like execution of sentence of death, (2) the maintenance of justice, e.g., in suppressing riots, or executing arrest, or killing in course of violent crime, e.g., a woman who kills a person who attempts to rape her.

Excusable Homicide : This is the homicide caused unintentionally by an act done in good faith. This includes (1) killing in self-defence when attacked, provided there is no other means of defence, (2) causing death by accident or misadventure, (3) death following a lawful operation, (4) homicide committed by an insane person.

The above terms are not used in Indian law.

(S. 44, I.P.C.). INJURY : Any harm whatever illegally caused to any person in body, mind, reputation or property.

Sec. 299, I.P.C. CULPABLE HOMICIDE : Culpable homicide is causing death by doing an act (1) with the intention of causing death, or, (2) with the intention of causing such bodily injury as is likely to cause death, or (3) with the knowledge that such act is likely to cause death.

Explanations : (1) A person who causes bodily injury to another who is suffering from a disorder, disease or bodily infirmity which accelerates the death of the person, shall be deemed to have caused his death. Infirmity is any inability of an organ to perform usual function. It may be temporary or permanent. (2) Where death is caused by bodily injury, the person who caused such bodily injury shall be deemed to have caused death, although by skilful treatment, the death might have been prevented. (3) The causing of the death of a child in the mother's womb is not homicide. But it may amount to culpable homicide to cause the death of a living child, if any part of that child has been brought forth, though the child may not have breathed or been completely born.

S.300, I.P.C. MURDER : Culpable homicide is murder (1) if the act by which the death is caused is done with the intention of causing death, or (2) if it is done with the intention of causing such bodily injury as the offender knows to be likely to cause death, or (3) if it is done with the intention of causing bodily injury which is sufficient in the ordinary course of nature to cause death, or (4) if the person committing the act knows that it is so immediately dangerous, that it must in all probability cause death, or such bodily injury as is likely to cause death and commits such act without any excuse.

Exceptions : Culpable homicide does not amount to murder, if the act by which death is caused is done: (1) under grave and sudden provocation, (2) in good faith of the right of private defence of person or property, (S.96 to 106, I.P.C.), (3) for the advancement of public justice, (4) without premeditation, and (5) when the person above the age of 18 years takes the risk of death with his own consent.

COMMENT : During a quarrel, death may occur from rupture of the heart, the bursting of an aneurysm, cerebral or subarachnoid haemorrhage, hypertension or any other natural disease. If it can be proved from autopsy that death resulted from a natural cause, and that the infliction of the injury did not operate in any way either immediately or remotely, the assailant will not be responsible for the death of his victim. In these cases, death might have taken place about the same time and in the same circumstances whether there was a quarrel or not.

Death may occur from a slight injury inflicted on a previously diseased organ, e.g., miliary aneurysms, fatty degeneration of the heart, duodenal ulcer, ulcerative colitis, enlarged spleen especially due to malaria, amoebic abscess, hydatid cyst, tuberculosis, disseminated sclerosis, Parkinsonism, etc. Dissemination of a neoplasm might be accelerated by direct injury.

Acceleration of the progress of an already fatal illness may also occur following injury. In law, a causal relation is established if trauma aggravates, hastens or precipitates a disease condition. It is not necessary that the trauma be an aetiologic cause. In such cases, if injury accelerates the process of dying or precipitates death, the assailant is not guilty of culpable homicide, but he can be charged of simple or grievous hurt, if it can be proved: (1) that he had no intention to kill his victim, (2) that he could not have possibly known the existence of that disease, and (3) that the same injury would not have caused death when inflicted on a healthy person. A person can be convicted of culpable homicide, if he causes bodily injury to another who is suffering from disease, disorder or physical weakness and thereby accelerates the death of the other person, if he had intention to kill or had the knowledge that such injury is sufficient or likely to cause death.

To substantiate a charge of murder or culpable homicide, it is necessary to prove that the injury inflicted on the deceased was actually the cause of death, and the act was committed with the intention of causing such bodily injury as the offender knew that it was likely, or sufficient in the ordinary course of nature to cause death. If these conditions are not satisfied, the assailant may be convicted under the offence of culpable homicide not amounting to murder, or grievous or simple hurt, according to the circumstances of the case, including the kind of weapons and the site of the violence.

A BODILY INJURY WHICH IS LIKELY TO CAUSE DEATH is one in which death is not merely possible but is likely. It constitutes a grave danger to the life of the affected person due to its severity or of the structures involved. It applies in special cases where person injured is in such a condition or state of health, that his or her death is likely to be caused by an injury which would not ordinarily cause the death of a person in sound health, and where the person inflicting the injury knows that owing to such condition or state of health, it is likely to cause the death of the person injured.

A BODILY INJURY WHICH IS SUFFICIENT IN THE ORDINARY COURSE OF NATURE TO CAUSE DEATH is one as a result of which death is very probable. Death must be the result of the direct effects of the injury. If there was a lesser probability of death resulting from the act committed, the finding will be that accused intended to cause injury likely to cause death. An injury sufficient in the ordinary course of nature to cause death, need not cause death inevitably and in all circumstances. The sufficiency is the high probability of death in the ordinary way of nature. It is not always easy to decide whether or not an injury is one that is sufficient in the ordinary course of nature to cause death. Some examples are: stab injuries or rupture of the heart; injury to the large blood vessels; stab on the abdomen, chest or other parts of the body injuring an internal organ or a large blood vessel; hurt causing rupture of spleen, liver or an internal organ; severe injuries of the brain and spinal cord; incised wound on the neck; compound fracture of the skull; squeezing of the testicles; penetrating wounds or rupture of the gastrointestinal canal; extensive burns and scalds; hanging or strangulating a person; smothering or gagging; drowning; failure to tie the cord after it is cut in a newborn; administering large dose of poison, etc. A number of injuries, none of which is sufficient in itself to cause death may together by their cumulative effect be sufficient to cause death.

The offence is culpable homicide, if the body injury intended to be inflicted is likely to cause death. It is murder, if such injury is sufficient in the ordinary course of nature to cause death. The distinction is fine but appreciable. The nature of the weapon has to be considered. A blow from the fist or a stick on a vital part may be likely to cause death; a wound from a knife in a vital part is sufficient in the ordinary course of nature to cause death.

S. 302, I.P.C.: Punishment for murder : Death, or imprisonment for life, and also fine.

S.303, I.P.C.: Punishment for murder by life convict (death).

S. 304, I.P.C.:Punishment for culpable homicide not amounting to murder : Imprisonment for life, or up to 10 years and also fine.

S. 304-A, I.P.C. Causing death by negligence: "Whoever causes the death of any person, by doing any rash or negligent act not amounting to culpable homicide shall be punished with imprisonment for a term which may extend to two years or with fine, or with both.

S.305, I.P.C.: Abetment of suicide of child or insane prson (ten years imprisonment).

S. 306, I.P.C.: Abetment of suicide : If any person commits suicide, whoever abets the commission of such suicide, shall be punished with imprisonment for a term which may extend to ten years, and shall also be liable to fine.

S.307, I.P.C.: Attempt to murder is punishable with ten years imprisonment.

S.308,I.P.C.: Attempt to commit culpable homicide is punishable with imprisonment up to ten years

S.309, I.P.C.: Attempt to Commit Suicide:

Whoever attempts to commit suicide and does any act towards the commission of such offence, shall be punished with simple imprisonment for a term which may extend to one year, or with fine, or with both.

Shooting, hanging and stabbing are a 'hard' way of committing suicide and typically a male choice; poisoning and drowning are 'soft' ways of committing suicide.

S. 319, I.P.C.: HURT : Hurt means bodily pain, disease or infirmity caused to any person. S. 312 to 328 of **I.P.C.** deal with causing hurt and their punishments.

Infirmity is any inability of an organ to perform usual function.

S.320, I.P.C. GRIEVOUS INJURY : Any of the following injuries are grievous. (1) Emasculation. (2) Permanent privation of sight of either eye. (3) Permanent privation of hearing of either ear. (4) Privation of any member or joint. (5) Destruction or permanent impairing of the power of any member or joint. (6) Permanent disfiguration of the head or face. (7) Fracture or dislocation of a bone or tooth. (8) Any hurt which endangers life, or which causes the victim to be in severe bodily pain, or unable to follow his ordinary pursuits for a period of twenty days.

Comment : Grievous hurt is hurt of a more serious nature. It is very difficult to draw a line between those bodily hurts which are serious and those which are slight. To make out the offence of voluntarily causing grievous hurt there must be some specific hurt, voluntarily inflicted, and coming within any of the eight kinds enumerated in this section.

(1)Emasculation : It means depriving a male of masculine vigour.

(2)Permanent privation of sight of either eye: The gravity of such injury lies in its permanency because it deprives a person of the use of the organ of sight and also disfigures him. It can be caused by gouging out of eyes, poking eyes, chemicals, etc.

(3) Permanent privation of the hearing of either ear : It deprives a man of his sense of hearing. Such injuries may be caused by a blow on the head or the ear, or by blows which injure the tympanum or auditory nerves or by thrusting something or pouring hot liquid into the ear which causes deafness.

(4) Privation of any member or joint : The term 'member' means an organ or a limb being part of man capable of performing a distinct function. It includes eyes, ears, nose, mouth, hands, feet, etc.

(5) Destruction or permanent impairing of the powers of any member or joint : In ordinary course of life, the use of limbs and joints of the body are very essential to the discharge of the normal functions of the body. Their deprivation causes lifelong crippling and makes the person defenceless and miserable. It causes great hardship to human body by its retention without normal function which is like amputation.

(6) Permanent disfiguration of the head or face : The word 'disfigure' in this section means to cause some external injuries which detracts from his personal appearance, but does not weaken him, e.g., cutting off a man's nose or ears. Branding a girl's cheeks which leaves permanent scars amounts to disfigurement.

"A cut on the bridge of the nose of a girl caused by a sharp weapon like razor or knife has been held to be permanent disfigurement even though the internal wall separating the nostrils was intact'.

(7) Fracture or dislocation of a bone or tooth: Fracture of either outer or inner table of the skull is considered a fracture. If a cut resulted only in a scratch or cut, and did not go deep to any length into the bone, it will not be fracture, otherwise it should be deemed to be one. It is not necessary that a bone should be cut through and through, or that there should be any displacement of any fragments of the bone. If there is a break by cutting or splintering of the bone, or there is a rupture or fissure in it, it would amount to a fracture. Dislocation means displacement. Mere looseness of teeth will not amount to dislocation. It has to be proved that the tooth was originally not loose, and that there was fracture or dislocation by the injury. Fracture or dislocation of a bone or tooth causes great pain and suffering to the injured person and is considered grievous hurt.

(8) Any hurt which endangers life, or which causes the victim to be in severe bodily pain, or unable to follow his ordinary pursuits for a period of 20 days : Some hurts which are not like those kinds of hurts which are mentioned in the first seven clauses, may be most serious. A wound may cause intense pain, prolonged disease, or lasting injury, but does not fall within any of seven clauses. A beating may not mutilate the sufferer or fracture his bones, but may be so cruel as to bring him to the point of death. The eighth clause provides for such hurts. Under this, three different

clauses of hurt are included. They are: (1) any hurt which endangers life, (2) any hurt which causes the victim to be in severe bodily pain for a period of 20 days, and (3) any hurt which prevents the victim from following his ordinary pursuits for a period of 20 days. The three clauses are independent of each other, and a hurt of any of the three clauses would be a grievous hurt.

Any hurt which endangers life : An injury can be said to endanger life, if it is itself such that it may put the life of the injured in danger. An injury caused on a vital part of the body cannot be called grievous hurt, unless the nature and dimensions of the injury or its effects are such that the doctor is of the opinion that it actually endangers the life of the victim. Administration of harmful drug to a person to make him unconscious is not grievous hurt, even though in some cases death may be caused if the drug is given in large dose. If the life of the person is not endangered, it is not a case of grievous hurt. To designate an injury as grievous hurt, danger to life should be imminent. Dangerous injuries are those which cause imminent danger to life, either by involvement of important organs or structures, or extensive area of the body. If no surgical aid is available, such injuries may prove fatal. The concept of injury dangerous to life is not very precise. As such, it is not enough if the medical witness makes simple statement that the injury in a particular part is dangerous to human life. He should place all relevant data, namely whether the injury caused haemorrhage or shock, affected important structures or organs, or was very extensive, or otherwise caused imminent danger, and should also state the various grounds on which he considers the injury to be a dangerous one. Some examples of injuries which endanger life are : stab on the abdomen or head or vital part, hurt causing rupture of spleen, squeezing testicles, incised wound on the neck, compound fracture of the skull, rupture of an internal organ, injury of a large blood vessel.

Any hurt which causes the victim to be in severe bodily pain, or unable to follow his ordinary pursuits for a period of 20 days : The length of time during which a sufferer is in pain, disease or incapacitated from pursuing his ordinary occupation is a defective criterion of the severity of a hurt. It is employed not only in cases where violence has been used, but also in cases where hurt has been caused without any assault, e.g., by the administration of drugs, the setting of traps, the digging of pitfalls, the placing of ropes across a road, etc. The extent of hurt and the intention of the offender are considered for giving punishment. It is difficult to prove that an injured person was in severe bodily pain for 20 days, but it is easier to prove that he was unable to follow his ordinary pursuits due to the hurt. The opinion of the doctor attending on the patient is important, on the point of his disability. A mere stay of 20 days in the hospital does not make injury grievous, unless the injured person was in severe bodily pain, or unable to follow his ordinary pursuits for a period of 20 days. Ordinarily, pursuits do not refer to the duties that constitute the job of a person. An injury which endangers life may cause severe bodily pain for 20 days, but an injury wich causes such pain may not be dangerous to life, but it is still a grievous hurt under this clause.

The line between culpable homicide not amounting to murder and grievous hurt is very thin. In the former, the injuries must be such as are likely to cause death, in the latter the injuries must be such as to endanger life. Where there is no intention to cause death, or no knowledge that death is likely to be caused from the harm inflicted and death is caused, the accused would be guilty of grievous hurt if the injury caused was of serious nature, but not of culpable homicide.

In case of hurt, it is the Court that must decide whether it is simple or grievous. **The duty of the medical witness is only to describe the facts and not to classify a hurt.** The entry made in the wound certificate as simple or grievous is only meant to guide the investigating officer.

ILLUSTRATIONS : (1) 'A woman was confined to a hospital for 17 days due to certain injuries inflicted on her, but her life was in danger for three days only. It was held that the injuries were grievous".

(2) "The thrusting of a lathi into the anus of a man has been held to be the causing of grievous hurt which endangers life".

(3) 'During an altercation, the victim was stabbed on the left forearm with a knife, due to which the radial artery was injured and the victim died soon after due to haemorrhage. It was held that the offence is neither murder, nor culpable homicide not amounting to murder, as the forearm is not a vital part. The accused was held to be guilty of voluntarily causing grievous hurt with a deadly weapon".

(4) "In case of injury to the abdomen, where intestines had come out of the wound, it was held to be grievous hurt".

"A deep injury which may be deep to the bone, but without a cut in the bone or fracture is not grievous hurt".

"Where death is caused as the result of an injury which is not intended to cause death, and was not in normal conditions likely to cause death, it is neither grievous hurt nor culpable homicide not amounting to murder.

Coma for 20 days due to head injury constitutes grievous injury.

Simple Injury: All injuries which are not grievous are simple.

S.321, I.P.C. : voluntarily causing hurt.

S.322, I.P.C.: Voluntarily causing grievous hurt.

S.323, I.P.C.: Punishment for voluntarily causing hurt : Imprisonment up to one year, or with fine up to one thousand rupees or both.

S.324, I.P.C.: Voluntarily causing hurt by dangerous weapons or means (3 years imprisonment).

S.325, I.P.C.: Punishment for voluntarily causing grievous hurt : Imprisonment for a term extending to seven years and also fine.

S.326, I.P.C.: Voluntarily causing grievous hurt by dangerous weapons or means : Imprisonment up to ten years and also fine.

Dangerous weapons or means include any instrument for shooting, stabbing or cutting or any instrument, which used as a weapon of offence, is likely to cause death; fire or any heated substance; poison or any corrosive substance; explosive substance or any substance which is harmful to the human body to inhale, to swallow, or to receive into the blood or by means of any animal.

S.340, I.P.C.:Assault: Wrongful confinement (imprisonment upto one year, S.342, I.P.C.)

S. 351, I.P.C.: An assault is an offer or threat or attempt to apply force to body of another in a hostile manner. It may be a common assault or with an intent to murder. S.352 to 358 I.P.C. deal with punishments for various types of assaults.

S. 362,I.P.C.: Abduction: Whoever by force compels, or by any deceitful means induces, any person to go from any place, is said to abduct that person.

S.299 to 377, I.P.C. deal with offences affecting the human body.

MULTIPLE METHODS OF SUICIDE: (1) A 42 year old woman was found with her head submerged in the blood-stained water of her bath tub. Her wrists, ankles and neck showed incised wounds. There was a star-shaped bullet wound on her forehead and at autopsy a 0.38 caliber bullet was recovered from the brain. In addition, she had consumed a large number of sleeping pills.

(2) A 40 year unemployed male took poison, tried to hang himself, stabbed himself with a knife and finally jumped out of seventh-storey window only to survive and land up in a hospital and police custody. He made all these attempts after stabbing his girl friend to death in a Bangkok hotel room in the morning, and writing on the wall with her blood. "If you think money is more important, we should die together".

DOWRY DEATHS

In some cases newly married girls are abused, harassed, cruelly treated and tortured by the husband, in-laws and their relatives for or in connection with any demand for dowry. In extreme cases, the woman is killed by burning or some other method.

Legal Aspects: S.304–B., I.P.C. (Dowry Death): Where the death of a woman is caused by any burn or bodily injury or occurs otherwise than under normal circumstances within seven years of her marriage and it is shown that soon before her death she was subjected to cruelty or harassment by her husband or any relative of her husband for, or in connection with, any demand for dowry, such death shall be called "dowry death", and such husband or relative shall be deemed to have caused her death.

S.498–A, I.P.C.: Whoever being the husband or the relatives of the husband of the woman, subjects such woman to cruelty shall be punished with imprisonment for a term which may extend to three years and shall also be liable to fine.

Cruelty has been defined as any wilful conduct which drives the woman to commit suicide or grave mental or physical injury to her or harassment of the woman with a view to coerce her for dowry.

S.113A and 113B, I.E.A., deal with presumption as to abetment of suicide by a married woman, and presumption as to dowry death.

Dowry deaths occur either by murder of a married woman or she herself committing suicide being unable to bear harassment or cruelty for not fulfilling the promises by her parents or her relatives or of those interested in her marriage. Such murders are invariably committed secretly either in the house or at a place where outsiders may not witness it. The bride may be burnt or killed by various methods. The usual defence in all dowry death cases is that either the woman committed suicide, or death occurred accidentally due to burns while cooking food.

The doctor should make a record of the history of the injuries and the persons responsible for it, when the victim is brought to the hospital, noting the time and the name of the person who gave the history. The condition of the victim should be recorded at frequent intervals. Care should be taken that no person has discussion with the victim before doctor records the statement of the victim to avoid any undue influence being exerted on her. A Magistrate may be called to record the dying declaration. Visit to the scene of crime by the doctor and other forensic scientists is very helpful in determining the manner of death.

Inquest should be conducted by a Magistrate or police officer not below the rank of deputy superintendent of police, and autopsy should be carried out by two doctors in case of dowry death, or if the age of the woman is less than 30 years, who dies in suspicious circumstances (Home Ministry circular).

WOUND CERTIFICATE: The casualty medical officer or any other medical officer may be called upon to examine injured person. Medico-legal injury cases should be examined without delay at any time of day or night. All details of examination of injured person, whether admitted into hospital or treated as outpatients have to be entered in an **Accident Register,** maintained in all Government hospitals. This register is a confidential record and should be in safe custody of the medical officer. It has to be produced in a Court of law if asked for. A detailed and accurate record should be made by the doctor at the time of the examination. The doctor is required to fill in a printed form of injury or wound certificate, one copy of which is sent to the investigating police officer in a sealed cover, and the other is retained for future reference. The following particulars should be noted. (1) Serial number. (2) Name, age and sex of the injured person and address. (3) Father's or guardian's name. (4) Date, time and place of examination. (5) Name and number of the accompanying police constable and the police station to which he belongs. (6) Names of the persons who had accompanied the injured person with their address. (7) A brief statement of the injured person as to how he was injured. This is to be recorded as "allaged by the patient". (8) Two identification marks. (9) The size of the victim, i.e., stature, body weight and development. (10) The consent of the person for examination should be taken. If the condition of the patient is serious, dying declaration should be recorded.

The following are the various entries in the wound certificate.

(1) Nature of each injury : All injuries observed, however insignificant they may appear should be noted. The nature of each injury, i.e., abrasion, contusion, incised wound, lacerated wound, stab wound, burns, fracture, dislocation, etc., should be noted. It may be convenient to deal with multiple injuries by grouping them according to their kind and severity. Alternatively, it may be better to group them anatomically, e.g., injuries of the head, of the trunk or of a limb. A lens should be used to get an accurate idea of the nature of the edges, ends and floor of a wound. The presence of any foreign material in the wound, e.g., metal, glass, hair, dirt, etc., should be noted.

(2) Size, shape and direction of each injury: All injuries should be measured with a tape and never guessed, and the amount of blood extravasated should be measured and photographs or sketches, showing the position and size of the wounds is desirable. The shape of the wound, e.g., circular, oval, triangular, elliptical, etc., should be noted and also the bevelling of the edges. The direction of the wound, i.e., horizontal, vertical, oblique, etc. should be noted with regard to the anatomical position of the body. In case of stab and firearm wounds, direction of the wound should be noted.

(3) On what part of body inflicted : The exact situation of wound with reference to some anatomical landmark, e.g., the middle line, a bony structure, a joint, the navel or the nipple should be mentioned. Technical terms should be avoided as far as possible.

(4) Simple, grievous and dangerous injury: Against each injury, it should be noted whether it is simple or grievous. The injured person must be kept under observation, if the nature of a particular injury cannot be made out at the time of examination, e.g., in head injury where the symptoms are obscure or abdominal injuries with vague symptoms. In all injuries, when a fracture of a bone is suspected, an X-ray examination should be done for confirmation. In such cases, the report should not be given hastily, if there is pressure from the police.

(5) By what weapon inflicted : In many cases, the examination of the wound and clothes give a fairly definite information about kind of weapon. With stabs and incised wounds there is not much

difficulty; wounds caused by broken glass may simulate either, but irregularity of the edges will be found in some parts of the wound, and if glass pieces are found it clears all doubts. Wounds made by blunt instruments, blows or kicks over bony prominences may simulate incised wounds, but an examination with hand lens helps the diagnosis. If medical officer is shown a weapon and asked, "Were the wounds caused by this weapon?" he should never answer in the direct affirmative, but should state that wounds could be caused by that weapon or were caused by similar weapon.

Any weapon sent by the police, which is alleged to have been used in producing injuries should be examined for marks of blood stains, hair, pieces of cloth, etc., adherent to it and should be returned to the police after it is sealed. A foreign body found in a wound, e.g., a piece of glass, a splinter of wood, the broken off point of a knife or the bullet, shots, or wadding of a firearm, may indicate the manner in which the wound was inflicted, and may even help to identify the weapon with which the injury was caused, e.g., the broken point of a knife with a missing point which it is alleged was the weapon used; or if the knife produced has a perfect point, it will be clear that it could not have been the weapon employed. A stabbing instrument is likely to break when it passes through the sternum or skull or enters a vertebra. The broken part remains within the body. Any foreign body found in the wound should be preserved. Presence of dust, coal or grease should be noted as they give a clue to the nature of injury.

The clothes should be examined for the presence of cuts, tears or burns, and it should be seen if these injuries correspond to the injuries on the body. If the clothing was very loose and was disarranged during the struggle or folded on itself, these may not coincide with the wounds.

(6) Whether the weapon was dangerous or not? A dangerous weapon is any instrument for shooting, stabbing or cutting or any instrument which used as a weapon of offence is likely to cause death.

(7) Remarks : The general condition of the patient should be noted under this column, such as conscious or unconscious, bleeding from the ears, nostrils, mouth, etc., paralysis, state of shock, etc. The age of the injury should also be noted. It is only possible to give an approximate time. The age of the injury is very important for its appearance may or may not correspond to the time when it is alleged to have been inflicted, and all the injuries found on a person may not have been produced on the same day.

A proper examination and documentation of a wound may reveal the nature of the wounding object, the direction of force, the approximate age of the wound, the character and manner of injury, etc. A careful search should be made for wounds in the concealed parts, e.g., axilla, nape of the neck, rectum, vagina, inner canthus of eye, etc.

Laboratory investigations recommended and treatment prescribed should be noted.

When a victim of suicide, homicide or accident dies in the hospital, the medical officer should report the matter to the police immediately. In civil cases, when a party wishes to have medical evidence given on their behalf, the medical officer is entitled to charge reasonable fee. In criminal cases, permission of the Government is required to give evidence for the defence. **When a dead body is brought to the hospital, do not examine the injuries.**

TORTURE

The World Medical Association (Declaration of Tokyo (1975), defined torture in relation to detention and imprisonment as "The deliberate, systematic or wanton infliction of physical or mental suffering by one or more persons acting alone or on the orders of any authority, to force another person to yield information, to make a confession or for any other reason".

S. 330, I.P.C.: Voluntarily causing hurt to extort confession, or to compel restoration of property is punishable with imprisonment up to 7 years.

S. 331, I.P.C.: Voluntarily causing grievous hurt to extract confession or to compel restoration of property is punishable with imprisonment up to ten years.

S. 339, I.P.C.: Wrongful restraint. Imprisonment up to one month (S.341, I.P.C).

The ethical aspects concerning the doctors with regard to torture are dealt in (1) Declaration of Geneva, 1948, states that no one shall be subject to torture or to cruel, inhuman or degrading treatment or punishment. (2) Declaration of Tokyo, 1975. (3) Principles of medical ethics relevant to the role of health personnel, particularly physicians in the protection of prisoners and detainees against torture and other cruel, inhuman or degrading treatment of

punishment (U.N. 1984). (4) World conference on human rights, 1993. Freedom from torture is a right which must be protected under all circumstances, including in times of internal or international disturbance or armed conflicts.

Torture may be carried out by (1) Criminal and terrorist groups, and (2) By the police or other security force personnel during the detention and interrogation of prisoners and suspects.

Objects: (1) To obtain information if a person is suspected to have committed a crime against person or property or indulged in antinational or terrorist activities. (2) To sign a document confessing a crime. (3) To take revenge against a person or his family members by rape, kidnapping, etc. (4) To obtain testimony incriminating others. (5) To spread terror in the community by militant groups or by dictators ruling the country. (6) To destroy the personality of individuals who raise their voices against dictatorial rule, or oppression in the society.

Methods: (I) Physical abuse: (1) Beating is the commonest form. Fist, foot or an instrument, such as *lathi*, metal or wooden bars, whips, belts, cycle chains, etc., are used. The usual target is the back, but injuries may be seen on almost any part of the body. Abrasions, contusions, lacerations and fractures may be found. Bruises in abdominal wall and rupture of internal organs may be seen. Poking with stick, baton, rod, etc. is common. (2) **Falanga** (falaka, bastinado): In this canes or rods are used to beat on the soles of the feet, which is very painful and debilitating. Contusions are found deep in the tissues. Faint red lines may be seen, and often hyperpigmentation along the lines of injury. Aseptic necrosis may occur. (3) **Telefono:** It consists of repeated slapping of the sides of the head (over ears) by the open palms of the assailant. This may cause rupture of ear drums. (4) **Beating on the abdomen** while lying on a table with the upper half of the body unsupported. In this bruises and rupture of viscera may be seen. (5) **Beating the head** may produce contusions and lacerations of the brain, intracranial haemorrhages and skull fractures, cerebral cortical atrophy, etc. (6) **Fingers are twisted** or pencils, etc., are kept between two fingers and sqeezed hard. (7) Making the victim **chew hard on pieces of metals,** stones, etc., or pulling teeth by clips or forceps. (8) **Mutilation:** Chopping of ears, nose, fingers, etc. (9) **Disfiguration** by throwing corrosives on face or other parts of body. (10) Use of continuous **high-pitched sound.** (11) **Forced immersion of head** often contaminated with urine or faeces, until the stage of suffocation **(wet submarino, latina).** Foreign material or debris is found in upper respiratory and upper GI tract. (12) Tying of a **plastic bag** over the head up to the point of suffocation **(dry submarino).** Asphyxial signs will be present. (13) **Suspension of body by the wrists.** Bruises or scars about the wrists and joint injuries are seen. (14) **Suspension of body by the arms or neck:** Bruises or scars at the site of binding. Prominent lividity in the lower limbs. (15) **Suspension by the ankles:** Bruises or scars about the ankles. Joint injuries. (16) **Head down** from a horizontal pole placed under the knees with the wrists bound to the ankle **(Parrot's perch, jack).** Bruises or scars on the anterior forearms and backs of the knees. Marks on the wrists and ankles. (17) The victim is asked to **stand in hot sun** on one leg for prolonged periods. Dependent oedema may be seen. (18) **Forced straddling** of a bar (saw horse): Perineal or scrotal haematoma. (19) **Burns** caused by cigarette butts is common. Other methods are application of heated solid body, molten rubber dripped on skin, kerosene soaked rags wrapped around limbs and set fire, hot iron, throwing boiling water, or acid, etc. (20) **Cold:** Victim is made to lie on a cold and damp floor or on an ice slab without clothing. (21) **Electric shock (cattle prod):** A magneto delivering high voltage may be used in which burns may not be found on the skin, but it causes severe pain. Sometimes, main voltage of 110 or 240 volts is used, which produces burns, and can cause death in some cases. The genitals especially the penis and scrotum are the targets, and in females nipples, but the current may be applied anywhere. (22) **Heated metal skewer inserted into the anus (black slave):** Perianal or rectal burns. (23) **Dehydratrion:** Vitreous humor electrolyte abnormalities. (24) **Animal bites.**

(II) Mental Torture: (1) Solitary confinement in a dark place. (2) Blindfolding for a long time, or frequent transfer from one place to another blindfolded. (3) Starving the victim. (4) Causing mental anguish by giving false information to victim regarding tragedy involving wife and children. (5) Enforced use of psychotropic drugs.

(III) Sexual torture: (1) Infliction of injuries to private parts or introducing foreign bodies into the rectum or vagina, or mutilation of breasts or

genitals. (2) Raping the victim or undressing before others, or sexually tortured by trained animals, etc.

Sequelae: Infection, disfiguration of face, impairment of hearing and sight, psychic disturbances, and social stigma. Death may occur rarely.

The doctor should obtain complete history from the patient and examine scene of death. The whole body should be carefully examined and injuries evaluated.

Treatment: The principles of treatment are: (1) Developing a good rapport with the victim. (2) Empathising with the victim and his family. (3) Avoiding situation or objects that remind the victim of the torture event. (4) Psychotherapy.

DEATH IN CUSTODY

Death occurring in some form of custodial detention is commonly known as death in custody, such as police cell or prison. It should also include deaths resulting from police or prison officers attempting to detain a criminal or a person escaping or attempting to escape from police custody or prison.

(I) DURING ARREST:Traumatic asphyxia may occur if the offender resists arrest, and a number of policemen fall upon him to overpower him. If there is a struggle, blunt injuries sustained by the offender by the use of fist, arm or leg, riot stick, etc., by the police or from head injury from falls against ground, wall, etc., may cause death.

(B) CHOKE OR CAROTID HOLDS: A choke (bar arm control) or a carotid sleeper hold, do not involve the use of a mechanical implement.

(I) IN CHOKE (BAR ARM) HOLDS forearm is placed straight across the front of the neck. The free hand grips the wrist and pulls it back, which causes obstruction of the airway and carotid arteries and immediate unconsciousness. The hypopharynx is also occluded by displacement of the tongue. Fracture of the hyoid bone or larynx may occur if great force is used. Rarely death may occur due to hypoxia and release of catecholamines due to struggle, resulting in fatal cardiac arrhythmia, especially if the deceased consumed cocaine or similar drug.

(2) In the CAROTID SLEEPER HOLD, the arm is placed about the neck with the antecubital fossa centered at the midline of the neck. The free hand grips the wrist and the arm is pulled backwards, which compresses the carotid arteries and jugular veins but not vertebral arteries and the trachea. Consciousness is lost in 10 to 15 seconds. If pressure is released the victim regains consciousness in 10 to 20 seconds. The neck structures are not damaged. Rarely death may occur due to hypoxia and release of catecholamines.

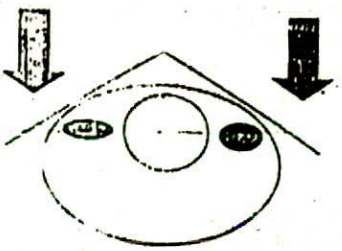


Fig. (10–1). Carotid sleeper, direction of force on neck aimed at compressing the carotid arteries.

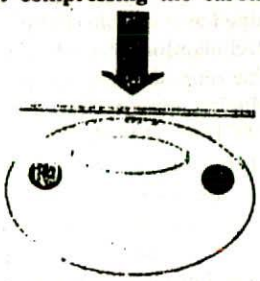


Fig. (10–2). Carotid hold. Force applied across the front of neck compressing the airway.

Most deaths occur immediately after an arrest in which there is a violent struggle. Some deaths due to coronary artery disease occur during the struggle.

(II) DURING LOCK-UP: If the offender was severely intoxicated, he may die of acute alcoholic poisoning, or he may aspirate the vomit and die of choking. The drunken offender may fall on a hard surface on the occiput. If on autopsy contrecoup injuries are found, it indicates deceleration injury rather than an assault with a weapon. Physical injuries may also occur from falls in drug addicts used to amphetamine, cocaine or hallucinogens. Torture by kicking and stomping, or a backward blow from the point of an elbow on the face, neck, or abdomen, fist blows, etc., inflicted with sufficient force can cause severe damage and death.

SUICIDE in custody is common. The common method is hanging. Sometimes a poison may be consumed, or he may jump from a height, or cut his throat or a large blood vessel.

Death may occur due to natural causes, usually cardiovascular. The emotional upset may precipitate an acute cardiac crisis in the presence of severe pre-existing disease.

SUDDEN DEATH DURING OR IMMEDIATELY AFTER A VIOLENT STRUGGLE:

A complete history should be obtained including medical records of the deceased. A complete autopsy

should be performed and microscopic examination of all organs especially the heart and complete toxicological examination should be done.

(1) EXCITED DELIRIUM: Following cessation of a violent struggle, the levels of catecholamines continue to increase for about 3 minutes, while the level of potassium drops considerably. These two factors predispose to the development of an arrhythmia. This is the time of "post exercise peril". Stimulants such as cocaine and methamphetamine can cause excited delirium. If a violent struggle occurs a fatal arrhythmia occurs. Cocaine has a double effect. It causes increased release of catecholamines from the adrenals and inhibits norepinephrine reuptake due to which coronary arteries contract, reducing myocardial perfusion.

(2) CATECHOLAMINE RELEASE: During high intensity exercise, e.g. a struggle, catecholamines are released from the adrenals into the circulation, the highest levels occur about 3 minutes after cessation of the activity. These substances increase the rate and force of contraction of the heart (due to which there is an increased demand for oxygen by the myocardium), the conduction velocity and the blood pressure. If the person suffers from heart disease, a focal area of myocardial ischaemia develops with fatal cardiac arrhythmia.

If the heart is predisposed to fatal arrhythmias by the action of alchol then, under conditions such as a violent struggle, the released catecholamines can produce a fatal arrhythmia.

In some cases, sudden death during or following struggle can be caused by natural disease that is not diagnosable anatomically, such as Wolfe-Parkinson-White syndrome, the prolonged Q-T syndrome, etc.

In such cases, the cause of death can be given as "cardiopulmonary arrest during violent struggle in individual under influence of alcohol, cocaine, etc".

Sudden cardiac death can also occur due to psychological stress, due to fatal or potentially fatal cardiac arrhythmias with and without underlying heart disease. This is caused by stress-induced sympathetic stimulation.

AUTOPSY: A visit to the scene of death by the doctor especially in case of hanging or mechanical traume is often useful. Detailed medical history should be obtained. A complete autopsy is essential. Diseases that can cause sudden and unexpected death, such as cardiovascular diseases, epilepsy, asthma, diabetes, etc., should be sought for. Blood should be examined for alcohol in cases where the offender has consumed alcohol.

Some methods of physical abuse may not leave any apparent marks of violence, such as hair-pulling, face slapping and blows to the side of the head which may cause perforation of the eardrum. Blows to the abdomen may not cause any apparent mark. Pinching, squeezing and blows to the testes may produce bruising of the skin and swelling and tenderness of testes. The soles may be struck with a baton, ruler, etc.

CAUSES OF DEATH FROM WOUNDS

The causes of death from the wounds are: (1) Immediate or direct. (2) Remote, delayed or indirect.

IMMEDIATE CAUSES : (1) Haemorrhage : Haemorrhage means escape of blood from the cardiovascular system.

Types: Haemorrhage is classified as capillary, venous, arterial or cardiac, depending upon its origin. Haemorrhage may be external or internal. When bleeding occurs due to various types of wounds, it is called traumatic haemorrhage. Bleeding occurring in the absence of trauma is called spontaneous haemorrhage. Petechiae are minute haemorrhagic spots, usually of capillary or venular origin. Ecchymoses are larger areas of extravasated blood. A haematoma is localised collection of blood, usually clotted, forming a tumour-like swelling in a tissue. Apoplexy is large effusion of blood into an organ, but the term is commonly used for cerebral haemorrhage.

The loss of blood from the circulation may be due to wounds of the heart, or of the larger blood vessels, or to a severe laceration of an organ or structure, which is very vascular. The haemorrhage may occur at the time of injury (primary haemorrhage). Secondary haemorrhage occurs from the same site as primary haemorrhage but is usually delayed for several hours (up to 24 hours or more) after the injury. It may occur due to a rise in blood pressure (which accompanies the recovery from shock), and muscular movements, which loosen the blood clot, and the erosion of vessel walls which occur in infection.

(a) EXTERNAL HAEMORRHAGE: It may produce syncope and death either rapidly, if a large vessel has been injured, or slowly if a number of small vessels have been damaged. The sudden loss of blood is more dangerous than the same quantity lost slowly. A minor injury may produce death from haemorrhage in persons with haemorrhagic diathesis or haemophilia. An approximate idea of loss of blood may be had from the soaking of clothes, and the surroundings of the victim, and from the anaemic state of the viscera.

(b) INTERNAL HAEMORRHAGE: It may occur in penetrating wounds, gunshot wounds, and due to

rupture of organs or vessels. The blood escapes from the divided vessels, either internally or externally as it remains fluid even after death. Blood in the pleural cavity does not clot, because of rapid defibrination due to movements of the lung.

Wounds can continue to bleed copiously after death due to passive leakage, especially if the injured part of the body is dependent, i.e., lower than the rest of the body. Blood can also accumulate in large quantity within the pleural and peritoneal cavities after death.

MECHANISM OF DEATH : (1) The rapid loss of one-third of the blood of the body causes death due to irreversible hypovolaemic shock. Due to severe fall in blood pressure, cerebral ischaemia and death from cerebral anoxia occurs. Children, women and old people die from the loss of much smaller quantity. (2) Haemorrhage can kill rapidly creating increased pressure in or on a vital organ which interferes with its vital function, e.g., an effusion of 300 to 400 ml. of blood into the pericardial sac interferes with the heart's action (cardiac tamponade); a small haemorrhage into the medulla will fatally interrupt the functions of the vital centres there; an intracranial haemorrhage may cause death from cerebral compression. (3) Bleeding into the trachea or bronchi can cause rapid death from asphyxia due to the blood acting as a mechanical barrier to respiratory efforts, which cause asphyxia. The victim literally drowns in his own blood.

AUTOPSY: The pathologic findings in death from haemorrhage are pallor of the skin and mucous membranes, and of all viscera (especially renal cortex), and a small amount of blood in the blood vessels. In the typical case, the heart is contracted and shows streaky minute subendocardial haemorrhages on the left side of ventricular septum, and papillary muscle.

(2) Reflex Vagal Inhibition: In this, there may not be visible external injury.

(3) SHOCK : Shock is a circulatory disturbance characterised by hypoperfusion of cells and tissues due to reduction in the volume of blood or cardiac output, or redistribution of blood resulting in a decrease of effective circulating volume.

Primary Shock: Primary or neurogenic shock (vaso-vagal shock or reflex cardiac arrest) results from a sudden reduction of venous return to the heart due to neurogenic vasodilation with pooling of blood in the peripheral vascular bed, especially in the subclavian area. There is yawning, sighing respirations, nausea and vomiting followed by unconsciousness, but the attack rarely lasts for more than a few minutes. There is pallor, coldness of the extremities, weak rapid pulse and low blood pressure. The cardiac output is unchanged. It may follow any from of stress. Psychological factors, such as fear, grief, anxiety, emotion and pain due to various causes also play a large part. It may occur when a few ml. of blood are withdrawn, or from the sight of blood or an anticipation of injection.

ACUTE NEUROGENIC CARDIOVASCULAR FAILURE: The sympathetico-adrenal division stimulates the cardiovascular system to meet the demands of the body in physical and emotional stress, especially in an emergency. The parasympathetic division has a tonic and inhibitory control on the cardiovascular system. These two divisions act together and provide a balanced control to meet the varying needs of the body. There is much individual variation and in some persons constitutional imbalance of the autonomic system seems to be present, with increased sympathetic or parasympathetic tone.

Sympathetico-adrenal stimulation occurs in emotional disturbances, such as fear, especially when accompanied by painful sensations. The peripheral nerves convey both pressor and depressor afferent fibres. Stimulation of the pressor fibres causes generalised vasoconstriction with a rise in blood pressure. Stimulation of the depressor fibres cause generalised vasodilation with a fall in blood pressure. Weak stimuli of afferent nerve fibres cause depressor effects and stronger stimuli cause pressor effects. Painful stimuli are associated with pressor effects. Strong and painful stimuli excite pressor afferent fibres of peripheral nerve. The sympathetico-adrenal division is stimulated by physical exertion, cerebral anaemia, hypoglycaemia, low oxygen tension or a high CO_2 tension in the blood. The stimulation is caused both by direct action on the medullary centres and by reflex action through the chemoreceptors of the aortic and carotid bodies. Sympathetico-adrenal stimulation results in rapid pulse, increase in myocardial contractility and irritability, extrasystoles and generalised vasoconstriction with a rise in blood pressure, and may lead to sudden, fatal circulatory failure from ventricular fibrillation, Hypotension resulting from pain or fear, from loss of effective circulating blood volume, drugs or reflex action will cause hypoxia. Hypoxia from a low crdiac output or respiratory inadequacy will render the ventricular cells more sensitive to catecholamine release and lead to ventricular fibrillation. Circulatory failure due to ventricular fibrillation usually occurs after several minutes.

PATHOLOGY: There may be some pre-existing cardiac disease, such as coronary artery sclerosis, cardiac hypertrophy or fibrosis, fatty myocardium, chronic valvular disease (esp. aortic valves), or syphilitic atresia of the coronary orifices. Signs of earlier chronic congestive failure, i.e., chronic passive congestion of organs and dependent oedema may be present. Pulmonary congestion and oedema are present and may be marked. Pleurae show petechial haemorrhages, which may be very numerous.

DIAGNOSIS: The possibility of the neurogenic cardiovascular failure should be considered when a healthy person dies suddenly without any adequate cause. The cause of death as ventricular fibrillation precipitated by reflex sympathetico-adrenal stimulation may be inferred, if some pre-existing cardiac lesion is found at autopsy, if the fatal symptoms were those of a hyperacute congestive cardiac failure (with acute dyspnoea and cyanosis), and if acute congestive visceral changes are present.

Secondary Shock : It is more serious and may be fatal. There is progressive circulatory failure and damage to the tissues. It is called secondary or delayed shock, because the circulatory disturbance usually develops gradually or after a latent period following the injury. Apart from haemodynamic alterations, there are disturbances in the molecules of cells due to diminished perfusion of the tissues. Intracellular metabolism is disturbed with anaerobic glucose metabolism and increased production of lactic and other acids, which may cause disruption of lysosome, release of their lytic enzymes and death of cells. The production of adenosinetriphosphate is decreased, which results in a reduction of protein synthesis, and disturbance in the transport mechanism and cell membrane permeability. Associated with this is a reduction in oxygen consumption, interference with enzyme system, metabolic acidosis, water and electrolyte disturbance and renal insufficiency.

PATHOGENESIS: Reduction of blood flow with poor cellular perfusion (anoxia) is the essential feature of shock. Three basic pathophysiologic derangements are: (1) reduction of actual circulating blood volume, (2) decreased cardiac output, and (3) widespread vasodilation of the capacitance vessels (capillaries and veins).

Shock resulting from diminution of actual blood volume is commonly called hypovolaemic or oligaemic shock. It may be caused by (1) local loss of blood or plasma at the site of wound, burns or operation, (2) loss of fluid by vomiting, diarrhoea, adrenal insufficiency, etc., as in dehydration, (3) escape of fluid into interstitial compartment due to generalised increased vascular permeability, (4) vasodilation in peripheral vascular bed, due to which blood is withdrawn into these vessels from general circulation, and (5) vasoconstriction of, or thrombosis in peripheral vascular bed which obstructs flow of blood, or post-capillary vasoconstriction resulting in pooling and stasis of blood in capillary beds. Hypovolaemia underlies or contributes to haemorrhagic, burn, traumatic and surgical shock. Secondary humoral and neural mechanisms produce vasoconstrictions in the arterioles and increased peripheral resistance. These compensatory changes are brought about by increased levels of circulating catecholamines, angiotensin, cortisol and aldosterone. Another compensatory mechanism is the tendency towards restoration of blood volume with improvement of venous return to the heart. The factors assisting this are (1) secretion of aldosterone and antidiuretic hormone, and (2) widespread arteriolar constriction. The central venous pressure, cardiac output and blood pressure, are usually low in hypovolaemic shock.

An ineffective cardiac pump produces vascular collapse in cardiogenic shock. The basic derangement

Table (10–1) Classification of shock

Types of shock	Clinical examples	Principal mechanisms
Cardiogenic:	**Myocardial infarction, rupture of heart, arrhythmias, cardiac tamponade, pulmonary embolism.**	**Failure of myocardial pump due to intrinsic myocardial damage or extrinsic pressure or obstruction to outflow.**
Hypovolaemic:	**Haemorrhage, fluid loss (e.g., vomiting, diarrhoea, burns).**	**Inadequate blood or plasma volume.**
Septic:	**Overwhelming bacterial infections:**	**Peripheral vasodilatation and pooling of**
blood;		
	Gram-negative septicaemia ("endotoxic shock") or Gram-positive septicaemia.	**cell membrane injury, endothelial cell injury with DIC.**
Neurogenic:	**Anaesthesia, spinal cord injury.**	**Peripheral vasodilatation with pooling of blood.**

is a decreased supply of blood to the tissues and venous return to the heart. It may be caused by (1) deficiency of filling, e.g., cardiac tamponade, and (2) deficiency in emptying, e.g. myocardial insufficiency (as in myocardial infarction), or mechanical obstructions (as in massive pulmonary embolism, ball-valve thrombus, etc.), and surgical or spontaneous damage to the cardiac valves. With failure of the heart, the central venous pressure rises. Compensatory neurogenic and endocrine responses lead to arteriolar vasoconstriction and high peripheral resistance. Initially the blood volume is usually normal, but later the peripheral tissue anoxia produces vasodilation and increased vascular permeability, leading to loss of fluid and electrolytes into the tissues.

Most of the cases of septic shock are caused by endotoxin-producing, Gram-negative bacilli, such as E.Coli, Klebsiella pneumoniae, Proteus, Pseudomonas aeruginosa, and Bacteroides. Pneumococci and Streptococci may produce a similar syndrome. In septic shock, the cardiac output is not low in the initial stages, but the total peripheral resistance is low due to arteriolar vasodilatation, which results from release of vasoactive mediators, such as complement components, but direct toxic injury to the vessels may also occur. Peripheral pooling of blood occurs. A relative hypovolaemia is produced due to the disproportion between the circulating blood volume and the expanded volume of the circulatory bed, due to which perfusion is impaired. Peripheral pooling of blood causes impairment of venous return leading to low cardiac output. Bacterial toxins cause endothelial damage and activate intrinsic and extrinsic clotting pathways, leading to disseminated intravascular coagulation, which further increases tissue anoxia. Activation of complement produces many factors, including those that increase vascular permeability and attract neutrophils. Accumulation of neutrophils in tissues, especially in the lung, release their enzymes which produce toxic free radicals, due to which pulmonary epithelium is damaged and produces acute respiratory distress syndrome (ARD). Endotoxins activate mononuclear phagocyte system and release TNF-a. This cytokine synthesises and releases a number of secondary mediators including prostaglandins and platelet-activating factors. TNF-a also promotes intravascular coagulation and capillary thrombosis. Bacterial toxins also cause direct injury to cells and tissues.

Stages of Shock: **(1) Nonprogressive phase:** In this reflex compensatory mechanisms are activated and perfusion of vital organs is not affected. **(2) Progressive state:** In this tissue perfusion is reduced with increasing circulatory and metabolic imbalances. **(3) Irreversible stage:** This occurs after severe injury to the cells and tissues.

During early shock a number of neurohumoral mechanisms, such as baroreceptor reflexes, catecholamines, antidiuretic hormone, activation of renin-angiotensin axis, and generalised sympathetic stimulation maintain cardiac output and blood pressure. There is tachycardia, peripheral vasoconstriction and conservation of fluid by the kidneys. During the progressive phase, vital organs suffer from significant hypoxia. Hypoxia causes impairment of intracellular aerobic respiration, followed by anaerobic glycolysis and excessive production of lactate, which causes metabolic lactic acidosis. Due to lowering of pH in the tissues, arterioles dilate and blood begins to pool in the capillaries. This causes diminished cardiac output, and anoxic injury to endothelial cells, leading to disseminated intravascular coagulation. When the tissue hypoxia is widespread, the function of vital organs deteriorates. The patient is confused and the urinary output begins to fall.

Lysosomal enyzmes leak due to widespread cell injury which increases the shock. Endotoxic shock may be superimposed.

Types: Shock can be classified on the basis of the stressful circumstances under which it occurs. **(1) Haemorrhagic shock:** This occurs when the blood loss is sufficient to impair peripheral perfusion, which produces a sufficient degree of systemic anoxia. Loss of 10 to 40 per cent of the total blood volume produces shock. **(2) Traumatic or wound shock:** This is the most common type of shock. The blood volume may be reduced due to haemorrhage from ruptured vessels, an excessive loss of plasma from damaged capillaries or venules at the site of injury, or a combination of both factors. In some cases, blood loss is not significant and the shock may be due to neurogenic mechanisms. It has also been suggested that the major factor is absorption of products of tissue autolysis from the area of injury, which causes a generalised increased vascular permeability. Infection of the wound and bacterial endotoxins may also play a part. **(3) Burn shock:** It results from loss of plasma, resorption of necrotic tissue and bacterial sepsis following extensive burns. **(4) Surgical shock:** It occurs either during or immediately after surgical procedures. It may be due to a combination of causes; anaesthesia, loss of blood and plasma, emotional reactions, infections and probably liberation of substances from severely

traumatised tissues that may influence the circulation. **(5) Cardiac or cardiogenic shock:** It results from the decreased cardiac output, reduced blood flow and deficient delivery of oxygen to the tissues following injury to the pumping mechanism. **(6) Septic shock:** It develops in the presence of severe infection. Loss of fluid from the circulation (generalised increased vascular permeability), or pooling of the blood in the peripheral vascular bed (vasodilation) cause circulatory failure. **(7) Endotoxic shock:** This is closely related if not identical to septic shock. It is seen in patients having Gram-negative infections. Bacterial endotoxins induce intravascular coagulation prior to endothelial damage.

Surgical shock does not occur in head injury unless it is extremely severe, or there has been excessive blood loss from scalp wound, other injury to trunk or limbs.

Post-mortem Appearances: The findings in secondary shock are circulatory changes, and degeneration and necrosis in various organs. The circulatory changes include congestion of all the internal organs, petechial haemorrhages in the serous cavities and oedema of the viscera.

Brain: The brain may develop ischaemic encephalopathy. The changes occur if the person survives for 12 to 24 hours. At first there is swelling or shrinkage of neurons. Later the nucleus becomes pyknotic. Later, nerve cells die and are replaced by fibrillary gliosis.

Kidneys : The renal changes are those of acute tubular necrosis. The renal changes begin within 24 hours, and become marked in another 7 to 10 days. The kidneys are normal in size, but in some cases they may be slightly enlarged.

Lungs : They are not affected in hypovolaemic shock. When vascular collapse is caused by bacterial sepsis or trauma **"shock lung"** is seen. The lungs resemble liver, are dark red, firm, airless and heavy. There is capillary congestion, interstitial and intra-alveolar oedema and haemorrhage, and necrosis of alveolar epithelial cells. The capillaries contain an accumulation of leucocytes, clumps of platelets and often microthrombi. In some cases, secondary or terminal pneumonia may be superimposed.

Heart : Fatty change occurs in about 20 per cent cases after 18 hours and becomes well-marked in 3 to 4 days. Subendocardial haemorrhages and necrosis sometimes appear in all forms of shock.

Gastrointestinal tract: Patchy mucosal haemorrhages and necroses ("haemorrhagic enteropathy") may be seen. The mucosa of the gastrointestinal tract is often congested and oedematous, and may show haemorrhage, erosions or ulcerations. The intestinal tract may contain blood-stained fluid. The blood is fluid and dark–red.

Liver : Gross appearance of the liver is not altered. Fatty changes develop after 18 hours, which may extend from the central areas to lobules. Central necrosis occurs with severe perfusion deficits.

Spleen: In septicaemia, there may be enlargement and softening of the spleen and non-specific toxic signs in other organs such as the heart and liver.

Adrenal : Focal lipid depletion of the cortex occurs after 24 hours and is well marked by third or fourth day.

Foci of necrosis may be present in the lymph nodes, spleen and pancreas.

Mental (nervous) shock: It is the mental state resulting from an unpleasant experience, which may be physical or psychic.

(4) Mechanical Injury to a Vital Organ : Severe injury to a vital organ, such as crushing of brain, heart, etc. is rapidly fatal.

Death may occur from a slight injury inflicted on a previously diseased organ, e.g., rupture of enlarged spleen, perforation of a chronic intestinal ulcer; bursting of an aneurysm, etc. In such cases, the assailant cannot be charged with culpable homicide but is convicted of simple or grievous hurt, if it is proved that his intention was not to kill his victim, that he could not possibly have known the existence of disease, and that the same injury could not have proved fatal when inflicted on normal healthy individual.

REMOTE CAUSES : Though the injury does not directly cause death, the victim may die after a varying period from remote causes. Even when the victim dies of complications, an assailant is responsible, provided death can be traced to likely result of the injury. In a charge of murder, death should be clearly connected with the injury. The assailant is responsible even if the victim was in a bad state of health when attacked, and if the injury inflicted by the accused accelerated or precipitated the death.

(1) Infection : Wound infection may be caused by : (1) Organisms normally present on the body surfaces, e.g., Streptococci, Staphylococci, B.pyocyaneus, Coliform bacilli, Freidlander's bacilli, and Cl.welchii. (2) Organisms invading the tissues

from the environment, e.g., Streptococci, Staphylococci, C.diphtheriae, Coliform organisms, Cl.welchii and Cl.tetani. **Primary wound infection** is caused by organisms, which are carried into the wounds at the time of the injury, e.g., from the skin, clothing, dirt, etc. **Secondary wound infection** is caused by organisms which invade the wound after injury, e.g., by airborne droplet infection, contaminated dressings, etc.

(a) DIRECT INFECTION : Infection at the site of an open wound, such as a stab wound, or a gunshot wound, and fracture of anterior cranial fossa communicating with an air sinus may cause meningitis. (b) REMOTE INFECTION : Uterine infection following upon criminal abortion may cause meningitis. (c) TRAUMA : At the site of infection, such as bruising of a pre-existing staphylococcal lesion of the bone may produce septicaemia or pyaemia. (d) Infection may be due to an indirect result of injury which creates a local area of lessened resistance, e.g., abscess may form around simple fractures of bone, contusion, or in traumatic infarcts of liver, kidneys and spleen. Dead tissue and extravasated blood provide favourable environment to bacterial growth. (e) If the general vitality is depressed, hypostatic pneumonia, pyelonephritis, cystitis, decubital ulcers, etc. may develop.

Pyogenic cocci may produce a localised abscess (usually in staphylococcal infections) or a diffuse spreading cellulitis (usually in streptococcal infection). B.proteus, B.pyocyaneus and B.coli produce suppurative lesions in association with pyogenic cocci. In clostridium cellulitis, the infection is confined to the connective tissues which produce gas, but the muscles are affected. In gas gangrene there is a massive and rapid necrosis of muscle tissue which causes severe toxaemia.

In septicaemia, there may be enlargement and softening of the spleen, and non-specific toxic signs in other organs, such as the heart and liver.

(2) Gangrene or Necrosis : Gangrene is death of a part accompanied by putrefaction. It results from severe crushing of part and tearing of blood vessels.

(3) Crush Syndrome : Traumatic tubular necrosis may occur in cases of trauma to the limbs in which necrosis of the muscles occur, which may be due to ischaemia of direct compression. It results from severe crushing of the muscles, especially those that involve the lower limbs, e.g., under fallen masonry, industrial and vehicular accident, etc. In traffic accidents, necrosis of muscle may occur without any prolonged crushing injury. Death usually occurs in one to two weeks after the injury. Crush syndrome is also seen in extensive burns and certain poisons, e.g. mercuric salts or carbontetrachloride. The cause is obscure, but may involve the juxtaglomerular apparatus and the renin angiotensin system, and disseminated intravascular coagulation effects on the glomeruli. There are no specific gross changes in the kidney. Microscopic examination reveals degeneration and necrosis of focal portions of distal part of Henle's loop and the distal convoluted tubules. Cellular infiltration is seen in the interstitial tissues around the degenerated and necrosed tubules. The distal tubular segments and the collecting tubules contain casts of haeme compound. The glomeruli and proximal convoluted tubules appear normal.

(4) Neglect of Injured Person : Death may occur from complications arising from a simple injury due to improper treatment, or negligence on the part of the doctor, or to negligence or wilful disobedience on the part of the patient.

(5) Surgical Operation : The assaulted person is not bound to submit himself for operation. If death occurs due to this omission, the assailant becomes responsible. If death follows a surgical operation performed for the treatment of an injury, the assailant is responsible for the result, if it is proved that the victim would have died even without the operation, and that the operation was thought necessary and was performed by a competent surgeon with reasonable care and skill. A doctor cannot swear that an operation would have saved the life of the victim. He can only affirm that it might have provided the victim a better prospect of recovery. When anaesthesia is given to an injured person before operation, and if the person dies during its administration, and if it is agreed that the patient would not have died but for its administration, the person causing injury is liable to be punished for culpable homicide. If hurt or wound is not fatal, and if it is clearly proved that death of the victim is caused by application of harmful medicines by himself or those about him, this cannot be regarded as murder. An operation on an assaulted person should not be undertaken unless it is clearly indicated and never without consultation with another surgeon.

(6) Natural Disease : Some natural disease may be present which was the actual cause of death, but in which death was accelerated by assault, e.g., persons suffering from miliary aneurysms, fatty

degeneration of the heart , etc. may die due to slight violence, which may be a precipitating cause.

(7) Supervention of Disease from a Traumatic Lesion : Fibrous scar tissue formed due to healing may contract and produce complications, e.g., fibrous scar in a hollow muscular organ may produce stricture and obstruction. If a fibrous scar is under continuous tension, it may stretch and produce complication, e.g., a fibrous scar in the wall of an artery may bulge into a traumatic aneurysm. Damage of an artery by a bullet may produce false aneurysm, which may rupture later. Direct injury to the coronary artery by thoracic trauma may be followed by coronary thrombosis. A wound of the abdominal wall after healing may be followed by strangulated hernia with fatal result. An injury affecting lower portion of spinal column or cord may cause paraplegia which may cause death from septic cystitis or bed sores and general exhaustion.

(8) Thrombosis and Embolism : The most common sites of thrombosis are in deep femoral, the popliteal and posterior tibial veins, and occurs usually in traumatic lesions of lower extremity, especially fractures of long bones. The factors which predispose to leg vein thrombosis after an injury are: (1) Local tissue damage, causing injury to veins. (2) An increase in clotting time of blood which is maximum at about two weeks after injury. (3) Immobility and bed rest. (4) General debility, especially in the old, leading to poor general circulation and cardiac output. The thrombus usually develops in 10 to 20 days and is detached in part or whole and travels to pulmonary artery. Other veins, such as the venous sinuses of dura mater or subclavian or axillary veins, external and internal iliac veins, prostatic plexus, uterine and ovarian veins, occasionally develop bland thrombi after traumatic injury. Arterial thrombosis and embolism is rare. Bullet wounds and stab wounds of the carotid arteries may injure the intima and a thrombus may develop at the site of the injury followed by embolism of middle cerebral arteries.

A large embolus in the pulmonary trunk often produces a noticeable fullness before the vessel is opened. The embolus which blocks the pulmonary trunk causes death in few minutes due to vagal inhibition, acute asphyxia or right-sided heart failure, and no infarction is found in the lungs. Infarction of lung is seen when the artery to a bronchopulmonary segment or smaller vessel is blocked. Pulmonary emboli should be differentiated from post-mortem clots. Pulmonary emboli are : (1) cylindrical in shape with parallel contours, (2) often branch and frequently curled, (3) their shape does not correspond to the shape of the vessels in which they are found.

When a large embolus obstructs the bifurcation of the pulmonary artery or one of its main branches, an immediate acute dilatation and failure of the right side of the heart occurs (acute cor pulmonale). When smaller branches of the pulmonary arteries are obstructed, generalised vasospasm occurs and causes sudden sharp increase in blood pressure in the lesser circuit with marked dilation and over-distension of the right ventricle. Systemic shock occurs with peripheral hypotension and interferes with coronary flow. The entire complex causes immediate stoppage of vital functions.

Unusual types of pulmonary embolism are sometimes produced by trauma. Fragments of bone marrow may be found as emboli in smaller branches of the pulmonary artery, usually following fracture of bones containing marrow. Fragments of liver parenchyma may enter torn hepatic veins after blunt force injury to liver and may be carried as emboli. Laceration of brain accompanied by tearing of venous sinuses may lead to pulmonary embolus. Foreign bodies may enter veins or arteries and are transported as emboli, e.g., bullet.

(9) Fat Embolism : Fat embolism is caused by (1) fracture of a long bone, (2) an injury to adipose tissue which forces liquid fat into the damaged blood vessels, (3) injecting oil into circulation, e.g., in criminal abortion, (4) occasionally due to natural disease without trauma as in sickle cell anaemia. Certain non-traumatic conditions, e.g., diabetes, following blood transfusion, fatty change in the liver as seen in chronic alcoholics, septicaemia, steroid therapy, acute pancreatitis, osteomyelitis, decompression sickness (Caisson's disease), in too rapid ascent to high altitudes without a functional pressurised cabin or under simulated flight conditions in decompression chambers, (5) in cases of severe antemortem burns. In every injury involving bone or subcutaneous tissue, fat embolism occurs to some degree, especially crushing of buttocks or breasts. Pulmonary fat embolism unless it is gross enough to cause ventilatory embarrassment by widespread plugging of capillaries has no significance.

The presence of fat droplets in the blood stream indicates that the injury was produced during life, except in case of burning and advanced putrefaction.

Charring after death may produce a picture similar to fat embolism. Large amounts of liquefied fat are derived from fat cells in the tissue in putrefied body. When cardiac massage is done, fat enters the blood vessels even if the circulation has stopped. If during resuscitation, the sternum or ribs are fractured, bone marrow embolism is seen in the lungs.

ORIGIN OF EMBOLI : (1) MECHANICAL THEORY : Fat emboli are produced as follows : (A) Trauma or fracture destroys fat cells and liberates liquid fat. (B) The trauma tears some veins in the vicinity of the fractures. (C) Pressure exerted on liquefied fat due to muscular spasm and swelling of injured soft tissue forces liquid fat into veins. Improper fixation or rough manipulation of fracture may favour this process. (2) PHYSICO-CHEMICAL THEORY : The major part of intravascular fat droplets in the lungs is derived from lipids normally present in the plasma. Following trauma, alteration of the normal plasma colloidal dispersion occurs which causes fusion of chylomicrons to form larger fat droplets. Fat emboli may be present in all cases of trauma if looked for by microscopy.

The emboli enter the pulmonary vessels and obstruct the flow of blood through the lungs. Cyanosis, precordial pain, rapid pulse and respiration, hyperpyrexia and petechial haemorrhages typically seen in the conjunctivae, on the chest and axillae may develop in 8 to 20 hours. There may be fever, thrombocytopaenia, fat globules in the urine, sputum and retina and renal failure. Death may occur with severe dyspnoea about 30 hours after the trauma. In such cases, fat globules are probably transported to the lungs in moderate quantities at intermittent intervals until the circulation is obstructed. A piece of lung tissue in 20% caustic potash is compressed by a coverslip and examined under the microscope as a rapid method of identification. After a few minutes, red cells disappear and fat is seen lying as plugs in the pulmonary capillaries. They appear highly refractile and show a narrow dark edge. The diagnosis is made by microscopic examination of frozen section of the lungs, stained for fat with Sudan III (orange colour), Scharlach R or osmic acid (black). The pulmonary vessels, arterioles and capillaries are seen filled with globular fat emboli. The lungs show congestion, oedema and slight hypostatic pneumonia. The brain, heart and kidneys may show few scattered emboli. Bone marrow fat is scanty in children, due to which fat embolism is rare. 20 to 100 ml. of free fat causes embolic death.

Fatal pulmonary embolism can be diagnosed after injury to a limb, when an embolus is found in the pulmonary artery or one of its branches, and when a part of a thrombus or the site of thrombus can be demonstrated in one of the veins of the limbs. Pulmonary emboli are cylindrical in shape with parallel contours, often branched, frequently curled and show no relationship in shape to the vessel in which they are found. Death usually occurs about the tenth day, but may be delayed up to three weeks.

Cerebral fat embolism develops when the fat emboli are forced through the pulmonary capillaries into the systemic circulation in sufficient quantity to affect the brain. About 14 to 18 hours after injury, clusters of petechial haemorrhages are seen in the skin of the shoulders, neck, upper chest, and death occurs in one to two days. At autopsy, petechial haemorrhages are seen in the white matter of the brain, and appear as a ring of red blood cells around a necrotic zone of brain tissue in the centre of which lies a small arteriole plugged by a fat embolus. Death from fat embolism of the brain occurs when vital centres are affected.

Brain embolism: Trauma to the head accompanied by disruption of the brain can result in small amounts of brain entering the dural venous sinuses, if the patient survives long enough and cause embolism.

(10) Air Embolism : The causes of pulmonary air embolism are: (1) Incised wounds of the lower cervical or clavicular regions involving the jugular or subclavian veins. Penetration by bullet does not usually produce air embolism. (2) A wound of the sagittal sinus inside the skull. (3) Faulty technique in giving intravenous injection with the gravity or similar apparatus. (4) Crush injuries of chest. (5) Positive pressure ventilation in newborn infant. (6) Injection of air or fluid mixed with air or soap water into the pregnant uterus for procuring abortion. (7) Caesarean section, version or manual extraction of the placenta may introduce air into uterus. (8) Oral-genital sex in a pregnant woman. (9) Injection of air under pressure in Fallopian tube to test its patency. (10) Subclavian vein catheterisation. (11) Air encephalography. (12) Artificial pneumothorax and pneumoperitoneum. (13) Caisson's disease.

Air entering the venous system is carried to the heart and pulmonary arteries, which causes mechanical obstruction of the pulmonary arterial vasculature. This causes churning of the blood and air producing froth. The churning can result in the

development of complexes of air bubbles, fibrin, platelet aggregates, erythrocytes and fat globules thus further occluding the vasculature, which causes death. Large volume of air causes obstruction in the pulmonary vasculature and also in the right ventricle. Paradoxical air embolism, i.e. air crossing from the venous to the arterial circulation, usually occurs due to a septal defect or a probe patent foramen ovale. Two factors favour pulmonary air embolism. (1) Fixation of walls of veins, e.g., in the clavicular and pelvic regions, the upper dorsal spine and the dural membranes. When these veins are cut, they do not collapse and haemorrhage occurs, followed by negative pressure in the vessel. (2) The suction effect of the respiratory movements and heart's action on such veins as the jugular, subclavian and vertebral has a tendency to create a negative pressure in the vessels during the phase of inspiration. Pulmonary air embolism is rarely seen following injury to peripheral veins. Air injected into a limb artery is not harmful, as most of the gas is absorbed rapidly. About 100 ml. of air introduced under pressure is necessary to produce fatal pulmonary air embolism.

Diagnosis : X-ray examination of the whole body may detect large quantities of air. Air bubbles in the retinal arteries can be demonstrated with an ophthalmoscope. The gas is collected from the heart and brought into contact with an alkaline pyrogallol solution, which turns brown in the presence of free oxygen and indicates ante-mortem air embolism.

At autopsy, the sternoclavicular joint should not be opened in cases of air embolism, as pulling back the sternum may suck air into the veins. The lower part of the sternum is cut at the manubrium. Clamping of ascending aorta and vena cavae prevent the escape of air into these vessels. The chest cavity is filled with water. The coronary arteries are incised and massaged and the escape of gas bubbles is observed. The epicardial veins usually have a beaded appearance, with numerous air bubbles along the length. Air in the coronary arteries indicate systemic embolism. In air embolism, right ventricle is distended with air under pressure and bright-red frothy blood is found in the right side of the heart,vena cavae, pulmonary arteries and coronary veins. The blood is fluid throughout the body, the viscera are congested, and petechiae are present in the serous surfaces and in the white matter of the brain. Make a small incision on the pulmonary artery and each chamber of the heart and look for the air bubbles rising to the surface of the water. The pericardial sac should be filled with water. A 50 ml syringe is filled with water and a large-bore needle attached. The right ventricle should be punctured. If air is present it will be seen to bubble up through the water in the syringe. Aspirometer will measure the amount of air which can be analysed by gas chromatography. In embolised air CO_2 is less than 15%, nitrogen is higher than 70% and oxygen is reduced to 8 to 15%.

Oxygen in the heart indicates air embolism, because it is not present in appreciable quantity if the gases were those of decomposition. Frothy blood resembling air embolism can be found especially in the right ventricle (1) due to handling the heart before it is opened at autopsy, (2) putrefaction , (3) post-mortem event, and (4) artificial respiration in the dying or recently dead. The source from which air was derived should be established, e.g., if source is regarded as pregnant uterus, air bubbles should be found in the veins draining the large pelvic veins and the inferior vena cava.

For the diagnosis of systemic air embolism, the internal carotid and the basilar arteries are ligated before the brain is removed. The skull vault should be removed, without puncturing the meninges. Examine the meningeal vessels for visible air bubbles. In acute cases, gas bubbles will be visible within the cerebral arteries, but not in the cortical veins. The presence of bubbles in the pial veins is an artefact. Then submerge the brain in water, release the ligatures, cut the vessels and slightly compress them and watch for air bubbles. An airtight, water-filled glass syringe with a needle can be used to collect gas from blood vessels, heart or cavities. In artificial respiration in a dying person right ventricle may contain frothy blood.

Systemic air embolism occurs when air in sufficient quantity enters a vein of the pulmonary system and is carried through the left side of the heart to block arterioles and capillaries in different parts of the body, especially in the brain and the heart. Penetrating wounds of the chest are the common cause, e.g., puncture wound in a lung which does not collapse may penetrate a vein and a bronchiole and allow air to enter the vein. Crushing injuries of the chest and major or minor surgical procedures upon the thorax also produce air embolism. One to two ml. of air may be enough to produce death, because the air travels directly from lungs to the left side of the heart, from which it can be forced into the coronary or the cerebral arteries. Air bubbles in the cerebral vessels can be due to artefact produced by removing the skull.

Death from air embolism commonly occurs within a few minutes and usually not delayed beyond 45 minutes.

(11) ADULT RESPIRATORY DISTRESS SYNDROME (ARDS): It occurs due to heavy impacts on the thorax, blast injuries to the chest, pulmonary infections, toxins, systemic shock, irritant gases, aspiration of gastric contents, near drowning, etc, in which there may be "diffuse alveolar damage". The victim may suffer from severe dyspnoea and progressive respiratory failure due to poor exchange of gases. The lungs become stiff and oedematous, and retain their shape after removal, and may be double in weight. Histologically, intraalveolar exudates, hyaline membrane and patchy alveolar haemorrhages are seen in the lungs. Later, cellular proliferation is seen and in 50% of persons who survive, lungs become fibrotic.

RESPIRATORY LUNG: It is similar to ARDS. It is seen within a few days, but usually after many days or weeks in a patient who dies after being on mechanical ventilation. The lungs are stiff, rigid, heavy, but not obviously oedematous. Microscopically, proteinaceous fluid and a mixture of proliferative cells may be seen in the alveoli.

(12) DISSEMINATED INTRAVASCULAR COAGULATION (DIC): It occurs due to trauma, infection and other acute events. It is a consumption coagulopathy associated with blood clotting mechanism. There is an abnormal activation of the coagulation process within the blood vessels. Damaged tissue from trauma and burns can trigger thromboplastin-initiated coagulation; entry of tissue cell elements, especially from red cells, brain and placenta are particularly potent. Particulate matter, such as bacteria, micro-emboli of all types including fat and air emboli, can precipitate coagulation via factor XII. Vascular endothelial damage and stasis of blood flow have similar effect.

Fibrin is consumed and precipitated in vessels causing both vascular obstructive effects and a from red cells, brain and placenta are particularly potent. Particulate matter, such as bacteria, microemboli of all types including fat and air emboli, can precipitate coagulation via factor XII. Vascular endothelial damage and stasis of blood flow have similar effect.

Fibrin is consumed and precipitated in vessels causing both vascular obstructive effects and a haemorrhagic diathesis from depletion of the coagulative system. There can be postmortem fibrinolysis, and as such it may be difficult to demonstrate fibrin histologically. Lungs, liver, kidney and adrenals should be examined for firbin. The complications are microvascular obstruction leading to infarction and bleeding in skin and internal organs.

(13) SUPRARENAL HAEMORRHAGE: Haemorrhage into the adrenals, sometimes bilateral, tends to occur within three weeks not related to direct injury to the organ. The adrenals are swollen (3 to 5 cm) due to bleeding in the medulla, the cortex being tightly stretched in skin and internal organs.

MEDICO-LEGAL QUESTIONS ON TRAUMA

(1) Can post-mortem injuries be distinguished from ante-mortem injuries?

Severe bleeding from a wound indicates that it is antemortem. Injuries which usually result in active bleeding may produce little or no bleeding if they were sustained during severe shock resulting in hypotension and peripheral vascular collapse. Injuries caused soon after death which lacerate vessels distended with fluid blood can cause haemorrhagic infiltration of tissues. Injuries inflicted shortly before death cannot be distinguished from those inflicted shortly after death. Wounds produced shortly before death are not always associated with thrombosis or fibrin deposition. Absence of swelling does not exclude ante-mortem injury. White blood cells are capable of movement for few hours after cardiac arrest. This makes a "vital reaction" to injury of doubtful reliability. The physical components of vital reaction are: swelling, effusion of lymph, leucocytic infiltration, pus formation or evidence of repair.

HISTOPATHOLOGY: In injury after somatic death, margination and a limited emigration of leucocytes may occur. Marked cellular exudation and reactive changes in the tissue cells are seen in antemortem wounds only. However, the absence of tissue reaction does not necessarily indicate postmortem origin of wounds due to (1) if the victim dies soon after infliction of injury, (2) in small wounds the degree of cellular injury may not be sufficient to cause emigration of leucocytes, or there may be resolution of the reaction, (3) the circulatory failure occurring in severe injuries may interfere with normal reaction.

The intensity of the local reaction to an injury depends upon: (1) the severity of the injury, (2) the vascularity of the injured tissue, and (3) the presence or absence of infection or foreign bodies due to which the age of an antemortem wound cannot be determined within narrow limits.

HISTOCHEMICAL CHANGES IN INJURED TISSUES: In trauma to the living tissue, two zones are seen around the wound: (1) close to the edge of the wound, there is a zone 0.2 to 0.5 mm. wide, which

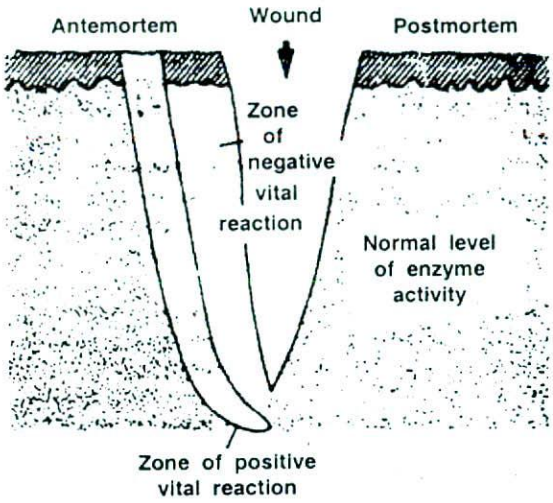


Fig. (10–3). Histochemical wound changes.

becomes necrotic with decreased enzyme activity (negative vital reaction). (2) Immediately beyond this layer, there is a 0.1 to 0.3 mm. zone of reaction, where a number of enzymes become increased in concentration during the reparative process (positive vital reaction), compared to the normal level in the areas outside the wound. In post-mortem wounds, positive vital reaction zone does not develop. In the positive zone esterases and adenosine triphosphatase increase within an hour after injury. At around two hours, aminopeptidase; at four hours, acid phosphatase, and at five hours, alkaline phosphatase activity increases. These changes can be demonstrated for a few days after death, if autolysis is prevented by refrigeration.

Tissue cathepsins increase almost immediately if the stroma is damaged, and can be demonstrated within 5 to 10 minutes. Serotonin appears in maximum concentration in about 10 minutes and histamine in 20 to 30 minutes after wounding. To establish the ante-mortem nature of the wound, the level of histamine should be at least 50% greater and that of serotonin, at least twice the concentration of the control skin. Post-mortem wounds do not show these reactions.

Enzyme reactions in a wound are specific indicators of its vital origin. They take place quicker than any other reliable change yet known. The reactions in wounds inflicted on different parts of the body are similar. The presence or absence of reactions is not affected by any period of delay between death and examination. The reactions are not affected by therapeutic measures, such as the application of antiseptic agents.

(2) Can a lethal degree of internal trauma be present without any external indication of trauma? Yes. A wedging type of blow to the lower chest and upper abdomen may tear the aorta, as in traffic accidents. The weight of the individual who applies his knee to the upper abdomen of another may cause a rupture of the liver and death. Manual strangulation and smothering may not leave external signs of trauma. Fractures of ribs, vertebrae or pelvis with accompanying lethal visceral injuries can occur without external indications of serious violence.

(3) Can resuscitative efforts produce injuries which cannot be differentiated from the injuries already present?

Table (10–2). Difference between ante-mortem and post-mortem wounds.

	Trait	Ante-mortem wounds	Post-mortem wounds
(1)	Edges:	The edges are swollen, everted, retracted, and wound gapes.	Edges do not gape, but are closely approximated.
(2)	Haemorrhage:	Abundant and usually arterial.	Slight or more and venous.
(3)	Spurting:	Signs of spurting of arterial blood on the body, clothing or in its vicinity present.	No spurting of blood.
(4)	Extravasation:	Staining of the edges of the wound and extravasation in neighbouring subcutaneous and interstitial tissues which cannot be removed by washing.	Edges and cellular tissues are not deeply stained. The stain can be removed by washing.
(5)	Coagulation:	Firmly coagulated blood in wounds and tissues present.	No clotting or soft clot.
(6)	Vital reaction:	Signs of vital reaction present, i.e., inflammation and repair.	No signs of vital reaction.
(7)	Enzyme histochemistry:	Increased activity of esterases, adenosine triphosphate, aminopeptidase, acid and alkaline phosphatase.	Diminished or no enzyme activity.

Yes. In peri-mortem injuries (that occur during the act of dying), haemorrhage may be seen involving the soft tissues. Contusion-abrasion of chest wall, fractures of ribs, fractures of the sternum, contusions of the heart, contusions and lacerations of the liver and spleen, rupture of the heart and duodenum usually occur during resuscitation.

(4) Can the time of infliction of the wound be determined?

Wound Healing : The changes vary according to the size of a wound, the type of wound, the tissue, and the age and health of the victim. Only an approximate time can be determined. Healing by first intention means union in a clean incised wound with good apposition of the edges. In healing by secondary intention granulation tissue is formed.

Histopathology: Vasoconstriction begins within few seconds. Dilatation of capillaries occurs within few minutes, accompanied by increased permeability to plasma protein. Fibrin appears in the wound within a few minutes, but can also appear in postmortem injuries. Migration of polymorphs may be seen in dilated small vessels in half to 4 hours but is not a reliable sign of antemortem injury. Basophilic mast cells lose their granules within 4 hours. In 4 to 12 hours, leucocytic infiltration is marked. At about 4 hours some extravascular emigration may begin. After 12 hours, a scanty mixture of lymphocytes and monocytes appear. Tissue oedema and swelling of vascular endothelium occurs. In 12 to 24 hours, leucocytes form a marginal palisade. Macrophages and mononuclear cells increase. Removal of necrosed tissue begins. In about 15 hours, mitoses are seen in fibroblasts. Epidermis begins to spread across and down the surface of the scab and down the sides of a cut into the wound. At about 48 hours, leucocytic infiltration is at its peak. Repair begins with many fibroblasts appearing, and elastic fibres are scarce. In about 72 hours, new capillaries begin to bud from vessels, and granulation tissue forms. In 3 to 6 days, collagen begins to form, and giant cells appear around necrotic debris. Haemosiderin appears from the third day. In 3 to 6 days, epithelium grows actively on the surface. In 10 to 14 days, vascularity decreases, and cellular reaction subsides. Fibroblasts are very active, and collagen is laid. Epidermis becomes thin and flat. Elastic fibres are less compared to adjacent undamaged tissue. Regression of cellular activity occurs by 12 days in both epidermal and dermal tissue. After two weeks, collagen and elastin increase, and vascular scar is formed, which gradually becomes more dense and avascular. Contraction starts about 5 to 7 days after wounding and stops at 6 weeks.

(5) Can there be much delay in death following the infliction of trauma?

Yes. The victim may die after a varying period from remote causes. The rupture of liver, spleen, aorta and other organs may be delayed by hours, days and sometimes weeks after the organ was weakened by the traumatic lesion.

(6) Which of the several injuries caused death?

The actual cause of death must be found out, and when there is more than one wound, it may be necessary to determine which one of them caused death, since the wounds may not have been made by the same assailant, nor at the same time. This may be done by examining the wounds individually and noting which of them involved injury to some vital organ or large blood vessel, or led to secondary results causing death.

(7) How long did the victim live, and could he have carried out voluntary acts after receiving a fatal injury?

In cases where the injuries are presumed to have caused rapid death, a medical witness may be asked to state whether a person is capable of speaking, walking or performing any other voluntary act, which would involve bodily and mental power for some time after receiving the injury. The length of time the deceased could have lived after infliction of injury will depend upon the nature of the injury, the degree of associated shock and the rate of bleeding. A very guarded reply should be given, because individuals vary in their reaction to injuries. Frontal lobe damage does not incapacitate immediately. If the brain stem is destroyed by a penetrating injury or aortic arch is completely transected, immediate incapacity occurs. Most other injuries do not cause sudden death or rapid loss of function. In severe injuries of the brain, sometimes a person survives, although the damage done might have been expected to have caused instant death. A person may remain conscious for several minutes before dying from a severe intracranial injury. Sometimes, a person may die from a blow which produces no obvious naked-eye changes in the brain. A single stab wound of the skull and brain are not immediately fatal, and the victim may walk or run. Stab wounds of the heart are dangerous, but often rapidly disabling or fatal. Immediate death is likely when the auricles are extensively torn or punctured,

but in less severe wounds, powers of voluntary acts and walking are retained for some time. In slanting ventricular perforation, at first there may not be bleeding but later there may be mild oozing which may cause death. Perforations of the right ventricle bleed more rapidly than those of the left, as it is relatively thin-walled. The victim may be able to run 100 to 200 metres before collapsing. Small perforations of the left ventricle may seal themselves with minimal blood loss. In fatal electrocution in which the heart stops functioning, the person can walk and talk for 20 to 30 seconds before dropping dead. Muscular powers are retained in ruptures of liver, spleen or kidneys, unless there is marked immediate blood loss. When the windpipe is divided, the voice is usually lost. It is not possible to say from an examination of wounds in a dead body, as to how long the person might have lived, or how much voluntary activity he might have performed before death, after infliction of the injury. It is safer to say what is ordinarily expected.

CASE: (1) Glaister reported a case in which the man shot himself with a 0.45 caliber pistol beneath the chin. The bullet entered the cranium and pulped the frontal and temporal lobes of the brain and exited through a 4 cm. defect in the skull. Following this injury the man walked from a garden shelter (where the shooting had occurred and where later pieces of skull and brain were found on the ceiling), walked around in the garden for at least 165 metres, returned to the shelter for some time, then walked home to his house, where he arrived at least one hour after the shooting. He spoke in normal fashion to the maid who admitted him, hung up his overcoat and umbrella, and walked upstairs to his room. Death did not take place until at least 4 hours after the shooting.

(2) A 28 year old man was shot at from a range of 3 metres in the anterior chest with a 12 bore sawed-off shotgun. He ran about 16 metres before he collapsed and died. At autopsy anterior wall of right ventricle showed a ragged 3 cm. opening. Multiple pellet perforations were present in the right lung. The right pleural cavity contained 2500 ml of blood and pericardial sac 400 ml of blood. Wad was found in both main pulmonary arteries at the lung hila, carried to these sites as emboli from the right ventricle.

(3) A 37 year old man was stabbed in the chest with a butcher's knife. Following the injury, he got into his car and drove four city blocks; lost control of the vehicle and crashed into a parked car. He proceeded to the hospital and died in the emergency room about 20 minutes after the injury. At autopsy there was a 6 mm slit-like cut in the right ventricle of the heart, and 1400 ml. of blood in the left pleural cavity and in the pericardial sac.

(4) A 23 year old man who was stabbed with a kitchen knife, walked and staggered a distance of 110 metres and died at the hospital a few minutes after arrival there. At autopsy, there was a 6 mm. cut in the left ventricle of the heart and the aorta and 1900 ml. internal bleeding.

(8) Can the degree of trauma be quantitated?

There is much individual variation in the reaction to trauma. At best the amount of degree of injury can only be roughly estimated as mild, moderate, severe, or extreme.

(9) What is the relationship of trauma and natural disease?

Trauma is an injury or wound to a living body caused by the application of external force or violence. There is strong evidence that trauma does not induce neoplasia, and that the co-existence of the two processes is purely co-incidental. Often, trauma merely calls attention to a pre-existing tumour that had not previously been noted. Since trauma disrupts tissue, it might activate a pre-existing tumour to grow and spread more rapidly. In rare cases there is apparent relationship, between tumour and some preceding trauma to the part. Often, trauma merely calls attention to a pre-existing tumour that had not previously been noted. Repeated minor trauma is called chronic irritation. With repeated minor trauma and disruption of tissue, the cellular proliferations of regeneration and repair become continual. In few cases, cancer develops after many years, e.g., cancer of the skin adjacent to a chronic osteomyelitic sinus that has drained for a number of years. Skin cancers developing in burn scars have been well recognised. Since trauma disrupts tissue, it might activate a pre-existing tumour to grow and spread more rapidly. Ewing's postulates should be satisfied before accepting a relationship between trauma and new growth. They are: (1) There must be evidence of previous integrity of the injured part. (2) Undeniable and adequate trauma must be proved. (3) There must be proof of reasonable time interval between injury and appearance of tumour. (4) The disease must develop at exact site of the injury. (5) The nature of the tumour must be proved.

The role of direct trauma in rupturing an aneurysm is not certain. A large, fragile aneurysm on the circle of Willis might be damaged by a substantial head injury.

TRAUMA AND MYOCARDIAL INFARCTION

A blow or some physical trauma may precipitate myocardial infarct or arrhythmia. The emotional upset that accompanies injury, or even the threat or fear of an injury, can cause death due to transient

hypertension or tachycardia that may precipitate a subintimal haemorrhage, arrhythmias, or cerebral or subarachnoid haemorrhage. Physical effort which can damage a diseased heart in some cases can be traced to unusual job or to the performance of unfamiliar or unaccustomed work, to accidents or other trauma, and to the extra physical demands while working with a defective equipment.

A heart attack my occcur while at work, either incidentally by normal progression of a chronic disease process or due to unusual physical or mental strain. The risk is greater in those who were otherwise sedentary and that long-term moderate exerscise is associated with a reduced risk. Some cardiologists feel that a heart attack never occurs after physical effort, while others believe it can occur. If the attack occurs within seconds or minutes after unusual effort, the causal connection can be established with certainty only in direct trauma to the heart occurring during work. Attacks occurring few days later may be due to haemorrhage in an atherosclerotic plaque in the coronary artery which initially narrows the lumen, but later causes occlusion.

Mechanical forces responsible for heart injury are: (1) Penetrating foreign objects, e.g., missiles and stab wounds. (2) Migrating foreign objects, e.g., needles, bullets, metal fragments, etc. (3) Non-penetrating mechanical forces transmitted to the heart, e.g., (a) Compression forces from direct blows to chest, from crushing chest injuries or transmitted from injury to the abdomen or legs. (b) Contusive forces and jarring forces from direct blow to the chest or transmitted from decelerative accidents. (c) Bursting forces from increase of intracardiac blood pressure due to direct compression of heart from chest injuries or transmitted from compression of the abdomen, and (d) Tearing, disruptive forces transmitted from decelerative accidents. (4) Intracardiac-introduced instruments, e.g., cardiac catheterisation and implantation of pace-makers.

SUBARACHNOID HAEMORRHAGE : An assault is rapidly followed by the signs of subarachnoid bleeding and subsequent death. A heavy blow to the head, jaw or neck can rupture, split or weaken the fragile wall of a large, thin-walled aneurysm. In fights, the victim is physically and emotionally active, so that adrenal response is likely to be present. Muscle tone, heart rate and blood pressure are increased by catecholamines, and it is likely thar raised internal blood pressure in a weak aneurysm is a far more potent reason for rupture than a blow on the head. Another possibility is that a person with an already spontaneously leaking aneurysm may have a rapidly developing neurological or even behavioural abnormality that causes a fight, or into a dangerous physical position, such as a fall or a traffic accident. Rupture can occur in young to middle aged persons during innocent activities, such as jogging, intercourse, and sporting exertion, probably because of a transient rise is blood pressure and pulse rate.

(10) Would the victim have survived had he been given more prompt medical assistance?

A very guarded reply should be given, depending on the nature and extent of injuries, because there is much individual variation.

(11) Can the wounds be altered from their original appearance?

The wounds may be altered in many ways. In the living, the wound may be altered by healing and treatment. In the dead person, the wound might have been altered by resuscitative measures applied and by insects, animals and decomposition.

(12) Are there situations where more than one type of weapon was employed with any frequency?

Such situations are not rare. In gang feuds, the victim may be suddenly attacked by a number of persons with different types of weapons. The victim may be waylaid and a number of different types of injuries inflicted. Sometimes, a suicide uses different types of weapons, as some methods fail to produce death.

Death need not occur very soon after the assault, if it can be established that assault could have precipitated an infarct in a myocardim already compromised by coronary stenosis. The blow may never actually be struck or it may fail to hit the body, yet the threatened person may suffer a transient hypertension or tachycardia that may precipitate subintimal haemorrhage that leads to death.