Thanatology:
Deals with study of death in all its aspects.

Death Two Types

Somatic Death/Clinical/Systemic Death.

(few minutes after death)

It is the complete and irreversible stoppage of the circulation,respiration and Brain Function

Molecular Death

(Few minute to hours)

It is death of tissues and cells individually which takes place usually in 1-2hours after stoppage of vital functions.

Brain death

Cortical Death

Brainstem Death

Combination of a & b.

Medicolegal aspects of Brain Death:

It is the confirmatory sign of Death which is very vital for-

- Organ transplantation
- Criminal responsibility

Vegetative state:

In this the patient breathes spontaneously, has a stable circulation and shows cycle of eye opening and closing which may simulate sleep and waking, but is unaware of the self and the environment.

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

Beating Heart Donor:

After Brain stem death has been established the retention of the patient on the ventilator facilitates a fully oxygenated cadaver transplant.

Suspended Animation

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.

Agonal Period.

It is the time between lethal occurrence and death. This period is variable and Various activity have been reportedly performed by the victim.

Cause of Death.

It is the disease or the injury responsible for starting the sequence of events, which are brief or prolonged and which produced death. It consists of Two Parts First parts includes framed in the following order i.e. a).Immediate Cause, b) Antecedent Cause., c) Underlying Cause. Second Part includes other Contributing Causes directly or indirectly Leading to Death besides the Interval of onset of the Terminal symptoms and the Death.

Manner of Death.:

Is the way in which the cause of death was produced-Natural and Unnatural i.e.Accident,Suicide and Homicide.

Mechanism of death:

It is the physiological or biochemical disturbances, produced by the cause of death which is incompatible with life eg. Shock, sepsis, Metabolic acidosis and alkalosis,Ventricullar fibrillation,etc.

Modes of Death:

Asphyxia, Coma and Syncope

Changes in the Body after Death

A knowledge of the signs of death help to differentiate death from suspended animation. The changes which taken place may be helpful in estimation of the approximate time of death. the signs of death appear in the following order.

1. Immediate (somatic death)

Insensibility and loss of voluntary power

Cessation of respiration

Cessation of circulation

2. Early (cellular death)

Pallor and loss of elasticity of skin

Changes in the eye

Primary flaccidity of muscles.

Cooling of the body

Post-mortem lividity

Rigor mortis

3. Late (decomposition and decay)

Putrefaction

Adipocere formation

Mummification

SUSPENED 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, cholera, after anesthesia, shock, sunstroke, cerebral concussion, insanity, etc. The patient can be resuscitated by cardiac massage or electric stimulator and artificial respiration.

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:

Loss of Corneal Reflex:

Opacity of the cornea:

Flaccidity f the eyeball:

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.

Retinal vessels: Fragmentation or segmentation [trucking] of the blood columns 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 ophthalmascope. 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.

Chemical changes: A steady rise in the potassium values occur in the vitreous humour after death.

The cooling of the body Algor mortis; ‘chill death’ after death is a complex process, which does

not occur at the same rate throughout the body. The body cools more rapidly on the surface and more slowly in the interior. 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 as the temperature of the air is approached. The body heat is lost by conduction, convection and radiation. Only a small fraction of heat is lost by evaporation of fluid from the skin. In serious illness, circulation beings to fail before death, and hands and feet become cooler than the rest of the body; this coolness gradually extends towards the trunk.

Normal body temperature – rectal temperature/Rate of temperature fall per hour

It cannot be assumed that the body temperature is normal at death. in cases of fat or air embolism, certain infections, heatstroke and in pontine haemorrhage, drug reactions, etc. a sharp rise in temperature occurs. Exercise or struggle prior to death may raise the rectal temperature up to 1.5^{o} to 2^{o} C. Low temperature occurs in cases of collapse, congestive cardiac failure, etc. During sleep the rectal temperature is 0.5^{o} to 1^{o}C. lower.

Factors Affecting Rate of Cooling:

The difference in temperature between the body and the medium.

The build of the cadaver.

The physique of the cadaver.

The environment of the body.

Covering on or around the body

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

Postmortem caloricity: In this condition, the temperature of the body remains raised for the first two hours or so after death.

This occurs:

when the regulation of heat production has been severely disturbed before death, as in sunstroke and in some nervous disorders,

when there has been a great increase in heat production in the muscles due to convulsions, as in tetanus and strychnine poisoning, etc., and when there has been excessive bacterial activity, as in septicaemic condition cholera and other fevers.

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 post-mortem staining, subcutaneous hypostasis, livor mortis, cadaveric lividity, suggillations, vibices and darkening of death. 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. It is caused by the stoppage of circulation, the stagnation of blood in blood vessels, and its tendency to sink by force of gravity. The blood tends to accumulate in the small vessels of the dependent parts of the body. Filling of these vessels produces a bluish-purple colour to the adjacent skin. The upper portions of the body drained of blood are pale. The colour of the hypostasis may vary from area to area in the same body.

It beings as patchy mottling of the skin. 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. It is usually well-developed within four hours and reaches a maximum between 6 and 12 hours. It is present in all bodies, but is more clearly seen in bodies of fair people than in those of dark.

Hypostatic congestion resembling post-mortem hypostasis may be seen a few hours before death in case of a person dying slowly with circulatory failure, e.g. cholera, typhus, tuberculosis, uramia, morphine poisoning, congestive cardiac failure, and asphyxia. In such cases, hypostasis will be marked shortly after death. 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 the stain depends on the position of the body. In a body lying on its back, it first appears in the neck, and then spreads over the entire back with the exception of the parts directly pressed on, i.e. occipital scalp, calves and heels. Any pressure prevents the capillaries from filling, such as the collar band, waist band, belts, wrinkles in the clothes, etc. and such areas remain free from colour and are seen as strips or bands called vibices. Such pale areas should not be mistaken for marks due to beating, or when they are present on the neck, due to strangling. Hypostasis is usually well-marked in the lobes of the ears and in the tissues under nails of the fingers. As the vessel walls become permeable due to decomposition, blood leaks through them and stains the tissues. At this stage, hypostasis does not disappear, if finger is firmly pressed against the skin. 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. 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. 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. 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. 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. in drowning, post-mortem 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 before the blood coagulates, these patches will disappear and new ones will form on dependent parts, but lividity to a lighter degree remains in the original area, due to staining of the tissues by haemolysis. When coagulation in capillaries takes place, the stains become permanent and this is known as fixation of post-mortem staining. This usually occurs in about six hours, but the condition of blood at the time of death exerts a considerable influence. Persistent fluidity of the blood appears to be due to presence of fibrinolysins. Hypostases may resemble bruises. In doubtful cases, a portion should be removed for microscopic examination.

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

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

The hypostatic areas have distinct colour in certain cases of poisoning, e.g.

In carbon monoxide poisoning, the colour is cherry-red.

In hydrocyanic acid poisoning and sometimes in burns the colour is bright red.

In poisoning by nitrites, potassium chlorate, potassium bicarbonate, nitrobenzene and aniline [causing methaemoglobinaemia] the colour is red-brown, or brown.

In poisoning by phosphorus, the colour is dark-brown.

In asphyxia, the colour of the stains is deeply bluish-violet or purple. In exposure to cold and refrigerated bodies the colour is pink, as the wet skin allows atmospheric oxygen to pass through, and also at low temperature haemoglobin has a greater affinity for oxygen. In septic abortion caused by Cl. Welchii the colour is often grayish-brown.

Internal Hypostasis:

Hypostasis also occurs in the internal organs and the dependent parts of the liver, spleen, kidneys, larynx, lungs, heart, intestines and brain show colour changes similar to that in the skin. Hypostasis in the heart can simulate myocardial infarction, and in the lungs it may suggest pneumonia; dependent coils of intestine appear strangulated.

Changes in postmortem:

Lividity occur when putrefaction sets in. in early stages, there is haemolysis of blood and diffusion of blood pigment into the surrounding tissues, where it may undergo secondary changes, e.g., sulphhaemoglobin formation. The capillary endothelium and the surrounding cells show lytic changes. Microscopically, the cellular outlines are obscured and the capillaries are not factive changes and it becomes impossible to determine whether the pigment in a stained putrefied area originated from an intravascular [hypostasis] or/and intravascular localized 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. In mummification, lividity may turn from brown to black with drying of the body.

Medico-legal Importance:

It is a sign of death.

Its extent helps in estimating the time of death

It indicates the posture of the body at the time of death

It may indicate the moving of the body to another position sometime after death.

Sometimes, the colour may indicate the cause of death.

MUSCULAR CHANGES

After death, the muscles of the body pass through three stages-

Primary relaxation or flaccidity.

Rigor mortis or cadaveric rigidity.

Secondary relaxations(Flacidity).

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. Muscular irritability and response to mechanical or electrical stimuli persists. Peristalsis may occur in the bowel, and ciliary movement and movement of white cells may continue. 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

This 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 are the contractile elements, and are made up of protein filaments of two types, actin filaments and myosin filaments which form a loose physico-chemical combination called actomyosin, which is physically shorter than the two substances uncombined. 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.They are energy dependent(ATP) This causes the muscle to contract.Rigor persists until decomposition(Absence of energy production ATP) 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 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. 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 physico-chemical 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.

When rigor is fully developed, the entire body is stiff, the muscles shortened, hard and opaque; knee, 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 anserine or goose skin. the testes may be drown 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 te 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 out symmetrical groups, which have not been disturbed. 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. 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. 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.

Time of onset:

In Jamaica, it begins one to 2 hours after death and takes further one to 2 hours to develop. In temperate countries, it begins in 3 to 6 hours and takes further 2 to 3 hours to develop.

Duration of rigor mortis:

In Jamaica, usually it lasts 18 to 24 hours in winter and 12 to 18 hours in summer. It lasts for 2 to 3 days in temperate regions. When rigor sets in early, it passes off quickly and vice versa.

Conditions altering the onset and duration:

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, while in children and old people it is feeble and rapid.

Nature of Death: In deaths from diseases causing great exhaustion and wasting, e.g., cholera, typhoid, tuberculosis, cancer, etc. and in violent death as by cut-throat, firearms or by electrocution, the onset of rigor is early and duration is short. In strychnine and other spinal posions, the onset is rapid and the duration longer. In deaths from asphyxia, severe haemorrhage, apoplexy, pneumonia, nervous disease causing paralysis of muscle, and perfusion with normal

saline, the onset is delayed. It may disappear very rapidly in case, of widespread bacterial infection, especially in gas gangrene, where putrefaction begins early.

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, if there is fatigue or exhaustion before death. In persons who run prior to death, rigor may develop rapidly in their legs, compared to other parts. After insulin injection it develops quickly, as the muscle glycogen is reduced.

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

Conditions simulating rigor mortis:

Heat stiffening: When a body is exposed to temperatures above 65ºC, a rigidity is produced, which is much more marked than that found in rigor mortis.The stiffening remains until the muscles and ligaments soften from decomposition and the normal rigor mortis does not occur.

Cold stiffening: When a body is exposed to freezing temperatures, the tissues becomes frozen and stiff, due to freezing of the body fluids and solidification of subcutaneous fat simulating rigor. It the body is placed in warm atmosphere, the stiffness disappears and after a time, the normal rigor mortis occurs.

Cadaveric spasm or instantaneous rigor: Cadaveric spasm [cataleptic rigidity] is a rare condition. In this, the muscles that were contracted during life bcome 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 of death for several hours afterwards. It occurs especially in case of sudden death, excitement, fear, sever pain, exhaustion, cerebral heaemorrhage, injury to the nervous system, firearm wound of the head, 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 muscles and frequently involves the hands. 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.

The nature of cadaveric spasm is obscure, but like rigor mortis, it may be explained on the basis of diminished or exhausted ATP in the affected muscles. 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.

This condition is of great **medico-legal importance.** 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 murde3r. but, ordinary rigor does not produce the same firm grip of a weapon, and the weapon may be placed in the had in a way which could not have been used by a suicide. 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. 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 the entering the water.

Secondary relaxation: **Muscles become soft and flaccid due to breaking down of actomyosin due to putrefaction. The reaction of muscle juice again becomes alkaline.**

PUTREFACTION

Putrefaction or decomposition is the final stage following death, produced mainly by the action of bacterial enzymes, mostly anaerobic organisms derived from the bowel. Other enzymes are derived from fungi, such as penicillium and Aspergillus and sometimes from insects, which may be mature or in larval stage. The chief destructive bacterial agent is Cl. welchii, which causes marked haemolysis, liquefaction of post-mortem clots and of fresh thrombi and emboli, disintegration of tissue and gas formation in blood vessels and tissue spaces. The other organisms include Streptococci, Staphylococci, B. Proteus, B. Coli., B. aerogenes capsulatus, etc. Bacteria produce a large variety of enzymes and these breakdown the various tissues of the body. 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. 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.

Autolysis:
Soon after death, cell membranes become permeable and breakdown, with release of cytoplasm containing enzymes. The proteolytic, glycolytic and lipolytic action of ferments leads to autodigestion and disintegratio of organs and occurs without bacterial influence.

The characteristic(3D) features of putrefaction are:

Discoloration-changes in the colour of the tissues,

Disfiguration-the evolution of gases in the tissues,

Dissolution-the liquefaction of tissues

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 he 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 body, the lower temperature, the exclusion f air, absence of animal life, etc., markedly delay decomposition. The important factors are seasonal, climatic variation, the amount of soil water, the access of air, and the acidity or otherwise of the soil water. In 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 centuries. 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:

Internally, decomposition advances at the same rate as seen externally. As the blood decomposes, its colouring matter transudes into the tissues, which become uniformly red, the colour becomes darker and finally black. 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 organs composed of muscular tissue and those containing large amount of fibrous tissue resist putrefaction longer than the parenchymatous organs, which because of the contents at the time of death, decompose rapidly.

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

Prostate: It resist 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:

Temperature: Putrefaction begins above 10ºC and is optimum between 21ºC and 38ºC. A temperature increase of 10ºC usually doubles the rate of most chemical processes and reactions. It is arrested below 0ºC, and above 48ºC.

Moisture: For putrefaction moisture is necessary, and rapid drying of the body practically inhibits it.

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

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 internal organisms. Clothes prevent the access of airborne organisms, flies, insects, etc., which destroy the tissues.

Manner of burial: If the body is buried soon after death, putrefaction is less. 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 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 more rapid if changes of decomposition are already present at the time of burial.

B] Internal:

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.

Sex: Sex has no effect.

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.

Cause of death: Bodies of persons dying from septicaemia, peritonitis, inflammatory and septic conditions, general anasarca, asphyxia, etc., decompose rapidly. Putrefaction is delayed after death due to wasting disease, anaemia, debility, poisoning by carbolic acid, zinc chloride, strychnine and chronic heavy metal poisoning, due to the preservative action of such substances on the tissues or their destructive or inhibitive action on organisms, which influence decomposition.

Mutilation: Bodies in which there are wounds, or which have suffered from other forms of violence before death, putrefy rapidly owing to the ease with which organisms gain access to the damaged tissues.

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

Putrefaction in water:

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 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.

INJURIES

A wound or injury is defined as disolution of the natural continuity of any of the tissues of the living body. Injury,
Legally, denotes any harm whatever illegally caused to any person in body mind reputation or property.
The injury may not be visible externally as fatal internal injuries may be inflicted internally in the

absence of external injuries. A Wound is a solution of the natural continuity of any of the tissues of the Living body. The word Trauma referes to an insult to the Living tissues.

General Principles:

a. **The Mechanical force which causes the change in the state of rest or motion of the tissues is due to the combination of the forward moving force and the counterforce causing transfer of energy to the Tissues of the body. This sudden change in the stae of rest or motion leads to damage of tissues.**
b. **Due to the complex nature of tissues in the Body which vary in their physical properties the force of impact does not affect the tissues uniformly**
c. 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(Shear force) 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.

General Characters of an Injury are dependent upon

a.The Nature of Object or Instrument causing the wound.
b.The condition under which the energy is discharged..
c. The mount of energy discharged during the impact.
d. The nature of the affected Tissues.

Mechanism of Wounding:

The body absorbs the natural forces, like gravity ,movement, routine movements like sitting and walking by the resilience and elasticity of its soft tissues and rigid skeletal framework. wound is due to the result forces which cross the limits of elasticity or resistance. The following are the factors which influence the wounding mechanism:

The amount of force applied which depends on Mass of the Object and Square root of the velocity with which it is applied i.e. Force=1/2 $M.V^{.2}$.

Eg. When a cricket ball(200gm) is pressed on the head it causes no damage however if the impact is at minimum velocity of 10m/sec it may end up in fracture. Hence apart from the mass the Velocity is the factor which plays the major role.

The transfer of the force through the body again a factor which contributes to the wounding power. If the wounding object pass through and through(eg. Perforating Bullet) the amount of damage is

less as compared to a wounding object lodging inside the body(Explosive bullet).

The distribution of Force over the Surface of the body is another factor, larger the area of distribution lesser the damage smaller the area of distribution more the damage(eg.pointed knife cause more damage than blunt weapon with equal amount of force).

The force acting on the surface of the body subjects the tissue to traction, shear and compression, hence the resultant damage depends not only on the type of the mechanical insult but also on the nature of the target tissue i.e Muscle,bone,hallow organ like intestine .etc.

Another very important factor is the movement of the body in the direction of the Force which adds up to the wounding. If the movement is towards the direction of the force the damage is least if against the direction of the force the damge is extensive.eg. Catching a cricket ball and moving the hands in the direction of the force will cause least damage than the contrary movment of the hand.

Classification:

Mechanical Injuries:

Abrasions

Contusions

Lacerations

Incised wounds

Stab wounds

Firearm wounds.

Fractures and dislocation.

(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, (2) Corrosive alkalis. (IV) **Injuries due to electricity, lightning, X-rays, radioactive substances, etc**.

Legally, injuries are classified into; (1) **Simple**, and (2) **Grievous**.

Others:

- **Self inflicted Injuries.**
- **Defense Injuries.**
- **Offensive Injuries.**

- **Unintentional Injuries.**
- **Fatal and NonFatal Injuries.**

Mechanical Injuries

Mechanical Injuries are Caused due to application of Mechanical force on any part of the body, like Blunt Force,Sharp Force and Firearms.

ABRASIONS

An abrasions is a destruction of the skin, which usually involves the superficial layers of the epidermis only. They are simple injuries, bleed slightly, heal rapidly and scar is not formed. Large abrasions can cause severe pain and bleeding. Abrasion is caused by friction of the skin against some rough or sharp surface resulting in the scraping away of superficial portions of the epidermis. Abrasion often take the form of parallel furrows in the skin surface.They are also caused by a lateral rubbing action by a blow, a fall on a rough surface, by being dragged in a vehicular accident, fingernails, thorns or teeth bite. This furrows may be broad at one end and tail away in the opposite direction.For an Abrasion to occur Some pressure and movement by agent on the surface of the skin is essential. If sufficient friction is applied, partial or complete removal of the epithelium any occur and the superficial layer of dermis is damaged. 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.
Microscopically the epidermal cells are flattened and their nuclei are elongated. Abrasions vary in size, depending on the extent of the body surface exposed to the abrading force. The size, situation, pattern and number of abrasions should be noted.

Types: Abrasions are of four types:

(1) Scratches: A Scratch or Linear abrasion is caused by horizontal or tangential movement of a pointed end of an object,it is wider at the starting point than the end ppoint.usually caused by a sharp or pointed object 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.

(2) Grazes : (sliding, scraping or grinding abrasion): These occur due to friction between the epidermis and wider part of the object causing it either due to horizontal or tangential friction.. They show uneven longitudinal parallel furrows with the epithelium heaped up at the ends of these lines, which indicate the direction in which the force was applied. The furrow may be broad at one end, and narrow in the opposite direction. Grazes are commonly found in Foad Traffic Accidents,Dragging,kicking etc.

(3) Pressure Abrasions: (crushing abrasions): This is a result of perpendicular application of force on the body surface,with minimal friction and is directed towards the depth of the body. The size and shape of the abrasion depends on the part of the weapon or the object coming in contact with the body.The pressure on the skin cause crushing of the superficial layers of the epidermis and are associated with a bruise of the surrounding area.They are

commonly seen in cases of hanging and strangulation and the teeth bite marks .

(4) Imprint Abrasions : (impact 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 follows the ridges of the object. The abrasion is slightly depressed below the surface, unless there is bulging due to underlying contusion or local edema. If the impact is forcible, the dermis is damaged with an underlying bruise.This ar commonly seen in , the pattern of the radiator grille, a headlamp rim or the tread of the tyre may be seen on the skin.

The pattern of the object causing it can be ascertained by the imprint and pressure abrasion caused by it on the skin surface and are called **patterned abrasions.** examples of patterned abrasion are: imprint of bicycle chain, weave of coarse fabrics, the spiral of electric wires, ropes, serrated knife, etc.

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

Fresh: Red in colour devoid of scab.

12 to 24 hours: Dark red scab due to drying of Lymph and blood.

1-2 days: Brownish red scab(serum or blood coagulation).

3-5 days: Reddish-brown or dark brown scab.

6 to 7 days: skrinkage of the scab. Epithelium grows and covers defect under the scab.

After 7 days: Complete healing with the Scab shrinking,drying and falls off.

Histology:In the first stage perivascular cellular infiltration is seen at 4 to 6 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, sub epithelial formation of granulation tissue is prominent. Reticulum fibers are seen at 8 days, and collagen fibres at 9 to 12 days.

Medico-legal Importance:

(1) impact and direction of the force can be determined. (2) Abrasions may indicated the possibility of serious internal injury. (3) Patterned abrasions help in determining the type of object of causation. (4) Time of Assault can be calculated based on state of Healing of abrasion. (5) Scene of Crime can be determined by the examination of Foreign particles from the wound and compared with the particles from the suspected scene. (6) Abrasion indicates the Manner of and circumstances surrounding the abrasion example, In throttling, crescentic abrasions due to fingernails are found on the neck.In smothering, abrasions may be seen around the mouth and nose. In sexual assaults, abrasions may be found on the breasts, genitals, inside of the thighs and around the anus.In struggle Abrasions are present over the face of the assailant

Conditions mimicking Abrasion:

1. **Excoriations of the Skin by Excreta, usually seen in Napkin area.**
2. **Ant Bites: usually found at mucocutaneous junction, nostril, mouth, eye lids, axilla, groin and genitalia.**
3. **Bed sores.**

CONTUSIONS (BRUISES)

A Bruise is an effusion of blood into the tissues, due to the rupture of blood vessels, caused by blunt trauma without disruption of the continuity of the skin. In contusion, there is a painful swelling,which may be present even in deep bruise.. Contusions may be present not only in skin, but also in internal organs, such as the lung, heart, brain and muscles. Surface contusions are due to extravasation of blood usually situated in the corium and subcutaneous tissues, often in the fat layer., The extravasated blood is diffusely distributed through the tissue spaces, and the margins are blurred. When a large blood vessel is injured, a tumour-like mass called haematoma is formed..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. **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 centers, 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

Due to variability in the Tissue density in the body the Bruise caused by similar force applied on different parts of the body may not be uniform in its formation and appearance. Hence the following factors influence the appearance and formation of a Bruise:

1. **Amount of Force:A greater force is more likely to cause a Bruise.With a very greater force a Haematoma may be produced with accumulation of large amount of clotted blood in the tissue of the affected part.**
2. **Type of Force: A soft blunt force may not cause a Bruise whearas a Hard Blunt Force will cause a Bruise, a Harder and Heavy Blunt force may likely cause Haematoma.Hence striking with a Stick,Iron Rod,Shoe,Stone,Whearas striking with a Sand bag may not.Minimal force may cause Bruise subject to hard resistant surface below the tissue like Bone.**
3. **Type of Tissue:** . 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 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. Resilient areas, such as the abdominal wall and buttock bruise less.Bruising of the scalp is better felt than seen. Bruising of the scalp with fluctuant centers can simulate depressed fracture. 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. In boxers and athletes, bruising is much less because of good muscle tone.

4. **Disease Condition:** Bruising is relatively more marked on tissues overlying bone. Chronic alcoholics,Senile individuals,Arteriosclerosis conditions, Individuals with purpura haemorrhagica, leukaemia, haemophilia, scurvy, vitamin K and prothrombin deficiency bruise easily.

5. **Age:** children bruise more easily because of softer tissues and delicate skin, and old person s bruise easily because of loss of flesh and cardiovascular changes.
6. **Sex:** Women due to their delicate skin and Increased Subcutaneous Fat bruise more easily than men. 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.
7. **Complexion of Skin:** In fair and light Brown skinned Bruising is more clearly seen than in those with dark or Brown skin Individuals. 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.

8 **Shifting of Bruise away from the site of Impact(Migrant or Ectopic Bruise)::** In Certain sites of the body the Bruises may not always appear at the site of impact. The bruise may appear at a relatively distant place, where the facial pattern permits it to reach the surface,Thei is due to the Blood escaping from deep torn vessels being prevented because of facial plane arrangements at the injured area.examples are Hemorrhages in the soft tissues around the eyes and in the eyelids due to impact to forehead ,anterior fossa of the skull or Direct Blunt trauma.. 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 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: The design on the surface of the weapon may be imprinted on the Bruise,indicating the pattern of the object causing the Bruise.Bruised made by the end of a thick stick may be round, but if any length of the stick hits the body, they are elongated and irregular. A blow with a rod, a stick or a whip produces two parallel, linear haremorrhages (railway line or tram line type). The intervening skin appears unchanged, because the rod forcibly dents the tissues inwards and momentarily stretches each side of the dent. This causes rupture of vessels in the marginal zones with a line of bruising, whereas the base of the dent becomes compressed and the vessels are not injured. When the rod is removed and the skin comes back to its normal position, the two sides of the depression remain as contused lines. In a bruise produced by a long rigid weapon, e.g., 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. 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. 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 line, the distance between which is roughly equal to the diameter of the whip. 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. Bruises from straps, belts or chains, leave a definite imprint.

Delayed Bruising: Deep Bruise may take several hours for it to surface wheras deepr bruise may not surface.A superficial bruise appears immediately as a dark-red swelling.Hence its advicable to reexamine 48 hours after the first examination.Similar may be the condition in Dead individual which is 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 and also due to draining of blood from vessels as a result of Postmortem examination making the Extravasated areas more prominent.Hence they are also called as comeout Bruises. The examination of whole body by ultraviolet light will sometimes clearly show otherwise undetectable areas of bruising.

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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 and bilirubin by the action of enzymes. The colour change is very variable, starts at the periphery and extends inwards to the center.

At first : Red-due to extravasation of blood as a result of rupture of capillaries.

18-24hrs: Dark Red-Due to distgration of RBS and staining of tissues due to Haemoglobin realeased as a result of action of enzymes from the tissues.

Few hours to 3 days: Blue due to deoxyhaemoglobin.

4th day: Bluish-black to brown as a result of formation of haemosiderin.

5 to 6 days: Greenish due to formation of haematoidin.

7 to 12 days: Yellow due to formation of bilirubin..

2 weeks: Normal.

It is difficult to estimate the exact age of a bruise with any degree of certainty and the fscotr responsible for formation and appearance of Bruise need to be considered.

The site of a bruise may contain crystals of haematoidin for a long period after the injury. 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 Cyanide or Carbon monoxide poisoning is likely to have a bright-red colour.

Medico-legal Aspects: (1) Patterned bruises may connect the victim and the object or weapon, e.g., whip, chain, cane, ligature, vehicle, etc. (2) Time of infliction of Bruise can be determined by colour changes. (3) The Size of bruise is proportional to the degree of violence.. (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.

Medico Legal shortcomings:: (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) Multiple contusions can cause death from shock and internal haemorrhage. (2) Gangrene and death of tissue can result due to ischemic Necrosis. (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. They are produced to make a false charge of assault.

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

LACERATIONS

Lacerated wounds are wounds in which the tissues are torn as a result of application of Blunt force to the body.Localised pressure by the impact causes displacement or crushing of tissue which sets up traction force causing tear or rupture of the tissues. Crushing occurs when the soft tissue are pinned against underlying Bone and displacement occurs when the underlying tissues are soft. They are also called tears or ruptures.

They are caused by blows from blunt objects, by falls on hard surfaces, by machinery, traffic accidents, etc. If the force produces bleeding into adjacent tissues, the injury is a ‘contused-laceration’ or ‘bruised-tear’. If the blunt force produces extensive bruising and laceration of deeper tissues, it is called “crushing” injury.

General Features of Lacerations:

a. Production: By Blunt Weapons or Objects.
b. Shape: Varies,usually Irregular.

c. Edges: Ragged and often Undermined.
d. Dimension: Depth varies, but magnifying glass examination reveals bridges of tissues connecting the edges.
e. Haemorrhage: Not pronounced..
f. Skin surrounding the wound margin: Bruising or Abrasion present

TYPES:

Split Lacerations: Splitting occurs by crushing of the skin between two hard objects. Blunt force on areas where the skin is closely applied to bone, and the subcutaneous tissues are scanty, may produce a wound which looks like an incised wound and is called incised-like or incised-looking wound. The sites are scalp, eyebrows, cheekbones, lower jaw, iliac crest, perineum and skin.

Stretch Lacerations: Overstretching of the skin, if it is fixed, will cause laceration. There is localized pressure with pull which increases until tearing occurs and produces the "Flap". It is seen in running over by a motor vehicle and in fractures.

Avulsions: Grinding compression by a weight such as lorry wheel passing over a limb or trunk may produce separation of the skin from the underlying tissues (avulsion) and crush the underlying muscles.

Tears: Tearing of the skin and tissues can occur from impact by or against irregular or sharp objects, such as, door handle of a car.

Characters:

Margins are irregular, ragged and uneven and their extremities are pointed or blunt.

Bruising is seen either in the skin or the subcutaneous tissues around the wound.

Deeper tissues are unevenly divided with tags of tissue at the bottom of the wound bridging across the margin.

Hair bulbs are crushed.

Haemorrhage is less because the arteries are crushed and torn across irregularly.

Foreign matter may be found in the wound.

Depth varies according to the thickness of the soft parts and the degree of force applied.

A laceration is usually curved.

The skin on side of wound opposite to direction of force is usually torn free or undermined.

Ante-mortem lacerations show bruising of margins, eversion and gaping of the margins and vital reaction. Lacerations are usually seen in accidents and assaults. Suicidal lacerations are very rare, as they are painful.

Medico-legal Importance:

The type of laceration may indicate the cause of the injury or the shape of the blunt weapon.

Foreign bodies found in the wound may indicate the circumstances in which crime has been

committed.

The age of the injury can be determined.

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.In splinter injuries due to explosions or Firearm a combination of Contusion,Laceration and Abrasion exist as pattern.Similar pattern is usually seen in injuries due to Road traffic collisssions.

INCISED WOUNDS

An incised wound(Cut, Slash, Slice) is a clean cut through the tissues, which is longer than it is deep. It is produced by the pressure and friction against the tissue by an object having a sharp cutting edge, e.g., knife, razor, scalpel, etc.

Characters:

(1) The edges are clean cut, well defined and usually everted and free from contusions.

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

3.The length is greater than its width and depth and has no relation to the cutting edge of the weapon.

4.It is usually spindle shaped due to greater retraction of the edges in the centre.

5.Haemorrhage is more as the vessels are cut cleanly.

6. It is deeper at the beginning because of greatest pressure. This is known as head of the wound. Towards the end of the cut the wound becomes increasingly shallow, and finally the skin alone is cut. This is known as tailing of the wound.

7.If the blade of weapon enters obliquely, one edge is beveled at the expense of the other; if the blade is nearly horizontal, a flap wound is caused.

AGE	Fresh	Haematoma formation.
	12 hours	The edges are red, swollen and adherent with blood and lymph; leucocytic infiltration.
	24 hours	A scab of dried clot is seen on the wound; vascular buds begin to form
	36 hours	The capillary network is complete; mitotic activity in the basal cells
	2 to 3 days	The wound is filled with fibroblasts and capillary buds grow in from the cut surfaces
	3 to 5 days	Definite fibrils are seen; vessels show thickening and obliteration
	1 to 2	Scar is formed

	weeks	

Medico-legal Importance:

(1) They indicate the nature of weapon.

(2) They give an idea bout the site of impact and direction of the force.

(3) The age of the injury can be determined.

(4) Position and character of wounds may indicate mode of production, i.e. suicide, accident or homicide.

Characters of Self Inflicted Incised Wounds:

(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.

Suicidal incised wounds: Circumstances should be examined, usually in an isolated, abandoned place examples room, place of residence. Relationship crisis, Financial Crisis, Career Crisis, Domestic Crisis are the main Factors, may or may not be associated with death note. The following are the character of the Wounds

a. **Site::** throat, wrist and front of chest, in general accessible regions of the body. On extremities its is usually found on the flexor surface of the wrists, outer side of the left thighs, and the front of the abdomen and chest. Suicidal wounds of chest are usually on the left side and directed downwards and inwards.

b. **Number and Depth::**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.

c. unintentional cuts: are found on the fingers, where the blade has been gripped.

d.**Clothing:** A person who commits suicide exposes his body by opening his clothes and then inflicts wounds.

Homicidal wounds:

a.Site:Any part of the body over a wider area and are usually horizontal.

b.Direction:. They are usually directed from below upwards.

c. Number and Depth: Multiple and deeper in nature.

d. Defensive Injuries:They are present generally on the extensor surface of Forearm,Arm,Front ofThigh and Legs.

e. Clothing: Usually associated with the Cut of the corresponding clothing.

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Accidental incised wounds:

They may be situated anywhere on the body, but are commonly seen about the hands. Usually caused by falling upon a sharp-cutting weapon or objects like broken metals,glasses.It may also be caused while handling sharp cutting instruments like knife.

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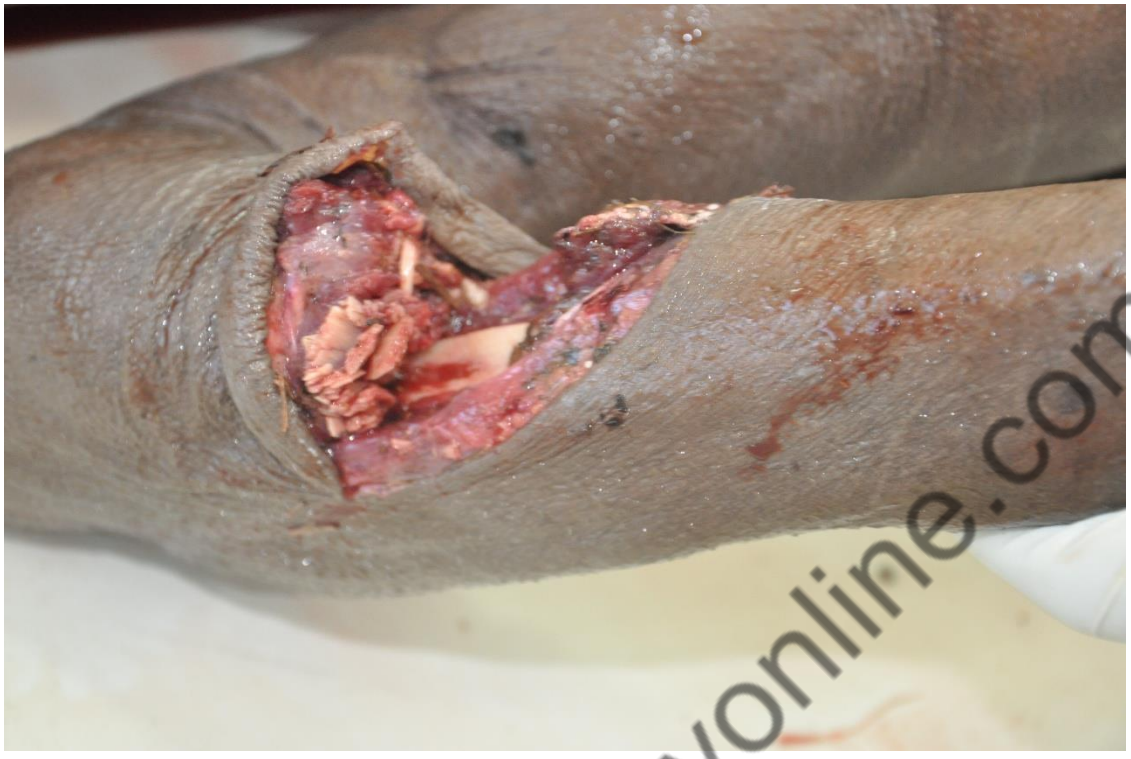
Scene

The suicidal person finds some quite place where he is unlikely to be disturbed, such as his/her bedroom or a locked bathroom. If the person kills themselves outside, the body may not be found for several days. When a person cuts his throat indoors he will often to it in front of a mirror and splashes will frequently be found on the glass itself. A homicidal assault may lack the privacy associated with a suicide as regards locality of the scene. In case of suicide one rarely finds any associated with a suicide as regards locality of the scene. In case of suicide one rarely finds any disorder at the scene. A suicidal person may walk considerable distances after cutting his throat or wrist and during this period of locomotion he may collapse and get up several times before the final collapse and at each place where he collapses or even stops, pools of blood are to be expected where a homicidal attach has taken place or considerable disturbance at the scene is almost invariable, unless the victim was rendered unconscious before the attach or was very old or where more than one assailant was involved.

Chop Wounds: They are wounds caused by a blow with the sharp cutting edge of a fairly heavy instrument like an axe, butcher's knife, and etc. The dimensions of the wound correspond to cross-section of penetrating blade. The margins are sharp and may show slight abrasion and bruising with marked destruction of underlying organs. Usually the heel of the axe strikes the surface first which produces a deeper wound than the toe wound. The deeper end indicates the position of the assailant. If the extremities are attacked there may be complete or incomplete amputation of the fingers or other parts. Most of these injuries are homicidal.



PUNCTURED WOUNDS

They are caused by Long narrow instruments with Blunt or pointed ends.Punctured wounds are described as Penetrating when they pierce deeply into tissues and as Perforating when they Transfix tissues and cause exit wounds.If a Sharp pointed weapon is used it is described as Penetrated or Perforating Incised Wound.If a Blunt Pointed instrument is used it is described as Penetrated or Perforating Lacerated Wound

The wound of entry is larger with inverted edges, and the wound of exit is smaller 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.

General **Characters**:

1.Production:By Pointed or Sharp Weapon

2.Shape:Linear or Irregular according to the shape of the Weapon.

3. Edges: Edges are often Everted from withdrawal of Weapon.

4. Dimensions: Depth greater than Length and Width.

5. Haemorrhage: Varies,profuse if ‘vessels are cut in depth.If cavity involved external haemorrhags is minimal.

6. Skin around the Margins: Rarely Contused.

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. Hence then viscera of a dead body on the autopsy table are not in the same positions, as when the same person was alive In a penetrating 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 penetrating wound is on the back of the chest, the depth of the wound will be less, as the lungs will collapse posteriorly.

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.

Complications: 1. The wound may get infected due to the foreign material carried into the wound. 2. Air embolism may occur in a Penetrating wound involving jugular veins. 3. Pneumothorax may occur in Penetrating wound involving Chest , 4. Asphyxia due to inhalation of blood in penetrating wound of Respiratory passage and 5. External hemorrhage is slight but there may be marked internal haemorrhage or injuries to internal organs.

Concealed Puncture Wounds: These are puncture wounds caused on concealed parts of the body, such as nostrils, fontanella, fomix 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.

Medico-legal Importance:

(1) The shape of the wound may indicate the class and type of weapon.

(2) The depth of the wound will indicate the force of penetration,

(3) Direction and dimension of the wound indicate the relative positions of the assailant and the victim.

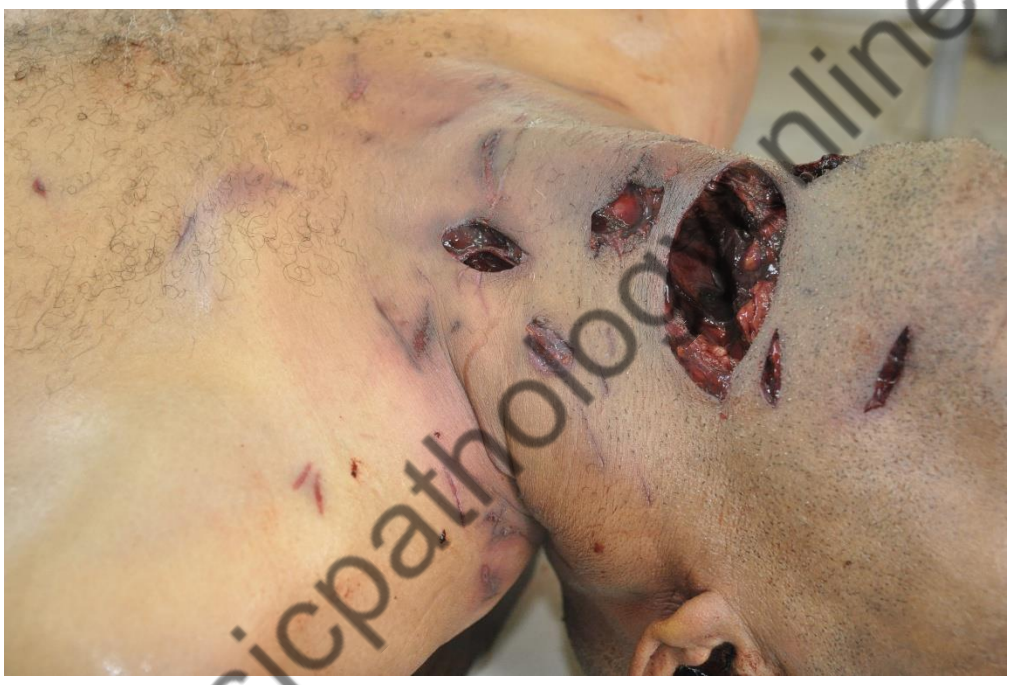
(4) The age of the injury can be determined.

(5) Position, number and direction of wounds may indicate mode of production i.e. suicide, accident or homicide.

(6) If a broken fragment of weapon is found it will identify the weapon.

Stab wounds are mostly suicidal or homicidal.

Accidental wounds are rare.



CIRCUMSTANCES OF INCISED AND PUNCTURED WOUNDS:

(1) Accidental Wounds: They are caused by (i) Fall upon a sharp edged object. (ii) Impact by sharp objects, e.g., glass pieces. (iii) Unintentional cut or stab by sharp edged or pointed instrument, e.g., knife, razor blade, house-hold utensil, etc.

(2) Suicidal wounds:

They are multiple and parallel, and superficial in any one area.

They are present in several limited accessible areas of the body, such as neck, wrists, groin and rarely on backs of legs or on chest.

Hesitation marks or tentative cuts are multiple, small and superficial, often involving only the skin and are seen at the commencement of the incised wound.

When a safety razor blade is used unintentional cuts are found on the fingers where the blade

has been gripped.

More than one method may be used for suicide.

In right handed persons, the most severe wounds are often found on the left side of the body.

Suicidal cut-throat wounds are usually seen above the thyroid cartilage, the direction is from left to right, the edges are ragged due to overlapping of multiple superficial wounds, hesitation cuts are present, tailing is present, they are multiple upto 20 to 30, superficial and parallel wounds may be seen on other parts of the body,, the clothes are not cut and circumstantial evidence may be helpful.

(3) Homicidal wounds:

Multiple gaping wounds on any part of the body including back.

(ii) Defence wounds may be found.

Hesitation wounds are absent.

Mutilation of the body, particularly the breasts and genitals, indicate sexual murder.

Post-Mortem Wounds: (a) Intentional mutilation: (i) Sex crimes. (ii) Sadistic murders. (iii) Attempted concealment of body by dismemberment. (b) Unintentional mutilation.

DEFENCE WOUNDS: Defence wounds indicate homicide. 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. **They are due** to the immediate and instinctive reaction of the victim to save himself, either by raising the arm,Forearm or Front of Thing and Leg to prevent the attack, or by grasping the weapon. If the weapon is blunt, bruises and abrasions,lacerations and fractures are present. 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 weapon, 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 is seen in the web between the blade is grasped. Rarely, defence wounds are found on the feet or legs, e.g., when the victim kicks at he knife, or curls up and tries to cover vital areas with his legs.

FABRICATED WOUNDS: 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. The direction is from behind forwards on the top of the head, from above downwards on the upper arm, from below upwards on the forearms, variable on the legs and vertical on the abdomen. 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.

Therapeutic Wounds: Doctors producing the wounds during the course of Theraphy or treatment of a patient in good faith and his benifet, 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.

WOUNDS AND WEAPONS:

Agents implying intent but not necessarily premeditation and frequently not homicidal

a) Hands b) Foots c) Boots d) Teeth

Agents implying intent but not usually homicidal

a) Whips, canes, ropes etc. b) Knuckle ,dusters.

Heavy agents often associated with homicidal attacks

a) Clubbing instruments b) Axes ,Knives and choppers

WOUNDS AND SCENE

An assault with a clubbing instrument may take place in almost any locality either in the outdoors or indoors. When it occurs indoors in a confined space, the injuries may be modified by the limitation of movement imposed upon the assailant. The amount of disorder at the scene will depend upon whether the victim was taken unawares and rendered unconscious i.e. incapable of defending himself or whether he has been able to make a determined effort to protect himself. In the letter case great disorder may be found. Of the findings at the scene of this kind the widespread distribution of blood in the forms of spurts, smears and pools is the most significant feature.

If only on blow has been struck, even though an extensive laceration has been inflicted, no blood may be projected from the wound because the leading edge of the instrument as it strikes the body expels blood from the tissue beneath it. the instrument itself may not even be blood stained. Any subsequent blows will force blood under considerable pressure through the torn vessels in the wound previously inflicted and this blood may be projected many feet in all directions, striking the walls, furniture, ceiling and of course the assailant. At the scene one may find blood as spurts or projected droplets from arterial bleeding or repeated blows to the body. Smears may be found where the injured person has brushed against the wall or other object. After the first laceration has been inflicted, the striking edge for a considerable distance behind, above or to the side of the assailant depending upon how he is using the instrument. The examination of collections or pools of blood may reveal where the injured person was lying and distribution of blood upon the clothing will provide valuable information as to the position of body after the injuries were received. Any weapon found at the scene must be handled with extreme care – it may have some blood or hair of the victim adherent to it and it may bear the fingerprints of the assailant. Such a print is of great importance that means the person whose blood stained fingerprint is present on the instrument, must have handled the weapon after the initial injury was inflicted.

CAUSES OF DEATH FROM WOUNDS:

Immediate Causes:

(1) Haemorrhage,

(2) Shock,

(3) Reflex vagal inhibition.

(4) Mechanical injury to a vital organ.

Remote causes:

Infection.

Gangrene or Necrosis.

Crush syndrome.

Neglect of injured person.

Surgical operation.

Natural diseases.

Supervention of a disease from a traumatic lesion.

Thrombosis and embolism.

Fat embolism.

Air embolism.

SCENE OF DEATH – STAB WOUND

At the scene of death the principal objective of the medico-legal investigation is the recognition of the manner in which the injuries were sustained.

Obtain information about the circumstances of death from police and witness at the scene

Photograph the scene and make sketches before anything is marked

Make a general observations about the scene. Not any evidence of struggle, such as overturned furniture or trampled ground.

Describe the position of the body and the hands

Examine the body including basic hands and forearms for defense wounds. Make notes and record in body diagram.

Note whether the weapon is present or absent. If present record position of weapon in relation to body. If weapon is in person's hand, maintain whether it is loosely held or tightly grasped. The presence of weapon loosely in hands does not rule out homicide but weapon held tightly indicate suicide. If the weapon is absent from the body, crime should be suspected

If weapon present handle with care to preserve Finger Print, blood stains hair and fibers, retain in appropriate containers

Let the police handle the weapon. Request them to make the weapon available for the evaluation of injuries often at has been examined for FP and trace evidence

If not found, advice the police about the type of weapon that is likely to have been used.

if suicide is suspected, look for suicide note and inquire about the personal history of the decedent – social, financial, domestic and health problems, history of depression and suicidal threats.

11.If the injuries are suspected to be accidental, identify the object causing them.

Describe the position of the body and the hands.

Describe the clothing and note any tears, missing buttons and so on. Ascertain whether the weapon penetrated the clothing or whether the clothing was displaced from the area injury.

Note amount of bleeding at the scene. It indicates length of survival.

Make of note of injuries and record them on body diagram.

Tears of clothing's and injuries of genital areas should be noted.

AUTOPSY:

Photograph the clothing and injuries on the body.

Record injuries by words and sketches death, shape, size etc.

Whether before/after death

Cut, stab or laceration and time of inflection.

Kind of weapon-width/one or two edge sharp or blunt

Collect foreign materials such as hair, grass, fibers and so on that may be in the wound.

From depth estimate the minimal length of the blade of the weapon

Determine the direction of the weapon with the body

If homicidal Look for defense wounds.

Assess the findings to determinate rapidity of death of collapse and ability to wake/perform acts

If several wounds determine which one is fatal

Record the findings suggesting sexual assaults. Examination of external and internal genitals and face

Evaluate role of contributing factors such as pre-existing disease or intoxication in causing death

If suspected weapon is present, determine whether the wounds found on the body are corresponding with those caused by such a weapon.

MANNER OF DEATH.

In homicidal stabbing

Accessible and Inaccessable areas of the body.

Through clothing

Multiples stabs.

Single stab on Vital Region like Heart,spleen.

Associated injuries of struggle like Defensive or Offensive injuries.

signs of struggle.

Weapon may or maynot be at the scene.

Suicidal Stabbing

Circumstantial evidences related to Financial,drug,mental,marital etc to be studied.

Presence of Suicidal note in his clothing,hand or surroundings.

Accessable areas of the Body

Area Unclothed.

Multiple Superficial Stabs followed by Fatal wound

No evidence of Struggle, offensive or defensive injuries.

Weapon at the Scene.

Accidental

Circumstances leading to accident present.

Weapon of causation present.

No evidence of Offensive or Defensive Injuries.

Multiple Stabs unlikely

VOLIATIONAL ACTS IN WOUNDS:

This are voluntary activity before death in a rapidly Fatal injuries.

In certain cases the Medical witness may be asked whether it would have been possible for the deceased to have performed some voluntary act,such as walking,speaking,after the infliction of the injury though the existing injuries are in normal course cause rapid death.The biological variation in Humans is know to exist and so is the instincts and reaction to injuries.A Cut throat wound to neck,for instance may rapidly cause unconscious or fatality in one person,while another person with a similar wound may be capable of walking, running or doing some actions for a considerable time before collapsing Thus the activity may not be uniform,predictable or calculated.Unless it can be shown that an injury would have been immediately incompatible with life,it is seldom possible to state that a deceased person couldnot have performed some activity before his death.

Thermal Injuries

BURNS

A burn is an injury which is caused by application of heat or chemical substances to the external or internal surfaces of the body, which causes destruction of tissues. The minimum temperature for producing a burn is about 44^{o}C for an exposure of about 5 to 6 hours or about 65^{o}C for two seconds are sufficient to produce burns.

Varieties of Burns: The external appearances of burns vary according to the nature of the substance used to produce them.

A highly heated solid body or a molten metal, when applied to the body for a very short time may produce only a blister and reddening corresponding in size and shape to the material used. It will cause destruction, or even charring of the parts, when kept in contact for sometime. The epidermis may be found blackened, dry, and wrinkled. The hair may be singed or distorted.

Burns produced by flame may or may not produce vesication, but singeing of the hair and blackening of the skin are always present. Hair singed by the flame becomes curled, twisted, blackish, breaks off or is totally destroyed. Roasted patches of skin or deeper parts may be seen.

Burns caused by kerosene, oil, petrol, etc. are usually severe and produce sooty blackening of the parts and have a characteristic odour.

Burns caused by explosions in coal mines or of gunpowder are usually very extensive and produce blackening and tattooing due to driving of the particles of the unexploded powder into the skin.

Burns due to X-ray and radium vary from redness of the skin to dermatitis, with shedding of hair and epidermis and pigmentation of the surrounding skin. Severe exposure may produce burns with erythema, blistering or dermatitis, or ulceration with delayed healing and ill-formed scars. Fingernails may show degenerative changes and wart-like growths. Infra-red rays may cause necrosis of the skin.

Burns caused by ultraviolet rays (the sun or mercury vapour lamp) produce erythema or acute eczematous dermatitis.

Burns from corrosive substances show ulcerated patches and are usually free from blisters' hair is not singed and red line of demarcation is absent. They show distinct coloration and are usually uniform in character. Strong acids produce dark leathery burns upon the skin. Strong alkalis cause the skin to slough and leave moist, slimy, grayish areas. Hydrofluoric acid and bromine cause necrosis of the skin and tissues.

Electrical burns.

Degree of Burns: Dupuytren divided burns into six degrees, but they were merged into three degrees by Wilson. The precise depth of a burn can be measured by a high frequency ultrasound device.

Epidermal: (first and second degree Dupuytren).

Dermo-epidermal: (third and fourth degrees Dupuytren).

Deep: (fifth and sixth degrees, Dupuytren): In this, there is a gross destruction not only of the skin and subcutaneous tissue, but also muscles and bones are destroyed, and as such the burns are relatively painless. The appearances are similar to those of the second degree, but in a more severe form. The burnt part is completely charred.

Effects: The effects depend on:

The degree of heat: The effects are severe, if the heat applied is very great. The body of an adult does not burn completely in a burnt house, as the temperature usually does not exceed 650^{o}C. For

purpose of cremation, a human body has to be incinerated for one and half hours at 1000ºC. The ashes weigh 2 to 3 kg., and contain bone fragments which can be identified as human.

The duration of exposure: The symptoms are more severe if the heat is applied for a long time.

The extent of the surface: The estimation of the surface area of the body involved is usually worked out by the "rule of nine", 9% for the head and each upper limb; 9% for the front of each lower limb; 9% for the front of chest; 9% for the back of chest; 9% for the front of the abdomen; and 9% for the back of abdomen, 99% of the body. The remaining 1% is for the external genitalia. Involvement of 50 percent of the body surface will prove fatal even when the burns are only of the first degree.

The site: Burns of the head and neck, trunk or the anterior abdominal wall are more dangerous.

Age: Children are more susceptible, old people less.

Sex: Women are more susceptible.

Causes of Death: (1) Primary (neurogenic) shock due to pain, etc. (2) More than half of deaths from burns occur within the first 48 hours usually from secondary shock, due to fluid loss from burned surface. (3) Toxaemia, due to absorption of various metabolites from the burnt tissue persists up to 3 to 4 days. (4) Sepsis is the most important factor in deaths occurring 4 to 5 days or longer after burning. (5) Biochemical disturbances, secondary to the fluid loss and destruction of tissue, e.g., hypokalemia. (6) Acute renal failure, due to lower nephron nephrosis occurs on the third or fourth day. (7) Gastrointestinal disturbances, such as acute peptic ulceration, dilation of the stomach, haemorrhage into intestines. (8) Oedema of glottis and pulmonary oedema due to inhalation of smoke containing CO and CO_2, if the person dies in a burnt house. (9) Accident occurring in an attempt to escape from a burning house or by injuries due to falling masonry, timber or other structures on the body. (10) Pyaemia, gangrene, tetanus, etc. (11) Fat embolism is rare. (12) Pulmonary embolism from thrombosis of veins of the leg due to tissue damage and immobility.

Post-mortem Appearances:

External: The clothes should be removed and examined for the presence of smell of kerosene, petrol, etc. They should be put into airtight bottles and sent for chemical analysis. It is difficult to determine the time of death as body temperature, post-mortem hypostasis and rigor mortis cannot be assessed. The burnt areas will be found reddened and blistered or charred. Blisters may be present either in the main burn or as islands beyond the periphery. The whole of the burned area may form one large blister or be confluence of blisters. The degree of burning in each area should be assessed. Hair is singed, or completely burnt. In lesser degrees of burns, ends are bulbous at intervals. Heat rigor may be observed in the muscles. Portions of the body where clothing is tight, e.g., under the belt, shoes, brassier or buttoned collar are often comparatively unaffected. Sometimes, skin and hair in the armpits and the gums are spared. The colour of light hair changes on exposure to heat. At about 120ºC for 10 to 15 minutes, brown hair becomes slightly reddish. There is no change in the colour of the black hair. The face swollen and distorted. The tongue protrudes and may be burnt due to the contraction of the tissues of the neck and face. Froth may appear at the mouth and nose due to pulmonary oedema caused by heat irritation of the air-passages and lungs. In the hands, the skin detaches as glove, including the fingernails. By removal of the

superficial layers of the skin by wiping or rubbing, tattoo marks become visible. The blisters of a second degree burns cannot be distinguished from blisters seen in CO poisoning, deep coma, ante-mortem and post-mortem gasoline exposure and peeling of the skin seen in the early stages of putrefaction. When these various types of blisters burst, they leave a pale, moist, raw surface which becomes yellow, tan and finally dark brown and leathery as it dries.

Pugilistic Attitude (boxing, fencing, or defence attitude): The posture of a body which has been exposed to great heat is often characteristic. The legs are flexed at the hips and knees, the arms are flexed at elbows and wrists and held out in front of the body, all fingers are hooked like claws, contraction of paraspinal muscles often causes a marked opisthotonus, in an attitude commonly adopted by boxers. This stiffening is due to the coagulation of proteins of the muscles and dehydration which cause contraction. The flexor muscles being bulkier than extensors contract more. It occurs whether the person was alive or dead at the time of burning.

Heat Ruptures: If the heat applied is very great, skin contracts and heat ruptures occur, either before or after death. They are produced by splitting of the soft parts. These splits may be anywhere, but are usually seen over extensor surfaces and joints. These ruptures or splits in the skin may be several centimeters in length, and superficially they may resemble lacerations or even incised wounds. They can be differentiated by: (1) Absence of bleeding in the wound and surrounding tissues, since heat coagulates the blood in the vessels. (2) Intact vessels and nerves are seen in the floor. (3) Irregular margins. (4) Absence of bruising or other signs of vital reaction in the margins.

Sometimes, the charred skin cracks easily when an attempt is made to remove the body from a house destroyed by fire. These tears are commonly seen around joints, especially the elbows, shoulders and knees.

“Flash burns” refer to thermal burns due to sudden, brief exposure to flame. This type of exposure is common in explosions, or ignition of fine particulate material or upon ignition of highly inflammable liquids. All exposed surfaces are burned uniformly. If clothing is ignited, a combination of flash and flame burn occurs.

Human bodies burn readily, especially when the subcutaneous fatty tissues have ignited. Often, some parts of the body are preserved, if they are protected from the flames. In sitting persons, the buttocks may be spared; if the head falls forward between the knees, the abdomen is spared. The hands and feet may drop off if the burning is sudden and intense, and they may be preserved with slight damage because they fall away from the source of fire. Flexion of the limbs by heat may cause tumbling of a burnt body from a bed or chair to the floor, if the body was not well balanced. Partial burning of the abdominal wall associated with gas expansion within the intestines may produce rupture of the abdominal wall, in the charred burnt victim. The intestines may protrude through this defect. Flame burns usually have a patchy distribution and vary in size and shape. Sometimes, the body may be covered with a black or brown layer of smoke which does not penetrate into skin creases. On straightening the flexed neck or limb, the paler skin in the crease is exposed which may mimic a ligature mark. In severe burns, the skin may be stiffened, yellow-brown and leathery. Drying after death leaves a stiff, parchment-like surface. Muscles under the burnt area are pale, brownish and part-cooked. This occurs after death due to heated environment.

Black, brittle masses are found in the tissues merging into cooked dry muscle beneath. Brunt bone has a gray-white colour, often showing a fine superficial network of heat fractures on its cortical surface. The soft tissue of the face may be completely burnt exposing the skull. The outer tables of the exposed cranial vault may show a network of fine criss-crossing heat fractures. If the flame is unchecked, the body will be reduced to a shapless, carbonaceous mass and finally to heap of grey and yellow ashes.

Establishment of Identity: In a charred body, the weight and stature are unreliable, as they are greatly altered due to drying of the tissues, skeletal fractures, and pulverization of intervertebral discs due to the heat. The stature may be less by several centimeters and weight loss may be up to 60%. The features are changed due to contractions of the skin. Moles, scars and tattoo marks are usually destroyed. Dental charts should be prepared and X-rays of the jaws taken, which can be compared with previous charts of the suspected person. Complete X-rays of the body of the victim are useful to locate possible old fractures, bony abnormalities or foreign bodies. In a badly charred or incinerated body, the sex can be determined by finding the uterus or prostate which resist fire to a marked degree, and by pelvic bones, and age by teeth and by observing centers of ossification in the bones and the condition of the epiphyses. If the whole body is destroyed, personal effects such as keys, watch, buttons, belt-buckle, cuff-links, etc., may help in identity.

Internal: Heat haematoma occurs when the head has been exposed to intense heat, sufficient to cause charring of the skull. It has the appearance of extradural hemorrhage, but is not accompanied by any signs of injury by blunt force. It consists of a soft, friable clot of light chocolate color, and may be pink, if the blood contains CO. The clot has a honeycomb appearance. The thickness of the clot varies from 1.5 to 15mm, and the volume up to 120 ml. The adjacent brain shows hardening and discoloration from the heat. The distribution of the clot follows closely the distribution of the charring of the outer table of the skull. The parietotemporal region is the most common site of such hemorrhage. The mechanism of its development is obscure. Possibly, the blood, may come from the venous sinuses or the diploic veins by the shrinkage of the brain due to heat.

The skull fractures occur most commonly in areas where the skull has been severely burned. There are two types of thermal fractures of the skull. (1) Intracranial increase of steam pressure causes separation of ununited sutures or an intracranial explosion occurs, producing fractures with gaping defects and widely separated bony margins. (2) The fracture occurs due to rapid drying of the bone with contraction, and only involves the outer table of the skull. In this type there is no displacement, and the lines of fracture are frequently stellate. Skull fractures are usually seen on either side of the skull above the temples. They consist of several lines which radiate from a common centre. Heat fractures usually do not involve the sutures of the skull even in young persons with un-united sutures. Heat fracture may cross a suture line. Peculiar, characteristically curved fractures are often seen in bones of extremities exposed to very high temperatures.

Even in cases of severe external charring, the internal organs are usually well preserved, as the tissues of the body are poor heat conductors. Sometimes, brain, liver, lung, etc., may be cooked, i.e., hardened and discoloured. In death due to burns, the CO levels in the blood will be more than 10% and may reach 70 to 80%, though children and old person die at levels of 30 to 40%. The blood is cherry-red, which may change to brownish due to heat. The level of CO saturation of the

blood is dependant on concentration of CO in the inhaled air, the duration of exposure, the rate and depth of respiration, the haemoglobin content of the blood and the activity of the victim. CO may be absent in blood due to various reasons, such as rapid death, convection air currents, low production of CO, flash fire (as in the conflagration of a chemical plant.), inhalation of superheated air resulting in death by suffocation, in warfare, or in an explosion when death is instantaneous. If death has occurred from suffocation, aspirated blackish coal particles are seen in the nose, mouth, larynx, trachea, bronchi, esophagus and stomach and blood is cherry-red. Such particles are embedded in frothy mucus which covers the congested mucosa. The presence of carbon particles in the terminal bronchioles on histological examination is absolute proof of life during the fire. The soot is better seen by spreading a thin film of mucus on a clean sheet of white paper. The amount of soot in the air-passages depends on the type of fire, the mount of smoke produced and the duration of survival in the smoke-contaminated atmosphere. Presence of carbon particles and an elevated CO saturation together are absolute proof that the victim was alive when the fire occurred. If the mouth is open, some passive percolation of soot may be found at the back of the pharynx, but it cannot be carried beyond the vocal cords, and also it is not found in the lower esophagus and the stomach. In absence of CO in blood and soot in the airways, death may result possibly due to poisoning with CO_2 and/or O_2 deficiency. Poisonous gases like cyanide and oxides of nitrogen are produced due to burning of plastic and synthetic material. Burning of nitrogen containing substances, e.g. nitrocellulose film may liberate nitrogen oxide and nitrogen tetroxide. Burning of wool or silk liberate ammonia, hydrogen cyanide, hydrogen sulphide and oxides of sulphur. All these gases contribute to death. depending on the materials burning in the fire, various levels of cyanide are found in the blood, but the levels are usually less than 0.3 mg.%. Cyanide can be produced in significant concentration by decomposition of the body. Blood should be preserved by fluoride for analysis of cyanides. If flame or superheated air is inhaled, burns are seen in the interior of the mouth, nasal passages, larynx and air-passages with destruction of vocal cord epithelium and acute edema of the larynx and lungs. Death may occur rapidly by shock or acute respiratory insufficiency. The interior of the larynx, trachea and main bronchi may be thickened and blanched, or reddened. If the victim survives for a few days, inflammatory changes occur in the larynx, with sloughing of mucosa, ulceration and secondary infection.

Haemoconcentration is present, and frequently there is some tissue edema and excess of fluid in the serous cavities. The brain is usually shrunken, firm and yellow to light-brown due to cooking. The dura matter is leathery. The dura may split and the brain tissue may ooze out, forming a mass of frothy paste. The pleurae are congested or inflamed. The lungs are usually congested, and show marked edema; they may be shrunken and rarely anaemic. The vessels of the lungs may contain a small amount of fat due to a physico-chemical alteration of fat already present in the blood. Visceral congestion is marked in many cases. Petechial haemorrhages are usually found in the pleurae, pericardium and endocardium. The heart is usually filled with clotted blood. There may be inflammation and ulceration of Peyer's patches and solitary glands in the intestines. Occasionally, ulcers are produced in the duodenum (Curling ulcers), about the tenth day in extensive burns of the body. Curling's ulcers are usually sharply punched-out mucosal defects, which may be superficial or deep. Petechiae of stomach and duodenum, often with erosions, occasionally acute ulcers, is a more common finding. The large boel may also be involved. The spleen is enlarged and softened. The liver may show cloudy swelling. Fatty liver is not due to burns, but due to treatment with tannic acid. Jaundice may occur. The kidneys may show cloudy swelling, capillary thrombosis and infarction. The adrenals may be enlarged and congested. When

more than 30% of the skin surface is burnt, haemoglobinuria occurs. Depending on the materials burning in the fire, various levels of cyanide are found in the blood but the levels are usually less than 0.3 mg%.

Laryngeal Oedema: It may be caused by allergic anaphylactic reactions, infections, tumors, inhalation of flame or superheated air, inhalation of irritant gases, etc. the amount of oedema will decrease with post-mortem interval and only wrinkling of mucous membrane may be present. Microscopically, eosinophils may be seen.

Blood should be obtained from the heart or major vessels and placed in a tightly stoppered container. It need not be collected or kept under oil. If blood is clotted, the clot should be preserved.

Age of Burns: The ageing of the burns is very inaccurate and depends upon the agent, the extent, and their depth. Redness appears immediately, and vesication in about an hour. The exudates beings to dry in 12 to 24 hours and forms a dry, brown crust within 2 to 3 days. The red inflammatory zone disappears in 36 to 72 hours, and pus may form under sloughs. Superficial sloughs fall off in 4 to 6 days, and deeper sloughs within two weeks. After this, granulation tissue covers the surface and a scar is formed after several weeks.

Ante-mortem and Post-mortem Burns: In ante-mortem burns, a zone of hyperaemia (line of redness), which varies in width, but is usually 5 to 20 mm. is present at the edge of the burnt area, except in cases of immediate death. it is due to oedema of tissues and capillary dilatation and merges with the edge of the burn which may shows blistering or charring. It involves whole thickness of true skin. It is permanent and persists after death. if the whole body is burnt, line of redness will be absent. The ante-mortem blister appears as a raised dome and contains gas or fluid. The base and periphery show reddening with swollen papillae. Post-mortem blister is dry, hard and yellow. The protein content of serous fluid is not of much value to differentiate ante-mortem and post-mortem burns. In ante-mortem burns, the skin adjacent to burnt area shows an increased reaction for SH groups in all layers, and increase in enzyme reaction. Acid mucopolysaccharides are present in the superficial zone of burnt area. Burns produced shortly before or after death cannot be distinguished either by naked eye or by microscopic examination.

Circumstances of Death: The distribution of burns on the clothing may indicate the manner in which it was ignited, the posture of the victim at the time, the path taken by flames and to discover that unburnt cloth was saturated with some inflammable material. Splash patterns burnt into the floor and floor coverings, holes in the floor, particularly holes of the 'tongue and groove' type and the characteristic odour of petroleum fuels and solvents are all useful indicators of the use of inflammable material. Differentiation is mostly a matter for the police investigation. The inhalation of CO often causes severe muscular in coordination, weakness, and confusion due to which the victim is unable to escape and dies of asphyxia, the body being burnt after death.

Accident: Large numbers of deaths are accidental. Infants, children, epileptics, intoxicated or drugged persons or helpless from other causes may fall into a fire. When an intoxicated person goes to bed smoking, and drops a lighted cigarette, he may die due to burns. Lamps or stoves may

explode and set fire to the clothes. Clothes of women may catch fire accidentally while cooking. In such cases, burns are usually found on the front of thighs, abdomen, chest and face. There may be severe burning of the hands due to the victim trying to extinguish the fire by beating out the flames. The feet and ankles are usually not burnt. Multiple deaths from burns may result from plane crashes or automobile accidents. In industry, burns may be caused by explosions from inflammable liquids and by flashes from furnaces. The skin resting on the surface is well preserved.

Suicide: Occasionally, women commit suicide by pouring kerosene on their clothes before setting fire to themselves due to domestic worries, disappointment in love or acute or chronic disease. Extensive burns are seen over the whole of the body; only the skin folds, such as the axillae and the perineum being spared. Sometimes, a person may keep a piece of cloth in her mouth to suppress her cries. Sometimes, suicidal burning is a mode of public protest. In case kerosene, petrol, etc. is found on the body including head hair in high amounts, it is likely to be either suicide or homicide.

Homicide: Murder by burning is rare. If an inflammable fluid such as kerosene, petrol, etc., is poured on a person lying on his back and then burnt, there will be burning of the sides of the neck, sides f the trunk, between the thighs and other areas, especially if the clothing is absent in those areas, as the fluid runs downwards. Sometimes, fire, hot metals, boiling water and corrosive substances are used with criminal intent. A drunken man may push or throw his wife or child on the fire, and sometimes lighted lamps may be used as missiles. Burns may be inflicted on the pudenda of women as a punishment for adultery. Attempts may be made to burn a body after homicide with the object of concealing the crime. In such cases, the body should be examined for marks of violence, e.g., stab wounds, bullets, strangulation, etc. In cases of Individual dying due to Extensive burns under Alcohol intoxication/influence of drugs no accidental nature should be specifically ruled out.

Self-inflicted Burns: Burns are sometimes self-inflicted in order to support a false charge.

CONFLAGRATED HUMAN REMAINS - UNBURNT AND BURNT BONES

In some instances, burnt bones and ashes are forwarded to the forensic pathologist for inspection, if the police suspect some foul play after a body is partially or completely burnt. In an ordinaryhouse fire, the temperature seldom exceeds 1200ºC. It is, therefore, unlikely that the body of an adult will burn so completely as to leave no trace. If the body is not completely consumed, fragments of bones left would afford sufficient evidence to indicate whether they were human or not. The combustion of a body is rarely so complete as to reduce it to ashes. Hence, by shifting the ashes through sieves, fragments of bones can be collected and identified by a careful study.

Incineration of an adult human body for the purpose of cremation requires 1 ½ hour at 1600-1800ºC, and the resultant ashes weigh about 4-6 kg. Such human ash contains bone pieces which may still be identified.

Todd and Krogman working on a body burned in an auto, concluded:

When the soft tissue around the bones are small in amount (thin) the bones show sharp heat induced

fractures of the skull and limb-bones (usually transverse), charring, calcining and splintering, while with thick soft tissue, e.g., in femur, pelvis and nuchal areas of skull the substance of the bones shows the molten or guttered condition characteristic of fusion by heat.

A bone is white in appearance when burnt in the open, and black or ash grey when burnt in the closed fire. A burnt bone preserves its shape, but falls to powder when pressed between the fingers. It is said that it will be reduced to charcoal if treated with hydrochloric acid, but this is not necessarily true. If it is burnt to such an extent that organic matter is destroyed, no charcoal will be left on adding acid. When exposed to very high temperature, characteristically curved fractures may be produced in long bones and skull. A bone becomes so brittle and friable on prolonged exposure of the fire victim to such intense heat, that it is readily fractured during transport of the body, or its being moved, or during examination. A hyoid bone may similarly break on manipulation. A forceful stream from a fire-hose can fragment a bone like the femur, rendered brittle by exposure to such high temperatures. The skull bursts due to the formation of steam within the skull cavity as a result of intense heat. Such explosive post-mortem fractures are accompanied by gaping defects and separation of non-united sutures and protrusion of brain matter. Intense heat can lead to desiccation of skull, with the production of post-mortem thermal linear fracture, commonly located on either side of the skull, above the temples. They usually consist of several lines which radiate from a common centre. If the appearance is not typical, distinction from an ante-mortem skull fracture may be difficult. Besides, post-mortem mechanical fracture of any bone, due to the fall of a wall or a beam can also occur. It is important, therefore, to distinguish between post-mortem thermal fracture and post-mortem mechanical fracture.

In cases of suspected poisoning by some mineral, eg, arsenic, all the available ashes and burnt bones should be preserved for chemical analysis. This is because, despite its volatility, it is possible to detect arsenic in large pieces of burnt bones mixed with ashes in cases of arsenic poisoning, for the following reasons.

Much of the arsenic in bones is converted into arsenates, partially replacing the phosphates of the bones. Arsenates are non-volatile; hence arsenic can be detected in the bones even after strong heating for a long time.

Even if all the arsenic were present in the bones in the form of arsenic trioxide or some other volatile form, all the arsenic is not likely to be lost during the process of cremation, as complete combustion of a body does not, as a rule, occur in India. Hence, some of the volatilized arsenic is liable to be condensed on the cooler parts of the unburnt funeral pyre, where its presence may be detected.

When arsenic trioxide is heated with salts of sodium or earth group, part of the arsenic is converted into arsenite and becomes non-volatile.

Burns

The medico-legal investigation of a death from burns should be aimed at answering the following questions.

Was the person alive before fire started?

Did the burns cause death

If death was from causes other than burns, did the burns contribute to death?

Were there any natural diseases or injuries that could have caused death or contributed to

Were the burns sustained accidentally or did the person commit suicide?

Was the death of crime?

Was there any attempt to conceal crime?

What was the cause of the onset of fire?

What evidence was found to identify the decedent?

Occasionally, with multiple fatalities, one may be asked who died first.

Examination of scene may reveal information regarding the cause of fire and may also in the site of origin of the fire. The overall study of the circumstances of death at the scene may information concerning the manner of death.

From the scene of death all personal belongings such as keys, watch band, bell buckle, button cuff links and pieces of unburned clothing should be collected. These can be helpful in established the identity of the decedent.

HEAT

Three clinical conditions any result from exposure to high environmental temperature: (1) heat cramps, (2) heat hyperpyrexia, and (3) heat prostration.

Heat Cramps: (miner's cramps, stoker's cramps, or fireman's cramps): They are caused by a rapid dehydration of body through the loss of water and salt in the sweat. It is seen in workers in high temperature when sweating has been profuse,. The onset is usually sudden. Severe and painful paroxysmal cramps affecting the muscles of the arms, legs and abdomen occur. The face is flushed, the pupils dilated and the patient complains of dizziness, tinnitus, headache and vomiting. Intravenous injection of saline gives rapid relief.

Heat Hyperpyrexia or Heat Stroke: Heat stroke is a condition characterized by rectal temperature greater than 41°C; and neurological disturbances, sucha s psychosis, delirium, stupor, coma, and convulsions. The term thermic fever or "sunstroke" is used when there has been direct exposure to the sun. High temperature, increased humidity, minor infections, muscular activity, and lack of acclimatization are the principal factors in the initiation. Where there is 100% humidity, a temperature of 32°C in the environment may lead to heat stroke. Other factors are old age, pre-existing disease, alcoholism, use of major tranquilizers, obesity, lack of air movement and unsuitable clothing. Failure of cutaneous blood flow and sweating, the factors which control body temperature, leads to a breakdown of the heat regulating centre of the hypothalamus.

Clinical Features: The onset is usually sudden, with sudden collapse and loss of consciousness. In some cases, prodromal symptoms of headache, dizziness, nausea, vomiting, weakness, mental confusion, muscle cramps, restlessness and excessive thirst occur. The temperature rises to 40°C

to 43ºC or more. The skin is dry, hot and flushed, with complete absence of sweating. The pupils are contracted,. The pulse is rapid (usually more than 130 p.m.) and later becomes irregular. The breathing is rapid, (usually above 30 breaths p.m.) deep and of Kussmaul type. Blood pressure is low. Convulsions occur and the patient becomes delirious or comatose. The fatal period is 5 minutes to 3 days.

Post-mortem Appearances: They are not specific. The temperature remains high after death. C.N.S: The brain is congested and edematous and petechial hemorrhages are seen in the white matter . cerebral hemispheres are increased in weight and show flattening of the convolutions. Cellular changes with pyknotic nuclei, swollen dendrites, chromatolytic changes, degeneration of neurons and diffuse proliferation of mocroglia are seen. Changes occur in cerebellum rapidly which are more striking and consistent and consist of oedema of the Purkinje layer and swelling, disintegration and reduction of the Purkinje cells. If the person survives for 24 hours, complete degeneration of the Purkinje layer and gliosis are seen. Rarefaction of the granular layer occurs with prolonged survival. Hypothalamus shows oedema of the nuclei. Respiratory system: Trachea and bronchi contain frothy hemorrhagic fluid. The lungs show oedema, congestion and hemorrhages. Heart: Dilation of right auricle, flabbiness of muscle, petechial or confluent subepicardial and subendocardial hemorrhages and degeneration of myocardium. Liver: Congestion and centrilobular necrosis. Kidneys: Congestion, oedema and increase in weight. In case of longer survival, haemoglobinuric nephrosis is common. Adrenals: Pericapsular hemorrhages, engorgement of sinusoids and cortical degeneration. General: Petechial and confluent haemorrhages are seen in most organs.

Heat Prostration (heat exhaustion; heat syncope, or heat collapse): Heat prostration is a condition of collapse without increase in body temperature, which follows exposure to excessive heat. It is precipitated by muscular work and unsuitable clothing. There is extreme exhaustion and peripheral vascular collapse. The patient feels suddenly weak, giddy and sick. He may stagger or fall. The face is pale, the skin cold, the temperature subnormal. The pupils are dilated, the pulse small and thready and the respiration sighing. The patient usually recovers if placed at rest, but death may take place from heart failure.

SCALDS

A scald is an injury which results from the application of liquid about 60ºC or from steam. The destruction does not extend as deeply as in burns. Redness appears at once and blistering will take place within a few minutes. If blistered skin is removed, it will leave a pink raw surface and later the exposed dermis becomes brownish, hard and dry. Scalds show saddening and bleaching but do not singe the hair, and do not blacken or char the skin. Superheated steam soddens the skin which becomes dirty white colour.

Degrees of Scalds: (1) Erythema by vasoparalysis, (2) Blister formation due to increased permeability of the capillaries, (3) Necrosis of the dermis.

The injury is limited to the area of contact and is more severe at the point of the initial contact.

Scalding can occur through clothing. Scalded areas are usually large, but may be small if caused by splashing. Streaks of liquid run downwards from the main area causing lines of blisters. Sticky liquids. Such as syrup, oils and tar cause more severe scalds than hot water. There is usually a sharply demarcated edge, corresponding to the limits of contact of the fluid. The scalded skin may swell and exude serum. Scars of scalds are much thinner than those of deep burns and cause less contraction and disfigurement. Blisters have an hyperaemic zone around them. There is a reddening and swelling of the papillae in the floor of the blister. The blister fluid contains white and red cells. A post-mortem blister does not show hyperaemia in the surrounding area and the floor is not red. If inflammable fluids are used, signs of trickling of the burning fluid will be present on some parts of the body, e.g. if kerosene is splashed on a body lying on its back and then ignited, runs of burning liquid will be seen on the sides of the neck, sides of the trunk, between the thighs, etc. Inhalation of steam may cause thermal injury of the respiratory tract, producing death by asphyxia due to obstruction to airway by the oedematous mucous membrane. Death usually occurs from shock, fluid and electrolyte disturbance and secondary infection.

Occurrence: Scalds are usually accidental due to bursting of hot water bottles, bursting of boilers, splashing of fluid from cooking utensils, or pulling over saucepans or kettles by children, etc. Occasionally, children suck the spouts of kettles, which causes severe steam scalds of the mouth and air-passages with oedema of the glottis. Suicide by scalding is rare. Boiling water ma be thrown with intent to injure. Murder scalding is rare.

Spontaneous combustion: Spontaneous combustion of the human body not occur. A body can never be consumed without the application of fire or flame and it cannot be reduced to ashes without the surrounding objects being set on fire.

Preternatural Combustion: This is very rare. During putrefaction, inflammable gases are produced in abdomen due to the action of microorganisms upon organic matter. These gases are ignited if a flame is nearby, and cause partial burning of the neighbouring soft tissues, but complete combustion of the body does not take place. It is not a valid scientific phenomenon.

During life, inflammable gases, such as hydrogen, hydrogen sulphide and methane may be formed in the alimentary tract. Such gases when belched or let off from the anus, may be ignited on the application of a flame and cause a burn at the site.

The injuries caused by contact with electrical conductors depends upon: (1) The kind of current: Alternating current is 4 to 5 times as dangerous as an equal voltage of direct current. (2) The amount of current: The amount of current that will flow through or over the body may be determined by the formula CV/R. where C is the current in volts and R is the resistance of the body in ohms. The flow of the current through the body is great, if the voltage is high or if the resistance is low. Electrocution is rare at less than 100 volts, and most deaths occur at more than 200 volts. Currents of 10mA cause pain and muscle contractions, over 60mA are dangerous, and 100 mA is fatal. High voltages may cause the victim to be thrown clear, while lower tensions, around 240, cause muscle contraction, due to which the victim holds on to the source of the current. (3) The path of the current: Death is more likely to occur if the brain stem or heart are in the direct path of the current. (4) Duration of the current flow: The severity is directly proportional to the duration of current flow. For an electric shock to occur there must be contact by the body with both a

positive and negative pole, or alternatively, the 'earth'. The earth may be any object not insulated from the ground. When earthing of the body is poor, as with dry and rubber shoes, carpets, wooden floors and upstairs premises, fatal electrocution is uncommon. The effects of electricity depends on the voltage and the resistance offered by the body. Of the body is well insulated, if does not conduct the current and no harm results. Dry skin offers high resistance but the resistance is diminished when the skin is moist or covered with sweat. Blood has a low resistance, and as such within the body, electricity tends to be conducted along blood vessels. Predisposing factors are unexpectedness of shock, anxiety, fear and emotions, exhaustion, cardiovascular and other diseases.

Local Effects:

The current passes through the skin producing heat, which causes boiling and electrolysis of tissue fluids. The skin explodes and rolls back from the surface. A well-moistened skin may not show electrical burn, while a thick dry skin may show well-marked electrical burn.

The electric mark (Joule burn): It is specific and diagnostic of contact with electricity and is found at the point of entry of the current. These marks are round or oval, shallow craters, one to 3 cm. in diameter, and have a ridge of skin of about one to 3mm. high, around part or the whole of their circumference. The crater floor is lined by pale flattened skin. In some marks, the skin may break within or near the margin of the crater, resembling that of a broken blister. The skin of the mark is pale, but there may be mild hyperaemia of the adjacent intact skin, due to rapid dilation of pre-capillary vessels. When contact is more prolonged, the skin in the mark becomes brown and with further contact, there may be charring. Occasionally, the mark may have a distinctive pattern, that of the shape of the conductor. Rarely, the mark may be present as a circular hole, penetrating skin, muscle, and even bone, so as to simulate a bullet wound. These electric marks are produced by the conversion of electricity into heat within the tissues. They are commonly found on exposed parts of the body, especially on the palmar aspect of the hands.

Flash or Spark Burns: The intense heat which may result from flash-over produces burns, which resemble thermal burns. The burns may be as small as pinpoints, or deeply seated and contracted if contact is prolonged or very high voltage is applied. If the area of contact is relatively large, e.g. when a hot wire is grasped with a wet hand, or when a person is electrocuted in bath tub, death may occur without any visible skin burning. High voltage burns may be very severe with charring of the body. Multiple individual and confluent areas of third degree burns are seen. Very high voltage currents produce massive destruction of tissue with loss of extremities and rupture of organs. When bone is involved, periosteum may be elevated or superficial layers of the bone may be destroyed or fracture may occur. Sometimes, multiple lesions are found in the region of flexures of a limb where the current has passed across the joints, instead of passing around it. high tension electrical currents may produce multiple discrete lesions due to arcing from the conductor to the body without direct contact. Multiple burnt or punched-out lesions are produced due to the are dancing over the body surface over large areas which present 'crocodile flash burns'. Flash-over often produce 'arc eye'. There can be blast effect from very high voltage discharges.

Electric burns or splits: The splits are dry, hard, firm, charred, insensitive, with ragged edges, and their form is round, oval, linear, or of irregular shape. The depth of the lesion is much greater than appears on the surface. Shedding of the superficial layers of the skin is common, and some of this may be found attached to the conductor. Wrinkling of the skin may be found and occasionally localized oedema of a limb. Aseptic necrosis develops, which often extends beyond the burns in area and depth and may lead to sloughing.

Microblisters develop within the squamous epithelium and in the external horny layer, due to the cooking effect on the tissues. They represent defects through which the steam exited. Larger vacuoles are produced within the epidermal cells, the nuclei. These flattened cells usually stain darker than the normal cells with haemotoxylin and eosin. The nuclei of the vascular media tend to be twisted to resemble spirals, which may be seen at distant points from the site of contact with the electrode. These may be localized degeneration of the intima. Tearing of elastic fibres and the overlying intima is common and may cause secondary thrombosis.

Exit Marks: These are variable in appearance, but they have some of the features of entrance marks. There may be more damage of tissues, and they are often seen as splits in the skin at points where the skin has been raised into ridges by the passage of current; splitting of these ridges may be continuous or interrupted.

Post-mortem Appearances:

External: The examination of the scene may be much more important than the post-mortem of the body. The face is pale, the eyes are congested and the pupils dilated. Rigor mortis appears early, and post-mortem lividity is well developed. Usually there are external marks of electric burning, and contusion or laceration at the point of entrance and exit of the body. In some cases, the lesions may extend through subcutaneous tissues and involve muscles and bone. A number of grayish-white circular spots, which are firm to the touch and free from zone of inflammation may be found at the site of entrance and exit. Severe convulsions caused by electrical discharge may cause fractures of the spine or limbs. Extensive ecchymosed may be found on the skin of the trunk. In some cases, external lesions may be absent and frequently they are so slight as to require careful search. The clothing, including shoes, gloves and headgear should be examined for burns. Occasionally, only the hair is singed. Arcing of the current may produce characteristic pit-like defects on the surface of the hair. Any metallic objects on the body will produce corresponding burns on the skin because it becomes heated by the passage of the current. The color varies depending on the composition of the conductor, i.e., brown or black if of iron, or yellow-brown if of copper. This metallization is due to the volatilization of the metal, particles of which are driven into the skin. Metallization produced by low or medium voltage may be detected under low magnification or by histological or chemical examination. Current marks may be hidden inside the oral cavity, from putting live wires into the mouth or from drinking at a water fountain in contact with electric current. They may be found in the urethra due to urination on a high tension wire. In some cases, the entrance and exit marks cannot be determined grossly. Occasionally, the site of entrance may be determined by histochemical methods or by electron microscopy from the deposition of metal particles on the skin.

Internal: The appearances are usually those of asphyxia. The lungs are congested and edematous, and the brain, meninges and parenchymatous organs are congested. Petechial hemorrhages may be found along the line of the passage of the current, under the endocardium, pericardium, pleura, brain and the spinal cord. There may be necrosis of the intima, or of the complete wall of the blood vessels. Vascular thromboses may be seen in the vicinity of electrical burns. Skeletal muscle in the path of the current may show Zenker's degeneration, often with spiraling and fragmentation of fibres. High amperage has an explosive effect and may produce injuries resembling bullet, stab or cut wounds. Small balls of molten metal, derived from the metal of the contacting electrode, so-called current pearls, may be carried deep into the tissues. Heat generated by the current may melt the calcium phosphate, which is seen radiologically as typical round density foci ('bone pearls' pr 'wax drippings'). There may be bone necrosis, and zigzag micro fractures. Focal petechial

haemorrhages are found in the brain and spinal cord, especially in the medulla and the grey of the pyramidal nuclei and of the anterior horns and the Purkinje cells of the cerebellum; wide dilatations of the perivascular spaces especially in the brain stem and cervical cord; fragmentation of the axons, and changes in the myelin sheaths of peripheral nerves are noted. In some cases, irregular tears and fissures in the brain tissue and rupture of walls of arteries are seen. A foetus may survive the electrocuted mother or a surviving mother may abort after electric injury. Occasionally, no lesions can be found either externally or internally. Death in these cases may be due to vagal stimulation.

Cause of Death:

Circuits from any of the limbs to the head involve the brain stem and upper cervical cord. Arm-to-arm circuit may also involve the upper cervical cord. In these cases, death probably occurs from paralysis of medullary (respiratory) centers. Arm-to-arm or left arm to either leg circuits involve the heart and death occurs either from ventricular fibrillation or cardiac arrest without fibrillation. Death need not be instantaneous. Individuals may be able to walk some distance and talk before the onset of collapse and death.

Medico-legal Aspects:

Death by electric currents are usually accidental from defective electric appliances or negligence in the use of equipment. In industry, deaths may result from contact with live overhead cables or from handling of charged lamps, tools or switchgears. Rarely, death may occur during convulsive therapy to mental patients but cases of suicide, and even homicide have occurred. The viscera should be analyzed to know whether the victim was impaired at the time of the accident. Suicide is rare. A person usually winds wires round his fingers or wrists, which are then connected to the mains supply by means of a plug and the current is switched on.

Judicial Electrocution: Death penalty is carried out in the electric chair in some states is the U.S.A. The condemned man is strapped to a wooden chair and one cap-like electrode is put on the shaven scalp which is moistened with a conducting paste and the other on the right lower leg, and a current of 2,000 volts and 7 amperes I passed for one minute through the body. After titanic spasm and loss of consciousness, the same current is passed through the body a second time for one minute.

LIGHTNING STROKE

A flash or bolt of lightning is due to an electrical discharge from a cloud to the earth. The electric current is direct with a potential of 1,000 million volts or more. Along the track f the current much energy is liberated, most of which is converted into light. It is attracted b the highest points. It passes normally along the outside of a conductor, and as such, persons in buildings are relatively safe from electrocution. Dry skin and dry clothes are bad conductors, whereas wet skin and wet clothes are good conductors. Lightning or atmospheric electricity differs from ordinary electric current only in degree. A lightning bolt may injure or kill a person by a direct strike, a side-flash, or conduction through another object. In a direct strike or a side-flash strike, the current can spread over the surface of the body, enter it or follow both routes. In a side-flash strike, the flash of lightning hits an object, e.g. a tree, and jumping from it, strikes the person. In conduction through another object, the lightning hits a metallic object, flows through it, and strikes a grounded person touching it.

Symptoms: Unconsciousness is immediate. In non-fatal cases the person complains of giddiness, ringing in the ears and headache. In severe shock, the individual may suffer from hemorrhages or

detachment, and later from loss of memory, anesthesia, paralysis, titanic convulsions, delirium, blindness, deafness or dumbness.

Post-mortem Appearances: Four factors are involved: (1) direct effect from electric discharge passing to earth, (2) surface 'flash' burns from the discharge, (3) mechanical effect due to force of displaced air around the flash by heat expansion, and (4) compression effect due to air movement in its return wave. The clothes are usually burnt or torn at the point of entrance and exit. In some cases, the clothes may be stripped off the body and thrown to some distance. In exceptional cases, clothing is not damaged even though the person is killed by lightning. Conversely, clothing may be burnt without any injury to the person. The expanded, displaced air causes disruptive or blast-like lesions, e.g., contusions, lacerations, fractures, ruptures of organs, wounds of almost any variety, burns, etc. Rigor mortis may appear soon and pass off quickly. Intense edema of the skin develops at points of entry of current in those who survive, probably due to paralysis of local capillary and lymphatic vessels.

The burns may be:

Linear: These vary from 3 to 30 cm. or more in length, and 0.3 to 2.5cm. in width. They are often found in the moist creases and folds of the skin.

Arbores cent or Filigree Burns: (Lichtenberg's flowers) : They are superficial, thin, irregular and tortuous markings on the skin. These markings have a general pattern resembling the branches of a tree. This fern-like pattern of erythema in the skin is usually found over the shoulders or the flanks. These marks are seen rarely.

Surface Burns: They are true burns and occur beneath metallic objects worn or carried by the person, which are fused by the flash. Internal findings are those of asphyxia.

Cause of Death:

Involvement of the central nervous system with paralysis of the heart or of the respiratory centre causes death.

Medico-legal Importance:

Less than half of the persons struck by lightning are killed. Death is always due to accident. Sometimes, the appearances left on the human body closely resemble those produced by criminal violence. Thus a person may be found dead in an open field or on the highway and body may show contusions, lacerations and fractures. In such cases, the diagnosis should be based on the history of a thunderstorm in the locality, evidence of effects of lightning in the vicinity of the body, and fusion or magnetization of metallic substances.

Electrocution

Most deaths from electrocution are accidental. In home, accidents results from contact with line electrical supply lines; the majority of the accidents being caused by defective equipment or negligence in the use of equipment. In industry, fatalities occasionally result from contact with line overhead cables or from handling of charged lamps, tools or switch gears.

Most cases pore nor investigative problems. Even if the death is not witnessed, examination of place for occurrence will reveal evidence indicating the possibility of electrocution. In white wasting or repairing walls the distance of wire is very important.

Lightning

At the scene of death there may be evidence of disruption of the ground, building and other objects caused by lighting.

The clothing of the victim may show extensive tears with scorches or burns. The shoe and tight clothing may be burst open. The body may, likewise, show extensive tears and splits in the skin, opening of body cavities and fractures of the bones. The metallic objects on the body may show melting.

REGIONAL INJURIES

HEAD INJURIES

The head constitutes of contrasting substance and textures arranged in concentric planes like Soft tissues,Bones,Air,and Fluid.Thier Anatomical relations and their own consistency determines their reaction to the application of force. Head injuries are generally caused due to application of Blunt Force but they may also be caused by Sharp Edged or Sharp pointed weapons. Open Head Injury are due to impact by Sharp objects and Missiles. Closed head injury are due to application of Blunt Force. Blunt force impact to the Head may result in injury to the contents of the skull, either alone or in combination with fracture of the skull,it is exceptional for the skull to be fractured without some intracranial injury. Injury to the contents of the skull may affect the brain or Meninges and their related vessels. In head injury the extent of injury to the skull and its contents may not be proportional to the amount of force applied to the head. The application of Moderate force to the head may cause severe intracranial hemorrhages while greater force may produce no injury.The skull or its contents may be injured without any external evidence of the injury.

SCALP:

Wounds of scalp may or may not be associated with fracture of the skull or intracranial contents of skull. Most wounds are caused by application of blunt force to the head, e.g., from falls or blows, and such wounds are contusions or lacerations. Such wounds of scalp take the form of Contusions and Lacerations. Bleeding from scalp wounds may result in considerable Flow due to sharp edged weapons.

Contusion and Laceration of the Scalp:

Contusion: Contusion of the scalp often result from the crushing of the soft tissues against the underlying bone. The scalp contusions may also appear due to fracture of Skull wherin blood will extravasate from ruptured diploic veins. 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 localized 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. In addition to frank bleeding after injury, marked oedema may occur, and the layers of scalp may be greatly swollen and thickened by a jelly-like infiltration of tissue fluid. 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. Bruising of the scalp is better felt than seen. Multiple contusions of the scalp may fuse 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. They bleed profusely and a fatal blood loss can occur from an extensive 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.

Laceration:

Lacerations of scalp may occur from splitting of the soft tissues aganst the underlying bone or from the tearing of the tissues by fragments of fractured bone. Lacerated wounds of the scalp are often linear in shape and may resemble incised wounds. Laceration involving galea aponeurotica,if infected may spread widely in the subaponeurotic space.The veins of the scalp and the face are connected with the parasigittal,lateral and cavernous sinuses through emissary veins which pass through foramina in the skull **or through an unnoticed fissured fracture.**.Infected wounds of the scalp and the face may be complicated by thrombophlebitis of scalp and facial veins and this process may extend through the emissary veins to the intracranial sinuses.

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 hemorrhage or detachment or rupture of the retina and traumatic cataract. The eyes may be gouged out with the fingers.

Black eye: It is caused by

1) direct blow in the front of the orbit, bruising lides.
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.

LIPS: A blow with a fist or blunt weapon produces injuries.

Facial bones: A blow with a blunt weapon or first often fractures the nasal bones, and also ethmoid bone 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 first, 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 10mm. the temporal bone is thinnest, 4mm.

Mechanism of fracture of skull:

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 well 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 injuries of the skull occurs from a fall on the feet or buttocks.

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 sufficient to fracture empty human skulls lacking normal soft tissue coverings. The kind of fracture which follows violence depends on the weight and velocity of the agent, the amount of force, the point of impact, the thickness of the bone and the mobility of the skull at the time of the blow. 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 a fractured skull. A thin skull with a fracture may produce less brain damage than thick skull without it.

1) **Fractures due to local deformation**: If a small mass traveling 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:
    a. Between two external objects, such as the ground and a wheel of a car.
    b. 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 condyless of the atlas.

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

TYPES OF SKULL FRACTURES:

Fissured fractures: These are linear fractures or cracks in the bone involving the whole thickness of the bone or one or other table only. They are 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. 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 ma start at the counter pressure, e.g. in bilateral compression the fracture often starts at the vertex or more commonly at the base. The fracture line tends to follow a devious course and is usually no more than hair's breadth.

Depressed fractures: In this portions of fractured bone are driven inwards into the skull cavity. Their pattern often resembles the weapon or agent which caused it. They are caused by blows from heavy weapons with a small striking surface, e.g., stones, sticks, axe, hammer, etc.

Comminated fractures: In this the bone is broken into several pieces. They are caused by fall from a height, vehicle accidents and from blows by weapons with a large striking surface, e.g. heavy iron bar, axe, thick stick, etc. Fissured fractures may radiate for varying distances from the area of comminution.

Pond or Indented fractures: This is a simple inbuckling of the skull, which results from the obstetric forceps blade, a blow from a blunt object or forcible impact against some protruding object.

Gutter fractures: They are caused when part of the thickness of the bone is removed so as to from a gutter, e.g. glancing bullet wounds. They are usually accompanied by irregular depressed fractures to the inner table of the skull.

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 thirds. 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. They are rare and occur after falls from a height into the feet or buttocks. This drives the vertebral column into the skull.

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.

Diastic or sutural fractures: separation of the sutures occur only in young person due to a blow on head with blunt instrument.

Fracture of the skull occurring opposite to the site of force is known as conrte-coup fracture. This usually occurs when the head is not supported.

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. Fracute 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. Fractures lines usually open into the basal foramina. The sphenoidal fissure is most commonly affected. The foramen magnum is not spared in spite 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 maybe 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 orbit 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) Longitudianl, 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.

Fractures of anterior cranial fossa may involve the frontal, ethoidal or sphenoidal air sinuses, with loss of blood from nose and mouth. If the dura and nasal mucosa are torn, CSF and even brain tissue can leak in to the nose. Leptomeningitis may result due to bacteria passing upwards from the nose. Fractures involving paranasal sinuses may cause cranial pneumatocele. Air collects beneath the pericranium of the outer table and may be limited or spreads diffusely. Extradural, subdural or subarachnoid pneumatocele are quite rare. Fractures of the middle fossa passing through the basiocciput or sphenoid bone may communicate with mouth, from which blood will run. 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. 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 te posterior branch of the middle meningeal artery as it crosses a fracture of the temporal bone produces severe extravasation of blood is seen behind the mastoid process or a large haematoma in the soft tissues of the back of the neck. If the fracture reaches foramen magnum, cerebellar contusions may result, and the subsequent oedema may herniated the cerebellar tonsils fatally through the foramen. Cranial nerves may be injured by stretching or bruising but they are usually not severed.

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 is 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, localized 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.

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 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.

Complications:

1) Haemorrhage.
2) Pain and dysfunction.
3) Damage to surrounding structures.
4) Shock.
5) Portal of entry for bacteria.
6) Fat and bone marrow embolism.
7) Depressed fractures of the skull pressing the brain may cause severe dysfunction, coma and death.

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. Contusions are produced in the brain which can be a] contusion haemorrhages, b] contusion necroses, c] contusion tears.

MECHANISM OF CEREBRAL INJURY

The brain is damaged By:

1) Penetration by a foreign object, such as knife, bullet, etc., or fragments of skull in a depressed fracture.
2) Deformation of the brain in closed head injury.

BLUNT FORCE IMPACTS TO THE HEAD

1) **Minor impacts:** A minor blow, e.g., by the first to the resisting but moveable head produces a momentary acceleration of the head, without deforming the skull. As the head is eccentrically affixed to the spinal column and the neck, the acceleration is rotational. The

peak of acceleration is reached shortly after the head is set in motion, but the brain is inert to such fast acceleration. Due to this, a positive pressure is produced between the brain and skull at the site of the impact and a negative pressure over the opposite surface of the brain. This is indicated in figure [141] by the plus and minus symbols. The arrow with A indicates that the brain resists the pressure by the skull, which is moving in the direction of the other arrow. These are called acceleration pressures. The brain continues to moves, even after the head returns to its normal position, which reverses the pressure, and rapidly subsiding rotational movements of the brain relative to the skull follows. The initial acceleration pressures are the greater. This causes subdural and subarachnoid haemorrhage and less commonly, gliding contusions in the upper marginal portions of the cerebrum.

In a minor fall on the head, sudden deceleration of the skull occurs and the brain continues to fall because of its momentum. At the site of impact, the brain is suddenly pressed against the skull. A negative pressure is produced on the opposite surface of the brain. These forces subside rapidly. If the head bounces, these forces may be reversed. They may produce subdural or subarachnoid haemorrhage.

2) **Major impacts:** The depression or flattening of the area of impact occurring due to a major blow acts as space occupying lesion and produces positive intracranial is greatest at the site of impact. As the head is accelerated at the moment of this deformation, two positive pressures act on the brain tissue underlying depressed area. this is shown in figure [142] by two plus signs, the black plus signifies deformation pressure. The pressure associated with the shifting of the brain [black arrow] is counteracted by the accelerating pressure [white arrow] which moves the brain to the opposite side. Because of this, the pressure associated with the shifting of the brain may not reach the opposite surface of the brain may not reach opposite surface of the brain of if it reaches, it may be neutralized there, by the existing negative acceleration the deformation pressure is not severe to produce contrecoup contusions, the brain may shift towards the foramen magnum and herniated the cerebellar tonsils which produce tonsilar herniation contusions.

In a severe fall the head is accelerated before the impact and deformation of the skull. If a person falls on the back of the head, the inertia of the brain produces a positive acceleration pressure over the fronto-orbital region and negative acceleration pressure over the

cerebellar regions. These pressures last until the deformation of the skull at the site of impact reaches its maximum. Due to the deformation, the brain is displaced towards the opposite side and two positive forces act on the fronto-orbital cortex. The positive force of deformation is decreased at the site of impact, by the still existing negative acceleration pressure. In falls on the head, the contusions are explained by this mechanism. When the rate of acceleratio of the head prior to impact is great, the contrecoup damage is more severe. The degree of acceleration does not depend on the height of the fall. It may be greater and cause more damage in a fall from sitting position than form standing position. The brain and skull accelerate at the same rate in a fall from a high building, and as such, acceleration pressures do not develop within the skull before the impact. In such cases there may be total fracture of the skull, and coup and contrecoup lesions may be minimal. Severe contusions are usually found when there is no fracture. It the shearing force within the tissue exceeds the cohesive strength of the vessel, contusion haemorrhages occur. A much greater force will produce contusion necrosis.

Contusions and lacerations of the brain: Contusion and laceration of the brain are two degrees of the same process. When a localized 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. 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.

Contusion haemorrhages: 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, accompanied by variable amounts of necrosis. Most often they occur in cerebral and cerbellar cortices and subcortical area. 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 previascular spaces along the shriveled and collapsed blood vessels. It spreads along the sulcus and often involves severeal 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 heir union especially in persons with hypertension or in alcoholics. Many contusions are only seen on section of brain.

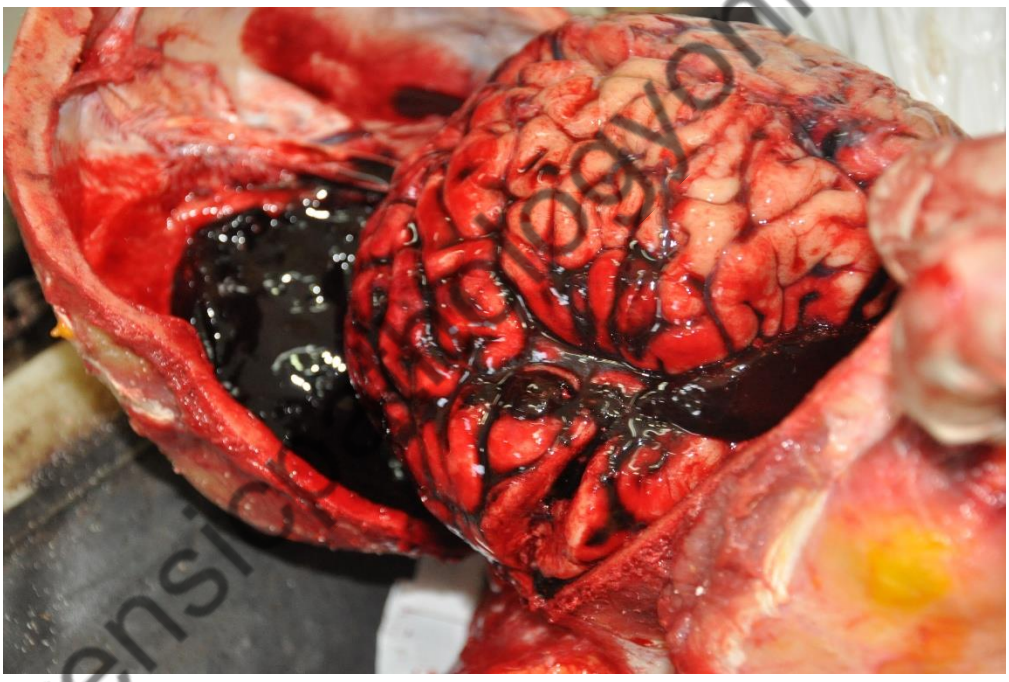
Blows to the back of head, usually due to falling backwards result in little or no occipital contusion, although there may be posterior cerebellar contusion or subarachnoid haemorrhage. They usually produce subfrontal and temporal lobe contusion. Blows to the top of the head produce minimal coup contusion but prominent contrecoup subcerebellar contusion or laceration, which even result in posterior fossa subdural haemorrhage. In serious impacts, corpus callosum is lacerated. Blows tot e 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 tot the front of the head usually do not produce cerebral 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 beings 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.

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 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.

Contusion tears: Cerebral lacerations are traumatic lesions in which

there is loss of continuity [tearing] of the substance of brain. When the parenchyma is completely disorganized, it is termed pulpefaction. These tears are caused by stretching and shearing forces within the tissues produced by blunt force. In infants up to fie 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. These slit-like, sometimes irregularly shaped lacerations often contain very little blood and may be mistaken for artifacts.



Secondary alterations: They are the lesions which develop after injury.

Swelling of the brain tissues: Brain swelling can be either due to an increase intravascular blood volume secondary to vasodilation or an absolute increase in the water content of the brain tissue [cerebral oedema]. If continued long enough, brain swelling progresses to cerebral oedema. A] Localised swelling surrounding brain damage results from reduced perfusion with blood. B] Generalized swelling may involve both cerebral hemispheres and also the brain stem and cerebellum. This is

usually known as post-traumatic brain swelling, and may develop very rapidly. The amount of fluid in the brain increases, and the total weight may increase by at least 100 g. it may cause death in 20 to 30 minutes due to pressure on the midbrain and the medulla oblongata, even though the trauma has not caused any intracranial lesions or a fracture of the skull. The irritation of the medulla by subarachnoid bleeding may lead to a rapid cariorespiratory failure. The swelling is caused by the acute shock which produces acidosis and swelling of astrocytes and oligodendroglia. It begins in the deeper white matter of the capillaries and small blood vessels in the brain and decreases the flow of the blood through the white matter which becomes pale. Any type of shock will produce brain swelling. Space occupying primary lesions of the brain, e.g., extradural or subdural haematomas, meningioma, etc., usually promote swelling. In diffuse brain swelling, there is usually symmetrical herniation of the cerebrallar tonsils through the foramen magnum. In asymmetrical herniation due to subdural haematoma, there is cerebellar tonsil herniation on the same side and a secondary brain stem haemorrhage [Duret haemorrhage] in the midbrain and pons. A rapidly expanding supratentorial mass causes transtentorial or uncal herniation.

Thrombosis of the superior longitudinal sinus may occur due to trauma, and this will produce cerebral oedema. Thrombosis of great vein of Galen also produces cerebral oedema. This may occur in any unconscious person due to pressure on one side of the neck by some object which diminishes blood flow to the brain via the internal carotid artery. Thrombosis of the internal carotid artery by severe arteriosclerosis or from a tear in the artery due to fractue of the petrous part of the temporal lobe cause swelling of one cerebral hemisphere. If the transverse sinus is obstructed, swelling of one temporal lobe is seen. At autopsy, the dura is stretched and tense, and the brain bulges, through the incision in the dura. The gyri are pale and flattened, and sulci are obliterated. The cut surface is pale, and the ventricles may be reduced to slits due to the swelling of adjacent white matter. In severe cerebral oedema, the hemispheres are pressed down upon the tentorium and herniated through the midbrain opening. The hippocampal gyrus may impact in the opening, lesser degrees causing grooving of the uncus. At the site of pressure, haemorrhage and necrosis occurs. The tonsils of the cerebellum may be impacted or ‘coned’ into the foramen magnum, and show discolouration or ischaemic necrosis of the trapped tissue.

Haemorrhage and necrosis: Compression f blood vessel during a period of low blood pressure or transient cardiac arrest produces

haemorrhage and necrosis. The cortex of the cingulated and hippocampal gyri show small lesions, where they are pressed aganst the margins of falx and tentorium respectively. They occur after hemiation of these gyri. Large lesions due to vascular compression are found in the lower half of the midbrain, which extends into the tegmentum of the upper pons. They develop from pressure on the midbrain by the hippocampal gyri. This is usually seen in the trauma due to the pressure of extradural or subdural haematomas. The interpeduncular fossa of the midbrain is narrowed. The blood supply of the midbrain is diminished when the mammillary bodies shift into the fossa. Oedema develps and often haemorrhages occur which sometimes involve entire tegmentum of the midbrain and produce unconsciousness. When there is gradual development of supratentorial pressure on the midbrain, the respiration is regulated by the medulla oblongata. In such case, the patient may survive for months or years, though unconscious.

The cerebral cortex may be injured in exploration of subdural haematoma. If the brain is swollen, the cortex may herniated into the burr-hole and may become haemorrhagic and necrotic due to obstruction to its blood supply. The necrosis may extend into the deeper parts which swell and produce further herniation. This mushroom-like protrusion of necrotic brain tissue is called ‘fungus’.

In falls on the forehead or the convexity of skull, incomplete tearing of the corpus callosum is seen. In extensive fractures of the skull, the midbrain or the lower medulla oblongata may be completely torn. Sudden overextensions of the head as in falls on face, accompanied by a somersault movement of the body in reverse, and in automobile collision produce tearing of the pyramidal motor fibres at the junction of he pons and the medulla oblongata. Local contusions and lacerations of the brain beneath the site of impact may or may not produce immediate unconsciousness, but the contrecoup injuries usually produce immediate loss of consciousness. The healing of surface laceration may cause adhesions between the brain and overlying dura mater.

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 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. There may be no coup damage at all, only contrecoup.

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. Occipital injuries produce severe and extensive contrecoup lesions in the frontal region. The irregular boney 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. 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. 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.

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 along the line of impact are called intermediary coup contusions. Contusions caused by fracture of the skull are called fracture contusions. Contusions in the corex 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 arrachnoid gramulations, during to and fro gliding of the brain within the skull in moderately severe impact. Contusions in the cerebellar tonsils and the medulla oblongata produced by momentary shifting of the brain toward the foramen magnum are called herniation contusions.

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. At low levels of acceleration/deceleration, anatomical damage of the axons does not occur, but there is physiological dysfunction. The axon may recover completely on 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 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 sever DAI, there is coma of more than 24 hours with brain stem signs.

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. The person involved in a traffic accident can walk around and talk to rescuers but may not, or only partially recollect the event. Such person should not be held fully

responsible for what he does or says in this period of post-traumatic amnesia. The police authorities should be advised to delay interrogating an accused or a complainant who has sustained a head injury until he completely recovers from his initial confusion. If unconsciousness is prolonged, it is certainly due to obvious brain injury. Concussion can be ruled out 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 develps later, it is due to other pathology. Blows to the neck or cervicocranial junction produce brain stem concussion. 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. This damage may result from raised intracranial tension, brain stem deformation or shearing strain, but it commonly occurs with acceleration, or deceleration injuries [the head freely moveable at some stage], 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.

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 myelim degeneration, etc., may be found. In mild DAI, some axons may be 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.

Cerbral compression: Any increase in the size of the brain, e.g., generalized 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 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 substaintia 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. 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 sometime 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 sometime after the trauma. A large extradural haemorrhage can 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. Patients who have sustained head injuries should be kept in hospital for observation for at least 24 to 36 hours.

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

i) Stretching of peduncles when the hemispheres shift.
ii) Deceleration against basisphenoid and dorsum sellae.
iii) Lateral shift of peduncle against tentorial margin.
iv) Stretch or avulsion from it of cranial nerves.
v) 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 fouth ventricle. 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 numberous 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.

Psychoneuroses following head injury: Two types of shock may result from head injury: 1] Surgical shock [collapse probably due to widespread vasomotor paralysis of central origin as part of concussion. 2] Emotional shock [nervous or mental shock]. This occurs particularly when the patient does not loose consciousness or becomes temporarily dazed due to a blow, and can recollect some of the circumstances of accident. Emotional shock is seen in a type of accident in which the person has time to realize the dangers before the actual physical injury.

According to the temperament and mental make-up of the injured person, emotional shock may be followed by almost any type of

psychoneuroses. The intensity of the symptoms has no relation to the severity of the head injury. complete recovery may occur from severe injuries, while minor injuries may be followed by severe symptoms. This anxiety state often develops due to apprehension concerning the possible after-effects of the injury, e.g., fear of partial or permanent disablement from work, loss of self-confidence, financial embarrassment, etc. in some cases, the injured person develops an intense fear of being placed in the same situation as that in which the accident had occurred, 'fright reaction', e.g., fear of resuming motor driving in traffic, and in some cases, distress when traveling as a passenger in a motor car, fear of climbing a ladder, etc.

Change of personality: Damage to the thalamus and frontal lobes may produce intense irritability with outbursts of temper and sometimes uncontrollable rage. Mental disorders [psychoses] may be precipitated by head injuries in predisposed individuals.

Epilepsy: Rarely, epilepsy may result from head injury. cerebral contusion and laceration may be associated with acute early epilepsy soon after the head injury. post-traumatic epilepsy appears from a few weeks to 2 years after the injury. at autopsy a depressed fracture pressing on the brain may be seen sometimes, or there may be old adhesions between the membranes and the skull.

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.

1) **Extradural haemorrhage:** The dura is a strong and grey-blusih 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. Extradicranial 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.
    a. A blow over the lateral converxity of the head may 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. Less commonly, the posterior meningeal artery near cribriform plate are injured.

b. A blow over forehead involves the anterior ethmoidal artery.
c. A blow over the occiput, or low behind the ear, may tear the transverse signmoid sinus and produce posterior fossa haematoma.
d. A blow on the vertex may cause haemorrhage from sagittal sinus.
e. Venous extradural haemorrhage accompanies fracture of skull [usually occipital], and is due to bleeding from the diploci 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 3 percent of cases of head injury. these haemorrhages are rare in the first 2 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 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. 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. bleding 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 localized concavity of the external surface of the brain. The clot is oval or circular, about 10 to 20cm. in diameter, 2 to 6cmm. Thick, weighs 30 to 300g. 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 sagital sinus has been injured. Usually, 100ml 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% of cases have a second haemorrhage; subdural, subarachnoid or intracerebral.

Extradural haemorrhage at the base of the skull is rare. Extraduarl haematoma may occasionally spontaneously become smaller due tot 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. As the pressure on the brain increases, the patient first becomes confused and may appear to be drunk. The confusion indicates shifting of the cerebral hemisphere under pressure towards the opposite side producing stress around the third ventricle and midbrain. 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. Later there is bilateral dilation and fixation of the pupils, decerebrate rigidity and death. 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. The usual cause of death is respiratory failure due to compression of the brainstem. 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% cases are fatal. At autopsy, gental removal of blood may show the break in the vessels and fissured fracture of the nearby skull, sometimes confined to inner table.

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 sis commoner than extradural haemorrhage. It is common in childhood or old age. In subacute subdural haemorrhage, coma occurs in a few days, and in chronic haemorrhage, in a few weeks. This occurs in the subdural space between the dura mater and the arachnoid due to:
    a. Rupture of bridging or communicating veins. These veins are commonly ruptured when the brain moves across the face of

the dura. The bridging veins over the upper and posterior aspects of the cerebral hemisphere are under the greatest strains in rotational movements of the head. As such, subdural haemorrhages are usually seen in these regions and tend to spread forwards and downwards due to gravity.

b. Rupture of inferior cerebral veins entering the sinuses at the base of the skull.
c. Rupture of dural venous sinuses.
d. Injury to cortical veins which are torn by sliding motion of the brain after the head has been arrested.
e. Lacerations or contusions of the brain and dura.
f. Laceration of the dura and middle meningeal artery so that the vessel bleeds into the subdural insead of the epidural space.
g. Re-injury of old adhesion between the brain and the dura.
h. Rupture of an aneurysm or a superficial blood vessel malformation in the brain through the arachnoid into the subdural space.
i. Secondary to disease, e.g., cerebral tumour, cerebral aneurysms, or blood disorders.
j. Berry aneurysms may rarely bleed directly into the subdural space or a subarachnoid haematoma can rupture into the subdural space.
k. 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. They may occur after fights or falls, and are especially likely to be found in alcoholics. Old persons and in children. It may occur in the absence of 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 exceeds 50ml. 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 and produce mental and emotional disorders, which in younger persons may be mistaken for schizophrenia, and in older persons for presenile or senile dementia. Usually, the vessels torn are so small that no main bleeding point can be discovered, either at operation or post-mortem.

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 [pachymeningitis haemorrhagica interna chronica] results from slight trauma in which symptoms appear some weeks or months later.

A) Acute Subdural Haemorrhage: It arises mostly from cerebral lacerations. They are often found over the frontal and temporal poles, and in the region of the sphenoidal ridge. 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. 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.

B) **Subacute Type:** 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 localized and deep, or it may occur as a widespread surface film. It presents usually 3 to 6 weeks after the injury. it is a frequent incidental finding at autopsy in old persons.

The haematoma in the subdural space originally consists of fluid blood. The red cells are recognisble up to 48 hours. There may be very early fibroblastic activity at the junction of the clot and the dura, and there is only fibrin on the arachnoid side. After 4 days, red cells begin to lose their shape. The fibroblastic layer on the dural side varies from 2 to 4 cells in thickness, and on the arachnoid side only fibrin is seen. The blood may begin to appear brownish after 5 days, but this is not reliable. At 10 to 12 days, a fragile membrane covers it. this can be lifted off the dura with forceps. Microscopically, it consists of delicate granulation tissue with thin-walled capillaries and actively dividing fibroblasts growing into the clot. Later the membrane becomes a tougher collagenous structure.

The lesion continues to extend after encapsulation due to:

1) Breakdown of original blood in the haematoma which increases the osmotic pressure of the fluid with absorption of CSF.
2) Bleeding from the thin-walled vessels in the organizing wall.
3) Chronic bleeding from the damaged vessels into terior of the clot. After a month or more, the haematoma is transformed into a cyst, containing watery brownish fluid, the chronic subdural haematoma.

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. Subdural haemorrhage is almost always of traumatic origin. There is no lucid interval in subdural and subarachnoid haemorhage. Death is due to secondary ressure upon he brain stem 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.

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.

4) **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 choroids 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 vessels on the surface of cerebral hemisphere. Focal haemorrhages result from force applied to the head, usually accompanied by shaking of the brain and its coverings within the skull.
2) Lacterations 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]. blood accumulates rapidly on the undersurface of the brain. With continued bleeding, blood passes along the fissures into the major cisterns and intot he 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 aneurismal 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 deonstrate the aneurysms, a constant stream of water should be poured over the base of the brain, a forceps or scalpel. Sometimes, the aneurysm is embedded in the brain surface. Intracranial aneurysms cause more than 50% cases of spontaneous subarachnoid haemorrhage.

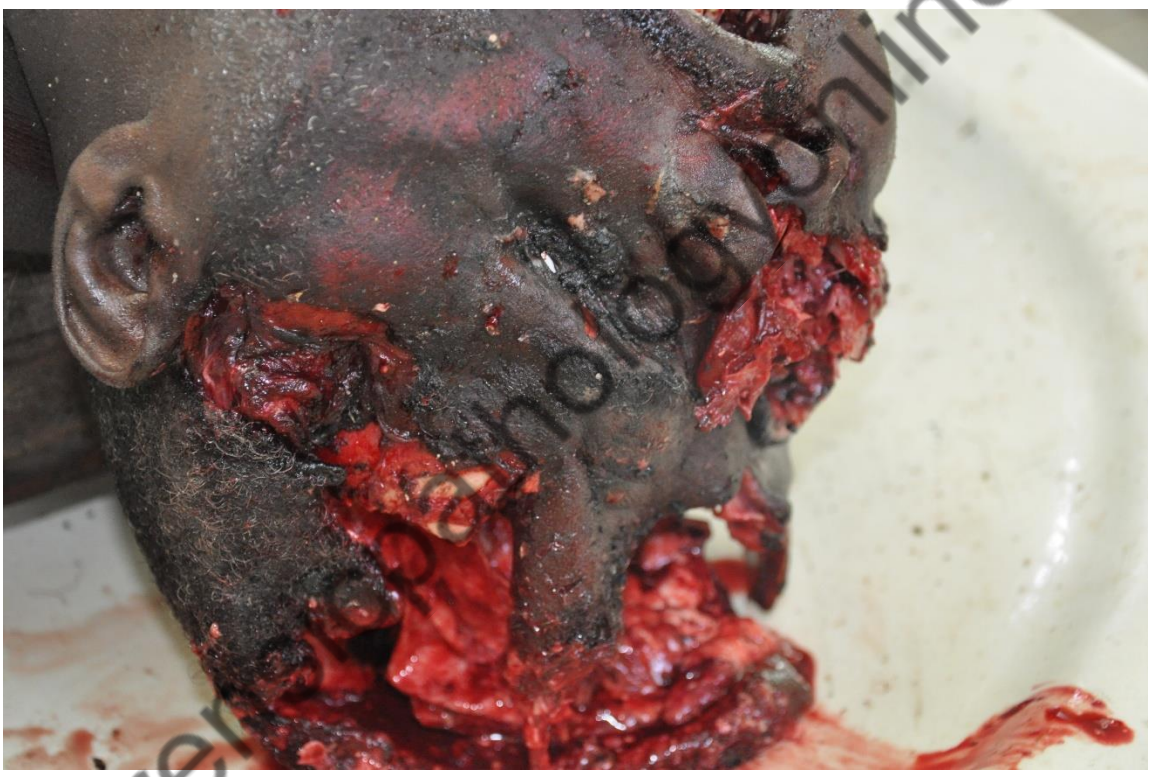
4) Angiomas and arteriovenous malformations cause spontaneous subarachnoid bleeding.
5) Asphyxia.
6) Blood dyscrasias, leukaemias, etc.
7) Tears of the ventricular ependyma.
8) Rupture of an intracerebral haemorrhage of non-traumatic origin [apoplectic haemorrhage or stroke] into the subarachnoid space.
9) Injuries to the side of the upper neck and jaw region, producing damage to the vertebral artery, which causes basal subarachnoid haemorrhage.

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 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.

Alcohol tends to increase all the effects of closed head injury, largely because of increased bone rotation due to lax neck muscles, but also possibly due to increased bleeding from congested vessels which do not contract down, and the bounding pulse of the drunken man.

In the mild form, the subarachnoid haemorrhage is present as splashes of haemorrhage over the areas of contusion. 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 cistern. A slightly yellow discolouration of the leptomeninges is seen as the subarachnoid haemorrhage becomes older.

It can be unilateral or bilateral, localized 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 mixed with CSF 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 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.
7) Intraventricular haemorrhages occasionally occur in the case of puerperal toxaemia in which there have been no fits.

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 pat of the head of the caudate nucleus and pallidum from tears of small branches of the anterior choroids artery. They usually occur due to sever 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 haemorrahge 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 inall kinds of craniocerebral injuries. Rarely, subarachnoid haemorrhage enters the ventricles through the foramen of Magendi. True traumatic ventricular haemorrage as a sole finding occurs due to the head striking a firm object, e.g., a fall or a fall-like injury. the bleeding may be from the choroids 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 ventricule. 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.lg., 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, 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 generalized 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 spontenously 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 chacolate 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.

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 cases 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.

Alcohol and head injuries: Intoxicated persons may stumble and fall and sustain head injuries, the effects of which may be difficult to distinguish from those of alcohol. As such, the examination of an intoxicated person must always include a thorough search for signs of a

head injury. it is wise to admit such persons into the hospital and watch.

Prognosis of head injury:

1) The presence of cortical laceration, both subfrontal and subtemporal is compatible with recovery with minimal neurological deficit.
2) Significant primary midbrain lesions in a person who was in coma, indicates that consciousness would not have been regained.
3) Significant traumatic intracerebral haemorrhage is rarely compatible with recovery even if it is surgically removed.
4) It may not be practicable to remove all the meningeal or intracerebral haemoatomata. In these cases, the associated primary brain damage may be such as to cause death.
5) If the meningeal haemorrhage is significant, the brain stem herniated, secondary haemorrhage present, and no other or minimal damage is present, then death may have been avoided by early surgery.
6) In non-penetrating head injury, injuries to other parts of the body may greatly affect the outcome, e.g., fractures with resultant shock, fat embolism, pulmonary complications, etc. in fact, any injury impairing the oxygenation of the brain is potentially lethal.

Comminution of the skull of 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.

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 tot striking the dashboard.

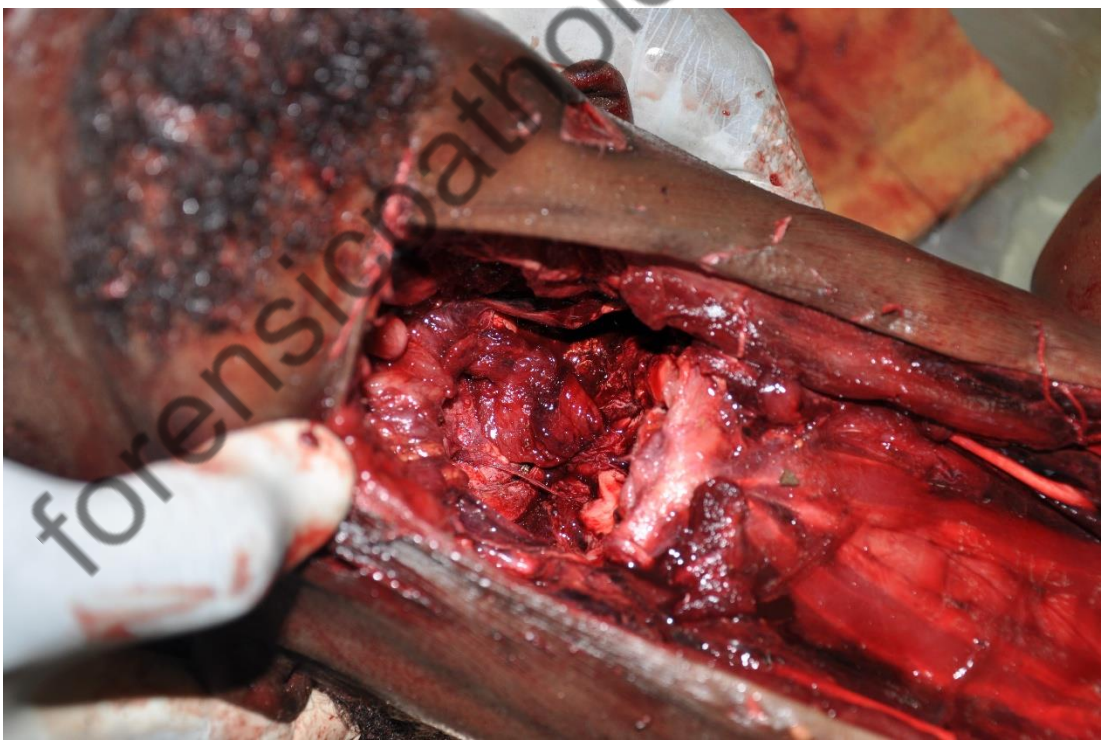
Suicidal incised wounds are more common than homicidal, but punctured wounds are usually homicidal. In wounds of the trachea and of the larynx below the vocal cords, speech is not possible. Wounds of the larynx and trachea are not fatal, if the large blood vessels are not damaged.

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.

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. Hyperflexion 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 one side may also cause strong contraction and fracture 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 is the commonest fracture of the thoracic, thoracolumbar, or lumbar spine. 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 geneal 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 lumber or dorsolumbar juction are rare. Hyperextension causes a comminution of the anterior portion of the body by avulsion of the upper from the lower half through the pull of the anterior longitudinal ligament attached to its rim. 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. He common sites of injury to the cord in order of frequency are: the lower cervical region, the thoracolumber 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. 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: This commonly occurs in railway and motorcar 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 fractureof 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.

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: Indirect violence through compression of the chest produces multiple and often bilateral fractures along the midaxillary line and in the region of the posterior angle of the ribs, and the fragments usually bend outwards. The middle ribs from 4^{th} to 8^{th} 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. Multiple unilateral or bilateral rib fractures give rise to a 'flail' or 'stove-in' chest, with consequent paradoxical respiration and interferes with blood return to the right atrium.

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.

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 paraenchymal 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 sometime 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 perforationof a lung or large bronchus can result in lethal mediastinal shift ot 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 intot he 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 f 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. Thin-walled blebs on the surfaces of the lungs indicate rupture of alveolar sacs from pressure on the chest. Stab wounds of the lungs are usually not fatal, unless a major pulmonary blood vessel has been severed. Pneumothorax is common.

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. They may be seen on the posterior ventricular surface, if the heart is driven against the vertebrae. Recent cardiac contusions are dark-red, haemorrhagic areas which are usually subepicardial. Large ones may be transmural. Recent contusions show interstitial red cell extravasation with separation and disruption of myocardial fibres. The regional vessels are dialted, congested and leucocytes migrate into adjacent tissue. Later, there is necrosis of muscle cells and the haematoma becomes organized and filled by granulation tissue, and finally a collagen scar is laid down. 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 periocardio-phrenic angle. Some injury to the related part of the liver is common. It may cause sudden death several days after the injury. Contusions may cause sudden death from ventricular fibrillation, or they may cause progressive circulatory failure and death after few hours or days. Once the heart or brain is contused, that portion dies. Foreign bodies, e.g., bullet, may remain embedded in the myocardium for years without producing any symptoms.

Cardiac concussion: Blunt cardiac trauma insufficient to produce gross or microscopic damage can cause ventricular fibrillatin or asystole [inhibition]. The functional disturbance may be caused by reflex coronary vasoconstriction and myocardial ischaemia or by abnormal autonomic responses [cardiac concussion; commotio cordis].

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. 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. 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 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. 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. 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. Separation of the heart from its attachments is possible due to traction force on the organs away from the neck. 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 intimae. The rupture of the aorta commonly occurs at the junction of the arch and the descending parts, just beyond the origin of the left 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, or a fall from a height or from compression of the trunk. It usually occur 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 thracoabdominal injuries. Death due to herniation of the abdominal contents into the chest 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 longitudinal splits on the lateral or posterior wall. 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:**
    a. In open pnumothorax, air enters the pleural cavity through an open wound in the chest wall. If less air enters the pleural cavity through the opening in the chest wall than through the trachea and bronchi, the lung can expand on inspiration and pulmonary ventilation is little affected. If more air enters the pleural cavity through the opening in the chest wall than through the trachea and bronchi, mediastinal flutter is produce. In this condition, the mediastinum is forced towards the normal side on expiration. It may cause severe circulatory embarrassment. In compressive or tension pneumothorax, defects in the injured visceral pleura or chest wall produce a valve-like action, which allows the air to enter the pleural cavity but prevents its escape. This results in the complete collapse of the affected lung, mediastinal displacement and cardiac and respiratory failure.
    b. In closed pneumothorax, air passes into the pleural cavity through a wound in the lung and visceral pleura. Collapse of the lung occurs due to pneumothorax.
2) **Haemothorax:** It is often associated with a pneumothorax. It may result from:
    a. A penetrating wound of lung.
    b. A penetrating wound of the heart or one of the large blood vessels, and

c. From injury to an intercostals or internal mammary vessel. Blood in the pleural cavity is often fluid due to;
    i. Insufficient production of thrombokinase, if tissue destruction is less,
    ii. Defibrination of blood due to mechanical agitation from respiratory movements of the lung.

3) **Chylothorax:** Effusion of chyle intot he right pleural cavity occurs from injury to the thoracic duct [a] by puncture, [b] by laceration from fractured rib ends, or [c] by rupture from indirect force.
4) **Interstitial emphysema:** In injury of the lung, air may be forced into the disrupted alveolar wall during inspiration. This air cannot be evacuated during expiration and tracks along the interstitial tissue of the lung. The air may reach the pleura and produce large subpleural blebs, which may rupture and produce a pneumothorax. Injuries of the trachea or large bronchi may result in the infiltration of the mediastinal tissue by air, which may extend upwards into the tissues of the neck, face and upper limbs. Circulatory failure may result from mediastinal and cervical interstitial emphysema due to compression of large veins in the thorax and neck.
5) **Arterial air embolism:** Air may be aspirated in to the punctured or lacerated pulmonary veins and carried into the systemic circulation, and lodge in the terminal segments of the cerebral arteries.
6) **Cardiac tamponade:** Accumulation of blood in the pericardial sac is the commonest cause of cardiac tamponade. Intrapericardial haemorrhage may be caused by
    a. Penetrating wound of the heart or large blood vessel.
    b. Lacerations of the ribs or the sternum.
    c. Rupture of the heart or aorta from indirect force.

When the parietal pericardium is punctured or lacerated, tamponade occurs if the blood accumulates in the pericardial sacfaster than it can escape through the wound in the pericardium. 300 to 400 cc. Of blood in the pericardial sac usually causes death. accumulation of blood in the pericardial sac interferes with ventricular dilatation during diastole and compresses the right atrium and great veins. Progressive circulatory failure occurs due to fall of arterial blood pressure and rise of venous blood pressure.

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. Severe or fatal internal haemorrhage may occur without any sign of injury on the abdominal wall especially if clothing overlies the area. sometimes, the skin of the abdomen and chest is not damaged, but he 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 inure abdominal viscera. When these wounds heal, the viscera protrude in the scar. A ventral hernia may take place. 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: 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. 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. 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 are often multiple and occur along the length of the antimesenteric border of the bowel. The jejunum is the commonest site of rupture, followed b 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 greater curvature. Traumatic rupture of the stomach may occur due to overdistention, as when an anesthetic 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.

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 an 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: Wounds of the pancreas are very rare. The pancreas may be injured by compression forces usually where it overlies the second lumber vertebra, when the viscera are crushed against the spinal column. 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. They are usually accompanied by injuries to other viscera. 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.

LIVER: It is the most frequently damaged abdominal organ and is second only to the brain in overall visceral susceptibility. 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 recognize. Blunt force to the abdomen may produce the following types of hepatic lacerations:

1) Transcapsular laceration 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) Subscapsular laceration over the convex surface of the liver under the site of impact.
3) Noncommunicating or central lacerations in the substance of the liver.
4) Coronal laceration over the superior surface due to distortion.
5) Lacerations of the inferior surface due to distortion.
6) Contrecoup laceration involving the posterior surface.

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. Fixation of any part of an organ makes the site more vulnerable to blunt trauma. 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: 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 contractiono f the abdominal muscle may predispose the spleen to rupture when it si 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 localized or generalized 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 from 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 amy be ruptured when a person is run over by a vehicle, or when 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: 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 ad 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 perforation, 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 catheterization 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.

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. 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: 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 man 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 the head, both vault and base can fracture, with extrusion of brain. When the person falls on to the feet, the tibia and femur can fracture at any level, often bilaterally. The femoral necks can fracture, the hip joints can dislocate, and there may be fracture dislocation of sacro-iliac joints, fracture of the spine at mid or upper thoracic level, and ring fracture of the skull. 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.

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 localized 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. Arteriospasm may occur due to an injury in the vicinity of a large artery, etc., e.g., the disruptive force of bullet passing near the femoral artery may produce arteriospasm even without injury of the vessels wall. Crushing injuries of the soft tissues in which arteriospasm occurs may or may not be accompanied by external signs of injury. a varying length of a main limb artery is affected by the spasm which usually lasts for about 24 hours, but may be prolonged to 3 to 4days. Associated spasm may develop: a) in the main branches of the vessel, distal to the site of contraction, b] in the trunk of the artery, proximal to the site of spasm, c] in the vessels of the collateral circulation.

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 organize, 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. An aneurysm develops at the site of the injury due to the protrusion of the inner coat of the artery through the opening. The injured aorta may retain overall integrity for several days or weeks, and later rupture with fatal haemorrhage.

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 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 perists 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 contracture 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.

If a nerve is completely divided Wallerian degeneration develops in the distal portion. The proximal segment develops initial limited degeneration. Later the axis cylinder of the proximal segment regenerated and extend towards the prolifering nerve sheath of the distal portion. Some function is restored, if contact is established between the segments. When a peripheral nerve is completely divided, spontaneous regeneration is uncommon. When a nerve is compressed or crushed, the general structure is preserved. The axis cylinders are interrupted and Wallerian degeneration occurs in the distal portion. Axis cylinders regenerate rapidly and restore function. Contusion, traction, or concussion of a nerve produce a transient nerve block. Axons do not degenerate, but conduction is temporarily lost. Normal function is restored within 7 to 40 days. Lesions of peripheral nerves may produce paralysis or sensory loss, with spontaneous recovery. Nerve injuries may develop some weeks or months after fractures and dislocations due to late compression by strapping, plasters or splints.

BONES: 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. 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. 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.

In a fall from a height, the tibias may be driven through the soles of the feet. The calcaneum is usually fractured if the heel is struck. Sometimes, the hip joints, pelvis or sacroiliac joints are injured. Other skeletal injuries, especially compression fractures of the dorsolumbar region may occur. The major blood vessels may be torn near their junction with the heart, and small horizontal tears of the intima of the carotid arteries may be found. The heart may be driven through the pericardial sac and the diaphragm. 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.

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 2 days. Acute inflammatory changes are seen within hours of injury and last for several days. After partial organization of blood clot, the fracture gap may be filled with condensed highly eosinophilic fibrin. Within one to 2 days, there may be migration of polymorphs into the necrotic tissue and later macrophages. Osteogenic granulation tissue is formed in four days. New vessels are formed and numerous fibroblasts are seen in 7 days. Proliferation and infiltration of osteogenic cells is seen at the same time. The fibroblasts lay down reticulin and then collagen, which is usually well marked by ten days. 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 obliterate by 30 days. Callus is formed into hard bone in about two months, although the bone still undergoes reconstructive modeling. After healing is complete, an approximate estimation of the age can be given from the extent of remodeling, the smoothness of edges, and the form of trabeculae passing through the line of fracture.

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 organized with intra-articular abdesions. 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, graduate 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

TRANSPORTATION INJURIES

Vehicle of transportation are responsible for a great number of fatalities all over the world. Ideally, Forensic Pathologist should visit the scene of death. in vehicular accidental deaths, retrospective visit may also be helpful in making conclusion about the death. photographs should be an integral part of the scene investigation A meticulous Autopsy helps to study the Nature and Pattern of Injury,Cause of death and circumstances

Surrounding the death which in turn may influence the Road Construction, Saftey precautions in Motor Vehicles and also help evaluate standards in Management of Victims of Road Traffic Collusions in the Emergency Medical services.

(A) **Injuries to Pedestrains:** Three patterns of injury are often seen:

(1) *Primary impact injuries (the first part struck),*

(2) *Secondary impact injuries (further injuries caused by the vehicles).*

(3) *Secondary injuries, (injuries caused by the victims striking other objects such as the ground).*

In primary impact injuries, the part of the body involved depends upon the position of the person in relation to the vehicle when struck. The injuries also depend upon the relative heights of the various parts of the vehicle, i.e., bumper, 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 vehicle. He may sustain a fracture-dislocation of the lumber or thoracic spine. 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 is facing the vehicle he may sustain intra-abdominal and intra-thoracic injuries. When he is thrown clear of the vehicle he may sustain head injuries. Frequently, bumper injuries are at different levels on the two legs or absent on one leg, which suggest the victim was walking or running when struck. An impact against a mud-guard or head lamp may cause fracture of the pelvis or fracture-dislocation of the sacro-iliac joints. The pubic ramus opposite to the buttock or hip involved is usually fractured. If the victim is struck from behind, striaelike superficial tears of the abdomen or the inguinal regions are seen due to over-stretching of the skin. When a pedestrian walks into the side of a vehicle, crushing abrasion or tears on the side or front of the face are produced. The chest or loin may be injured with fractured ribs and rupture of lung, diaphragm, spleen or liver. Tearing wounds may be caused by protruding objects such as door handles, or with cuts from broken glass. The most extensive fractures are produced by the head striking a flat surface. If the person is run over there may be:

(1) Tyre marks

(2) Abrasions and lacerations

(3) Burning of the skin.

(4) Deep crushing of the trunk or limbs with rupture of internal organs.

Injuries to cyclist and motor cyclists: The cycle is hit, and the person is thrown violently to the ground or into some other object or on the car. In being thrown he may injure the groins or legs by entanglement with handlebars or projecting levers or mirror and sustain tears. The head or shoulder may hit some object or they may be run over by the vehicle. Fractures of the skull are common due to secondary impact with ground.

Injuries to occupants of the Vehicles: The driver may be thrown forward. The horn boss may buckle the sternum and produce a transverse fracture and crush the heart or split the aorta. The heart, aorta and the lung root may be pinned and split against the spine. The wheel rim may crush the liver spleen or kidney. The throat may be crushed across the top of the steering wheel. These are known as “the steering wheel impact type of injuries”. If the driver puts force or his hands on the steering wheel to avoid impact on his chest, he may sustain injuries to wrists or forearms. If he applies breaks, fractures of the femur or pelvis, may occur. The driver and front passengers may split the head or face on the wind screen, or may sustain a fracture-dislocation of the cervical spine due to ducking of the head or being thrown up as well as forward. In head on crash the passengers move forward or sustain abrasions and lacerations on the shins and knees or fracture of one or both patellae. The femur or the pelvis may be fractured by being thrown against the dashboard or shelfedge. Sometimes the door may spring open and the person may fall out. When the car overturns the occupants may be pinned and crushed, showing traumatic asphyxia. Rarely the occupants are burned, asphyxiated or drowned whilst pinned in interior of a car.

INVESTIGATION OF AUTOMOBILE ACCIDENTS.

Scene: Tracks leading to and from the scene indicate direction of travel, speed and attempts to stop or avoid collision. The broken head lights, wind-shield, bumper, mudguards, parking lights, mirrors, spotlight etc., should be collected. Skid marks and other road markings are described, measured and photographed. The locations of vehicles, the type of road, the location and positions of victims on the road, distance and direction of the victims from the impact, and size and direction of blood on the road should be noted, photographed and drawings made.

The case vehicles: The vehicles involved in accidents are photographed from various angles to record the type and amount of damage. Measurements of vehicle are made to determine the amount of the formation and collapse of various structures. Photographs of the interior of the vehicle should be taken, especially areas of deformation and of the impact points. Look for human tissue, blood, hair, cloth fibres, etc., at the possible points of impact.

The accident victims: In fatal accident, photographs of the victim should be taken to show his position relative to the vehicle.

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 marks and dirt on the clothes must be noted. The clothes may show imprints of tyre, pieces of glass or metals.

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. The secondary impact injuries should be described.

AUTOPSY: The main objects are

A. Reconstruction of the accident:

Scene investigation is essential.

Drag marks may indicate that the victim had crawled from the vehicle and was not ejected. Injury sustained by dragging or scraping is usually superficial and show typical brush burning.

The victim's injuries indicate the mechanism of the accident.

B. Cause of the accident:

i. Natural disease processes in the driver.

Epilepsy

Diseases of the heart and blood vessels.

Hypoglycaemia

Rupture of congenital aneurysm of the cerebral vessels.

Brain tumour

Poor sight or hearing as a result of disease or ageing.

ii. Other causes: Alcohol, Barbiturates, tranquilizers, etc., Carbon-monoxide.

iii. Suicide: Suspicion may arise from the circumstances preceding the accident, e.g., family quarrels, financial crises, threats of suicide, history of depressing, suicide note, etc. In all cases the

absence of skid marks and a high speed impact against a highway object which is not particularly hazardous are significant factors.

iv. Other factors: Mechanical failure and Poor design and maintenance of highway.

v. Psychic factors: Disorganization of skilled reactions may occur in persons who are emotionally upset or suffer from anxieties.

vi. Pedestrians: Cardiovascular or cerebrovascular disease, poor vision, impaired hearing and reduced mobility.

C. Cause of death:

Severe blunt force injuries will point to a traumatic cause of death

Severe natural disease alone or in combination with significant trauma will point to a natural cause of death

Minor trauma may be sufficient by affecting diseased organs or vial areas, e.g., nervous centers of the brain or the conduction system of the heart.

D. Manner of death:

Accident

Accident and natural disease combinations

Suicide:

- Single occupant crashing into a fixed object.
- Pedestrian walking or running in front of moving vehicle.
- Other methods employed for suicide: (a) Drugs plus accidents. (b) Suicidal incised or stab wounds plus accidents. (c) Poisoning plus accident. (d) Homicide: (1) Deliberate running down of victim or vehicle. (2) Hit and run (failure to stop and render aid).

Driver:

A. To identify the driver and reconstruct the positions of the victims of the accident: The driver can be identified by:

Position in the vehicle upon discovery

The presence of steering wheel impact type of injuries

Finding of paint or glass from the door on the driver's side on the victim's body or clothes

Finding of cloth fibres, hair and tissue from the suspected driver on the driver panel, door or glass on the driver side or in the steering wheel and columns.

The brake or accelerator pedal design imprinted on the sole of the shoe.

Matching of the blood group of the driver with that of the driver panel, driver and steering assembly.

Finger prints on the steering wheel

Specific injuries caused by objects so positioned in the vehicle that a passenger could not have been so injured.

Witness to crash having no vested interest in the crash.

B. The passengers:

By position in vehicle upon discovery.

Finding of paint or glass from the door on the victim's body or clothes

Finding of cloth fibres, hair and tissue from the passenger on the doors and inside of the vehicle

Matching of the blood group of the passengers with that of the blood and different parts of inside of the vehicle.

Specific injuries caused by objects so positioned in the vehicle that the driver could not have been injured.

C. Do not know whether driver or passenger:

Confusing cross-transfer of evidence between vehicle and occupant.

No injury pattern clearly indicating whether driver or passenger

Ejection of occupants

No witness

D. The pedestrian:

1. Injuries which fit a predicted pattern:

Point of impact

Bumper fractures

Specific injuries that fit striking objects, e.g., patterned abrasions and contusions,

'Moving head' type of coup-contre-coup cerebral contusions

2. Cross-transfer of physical evidence:

Transfer of grease, paint or broken glass from striking objects to victim

Transfer of blood, hair, fibres and tissue to the vehicle.

Patterned imprints of fabric in paint or dust on the vehicle

Dents in metal at impact points that fit with the pedestrian's injuries

ii) **Evidence from a witness.**

E. Do not know; body lying on the road: I) Rarely may have no external injuries and be a vehicle-pedestrian accident victim ii) Injuries may not be visible, e.g., fracture of cervical vertebrae. Iii) Hit and run: no vehicle to examine for transfer of evidence. Iv) A dead person on the road may be run over by the vehicle.

F. Individual precipitated either accidentally or deliberately from the vehicle:

Permit rapid stop and following vehicle may run over or strike the precipitated individual.

Individual deliberately precipitated from the vehicle: (i) Usually dumped in darkness or in

unraveled area. (ii) High level of alcohol or drugs, or dead or incapacitated by injury. (iii) Pattern of injury may not fit with the scene where found.

To differentiate injuries caused by being hit from those caused by being run over.

To identify the vehicle in cases of hit and run:

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 vehicle.

Measurement of the leg fractures from the heels of the feet, and the height of other body injuries sustained from protruding part of the vehicle.

The body or clothing of the victim may show oil, rust, paint, glass pieces, etc., from the vehicle.

To determine the significance of previous injuries in case of delayed death: Previous injuries in case of delayed death: Morphologic proof that the trauma can be traced to the time of the accident will prove causal relationship, in delayed traffic deaths.

Precautions:

Collect all information about the circumstances of the crash including police investigation reports and photographs.

Do inspect the vehicle.

Take photographs and draw diagrams of all external evidence of injury.

Collect, preserve and retain anything that is likely to establish a correlation between the pedestrian victim and the offending vehicle, e.g., hair, blood, clothing, paint, grease, glass, etc.

Doctors Responsibilities:To determine the cause of death.

To confirm that death was due to injuries suffered in the accident.

Determine the extent of these injuries

Detect any disease or factors that could have precipitated or contributed to the accident or death.

To detect any criminal activity associated with the death

Document all findings for subsequent use in either criminal or civil actions

Establish positive identification of the body especially if it is burnt or severely mutilated.

Examination of the scene:

Time and date of accident.

Name(s) of decedent(s), whether Driver, passenger or pedestrian

Position’s of victims and degree of injuries

Brief background information about the decedent if suicide suspected

Findings indicating suspicion of foulplay

Make and position of vehicle and degree of damage

Behaviour of vehicle prior to crash

Points of impacts and relative positions of vehicles (if more than one)

Estimated speed of vehicle and posted speed limit

Condition of road.

Skid marks, brake marks

Weather conditions (rain, fog) and lighting condition

Autopsy

Reconstruction of the crash to determine its cause

Determination of the cause of death.

Determination of the Manner of death

Establishment of the identity of the decedent

Identification of the driver and reconstruction of the positions of the victims prior to the accident

Differentiation of injuries caused by being hit from those caused by being run over

Identification of the automobile in the case of hit and run incidents.

Determination of the significance of these injuries in a case of delayed deaths

Documentation of the findings to aid legal processes.

Scene investigation with photographs

Collection of evidence-clothing, paint traces, glass splinters, oil spots, rust, bloodspot

Complete autopsy with recording of injuries by description, drawings and photograph

Follow up studied – Examination of vehicle, driver, and alcohol intoxication.

Fall from heights is one of the major challenges posed by the Pathologist in concluding the Cause and Manner of Death.

All deaths due to fall may not necessarily be due to Trauma, Natural Causes should also be taken into Account besides other aspects like the Diseases process and Influence of Drugs (Alcohol) which should be carefully analyzed.

In deaths due to Trauma the Manner of death should be carefully analyzed by studying the circumstances surrounding the death.

The Deaths Investigation in falls:

Scene in falls

High level fall

Falls from a height (over 10-12 feet)

Experimentation.

Fall from high places usually come within the accidental or suicidal category; only vary occasionally they are proven homicidal in nature. One must remember the possibility that a dead body may be thrown down from the edge of a building and may sustain postmortem injuries.

Whenever a body is found near a high rise building or other place where a fall could have occurred, careful reconstruction of the scene is conjunction with postmortem findings is essential in order to ascertain whether:

- Fatal injuries are caused by a fall from height
- Fatal injuries were caused by some other means at ground level e.g. scene beating or vehicular accident.
- The immediate environment of the body is where the injuries were sustained.
- There are other signs/injuries on the body which indicate circumstances other than a fall.

Scene assessment:

The following items need to be considered

- The place from where personal fell
- The place from where the body was found
- The route taken by body to reach the ground

Low level falls (under 10-12 feet)

- Falls from higher level, descending to the point of final impact i.e. stair fall
- Falling from one's own height.

Falls from stairs:

- The presence of injuries to the body, if there are no injuries, then one should consider the possibility that the person died of natural causes and collapsed at the bottom of the stairs.
- If there are injuries, whether they are consistent with falling, or being propelled down stairs, bearing in mind the type of stairs, width, the number of flights involved, structure of stairs and wells etc.
- Possible evidence of a fight at, say, the top landing with the person receiving the most severe injuries there before falling.

All these possibilities should be considered along with post-mortem findings and circumstances.

Fall from one's own height:

- Tripping
- Stopping surface
- Sudden pushing
- Imbalance
- Obesity

It is always worth considering to measure height from the floor and furniture surfaces, such as chair or a bed, from which a person may have fallen, in conjunction with the type of floor covering.

Assessment type of material and hardness of surface of item of furniture.

Free falls:

Well over half of high voltage injuries occur in workers on towers and poles more than twenty feet above the ground. Free fall injuries therefore are the result. The nature and magnitude of the injury depend on

Height of fall

The impact of surface (stopping distance0

Body mass

Body orientation on impact forces

Distribution of impact forces

Patient's age which affect tissue tolerance

The majority of kinetic energy generated during fall is converted to mechanical energy which is dissipated through the tissues generating fractures and ruptures of visceral organs. The major treatment problem is failure to recognize existence of blunt injury in electric burn patient.

Experimentation:

A dummy body corresponding to Height and Weight of the deceased is tested for the alleged circumstances of fall, the experiment Photographed and Video graphed which after careful analysis will give a clue about the Nature and Pattern of Injury corroborating the Manner of death.

PRE-ACCIDENT ORGANISATION

Team of the pathologist, aviation agency investigation, local law enforcements agents, medical examiners, funeral directors and fire brigade and civil defence personnel to discuss and prepare plans for the handling of the accident.

should prepare a list of supplies and the place from which these can be obtained at short notice- like body pouches, name tag plastic bags, flashlights, photographic equipments and for recording of findings of the investigation.

Scene Investigation and Autopsy

Seal off the area of disaster to prevent spectators and press reporters from interfering with the investigation.

Take photographs of the wreckage and bodies, before and after placing identifying numbers.

Examine the aircraft and its lost parts including the seats, seat belts and surrounding structures

Help the medical investigators to examine the bodies of the victim before they are removed.

Pathologists

Photograph the body, with and without clothes and take x-ray (when indicated)

Describe clothing and personal effective data

Obtain Finger Print or ask police to do it

Make dental record or request dentist

Note Identifying features weight, color of eyes, skin and hair, tattoos, scan, old bony deformity,

absence of organs, surgery, or diseases

Describe and photograph , external injuries and burn and record them on body diagram.

Make internal examination, record and photograph.

Look for Natural disease Condition and describe in detail

Retain tissues for histological studies and toxicological analysis of hair samples for identification.

Collect Samples for DNA fingerprinting.

Follow up Investigation

Reconstruct the mishaps

Additional investigation to identify cause of death and accident

Study of Finger Prints, DNA, dental and medical records to identify

Relative interviewed to obtain information about the body features, clothes and other personal effects

Histology

Toxicology-pilot and crew

Help establish approximate time of death

In homicide assist in identifying the person responsible for death