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Chapter 2 - Medico legal Autopsy

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DEATH & ITS CAUSES

Death is characterized by an irreversible & permanent loss of vital functions which causes cessation of life.

1.Heart disease

Heart disease has remained the leading cause of death In India for more than two decades fuelled by unhealthy diets leading to high blood pressure and the buildup up blood fats (plaque) inside the walls of the arteries, inactivity, obesity and smoking.

2. Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease, including pulmonary hypertension, occupational lung disease, and interstitial lung disease, cause irreversible damage the airways and other lung structures to lower breathing capacity. Lung diseases are not curable, but can be managed using treatments that dilate major air passages and improve shortness of breath. Smoking, air pollution, occupational chemicals, dust and frequent lower respiratory infections aggravate lung conditions.

3. Diarrhoea

Diarrhoeal dieases are one of the biggest causes of under-5 deaths, killing between 800,000 and one million children, hospitalising 900,000 and causing 327,000 visits to clinics each year. Adding Rotavirus vaccine to India's universal vaccination programme in 2016 to protect children against the leading cause of severe diarrhoea in young children, helped lower numbers rapidly.

4. Stroke

Stroke, which was ranked as the sixth biggest cause of death in 2005, rose to become the fourth biggest killer in India. The risk factors for stroke, which is also known as cerebrovascular disease, are the same as heart disease, but the disability caused by a brain attack is often higher as it may cause partial or full paralysis

5. Lower respiratory infections

With improved diagnosis and infection management, lower respiratory infections such as pneumonia, lung abscess and acute bronchitis have slipped one position down to become the fifth biggest cause of death. It's among the most common infection in older adults and people with lowered immunity from other infections, such as seasonal influenza. Symptoms include shortness of breath, weakness, fever, coughing and fatigue that persist for more than a week must be investigated.

6. Tuberculosis

India accounts for 2.8 million of the 10.4 million new tuberculosis (TB) cases globally, according to the World Health Organization's Global TB Report 2016. India's national programme provides free medicines and treatment to all but many patients do not complete the full course of medicine, which must be taken for six to eight months for uncomplicated disease. This leads to drug resistant infection, which takes longer to treat using more toxic and expensive medicines.

7. Neonatal preterm birth

With 80.8% of India's 226 lakh annual births taking place in hospitals, health centers and clinics, deaths from premature birth-related complications such as low birth-weight have dropped since 2005, when it was the fourth cause of death.

8. Self harm

Ranked the 10th cause of death in 2005, self harm or suicide is now India's eighth biggest killer. Data from the National Crime Records Bureau (NCRB) has recorded a corresponding increase of 17.3%, 1,33,623 in 2015, up from 1,13,914 in 2005, in suicides over the past two decades decade.

9. Road injuries

Death from traffic accidents rose three points over two decades. Road accidents rose by 3.1% in one year, from 4,50,898 in 2014 to 4,64,674 in 2015 – with deaths going up by 5.1%, from 1,41,526 to 1,48,707 during the same period, shows NCRB data. States that show the sharpest increase were Kerala, Uttar Pradesh and Chhattisgarh.

10. Other neonatal conditions

Breastfeeding, vaccination against common infection and neonatal care is helping more babies thrive and survive neonatal infections such as septicaemia, birth asphyxia and birth trauma.

AUTOPSY:

Autopsy implies examination of the dead body with a view to searching primarily for the cause of death.

The medico-legal or forensic autopsy is performed on the instructions of the legal authority in circumstances relating to suspicious, sudden, obscure, unnatural, litigious or criminal deaths and the information so derived, to be applied for the legal purpose to assist the course of justice.

The present scenario of the constitution of the panel of doctors for conducting medico-legal autopsy in a particular case of unnatural or suspected death, is discussed keeping in view the legal, administrative, public interest and the academic aspects of the issues concerning such autopsies.

PURPOSE OF MEDICO-LEGAL AUTOPSY

Purpose of postmortem examination in a particular case of unnatural or suspicious death is to find out the following: -

1. To know the exact cause of death.

2. To find out the circumstances of death

3. To find out the postmortem interval.

4. In case of unidentified dead body, to establish identity of the deceased or to help to do so.

5. The period for which the deceased survived after sustaining injuries or exposure to poison.

6. To know the nature or the manner of death, whether natural, suicide or homicide.

7. Type of weapon or the poison used.

8. Whether one or more than one person was / was involved, in case of homicide.

9. Whether any natural disease process contributed in any way, to cause the death.

10. Whether any other offence was related with the death e.g. rape.

11. Is the injury, which has caused death, expected to cause death in ordinary course of nature.

12. Whether the dead body has been displaced from the original place of disposal.

13. To know whether more than one method or weapon were used or if more than one person were involved in the crime.

14. Whether the deceased received any treatment before death.

15. Whether there is anything on or with the dead body which may help identification of the assailant

16. In case of death due to assault, the relative positions of the victim and the assailant /s.

EXTERNAL EXAMINATION OF DECEASED BODY:

1. The examination of the clothing and worn accessories is an essential part of the external examination and all findings are to be clearly described. This is especially important in cases where the clothing or accessories have been damaged or soiled: each area of recent damage must be described fully and relevant findings are to be related to the site of injuries on the corpse. Discrepancies in such findings are also to be described.

2. All signs of recent or old medical and surgical intervention and resuscitation must be described. Medical devices must not be removed from the body before the intervention of the medico-legal expert.

3. The external examination should include:

a. age, sex, build, height and weight, nutritional state, skin colour and special characteristics (such as scars, tattoos or amputations);

b. post-mortem changes, including details relating to rigor and livores - distribution, especially their intensity, colour and reversibility - and putrefaction and environmentally induced changes;

c. findings on a primary external inspection which, if required, include sampling of stains and other trace evidence on the body surface and a reinspection after removal and cleaning of the body;

d. inspection of the skin including the posterior or downwardly facing surfaces of the corpse;

e. careful inspection of the head and the facial orifices. This includes hair and beard , nasal skeleton, lips, oral mucosa, dentition and tongue, ears (retro-auricular areas and external meati), eyes (colour of irises and sclerae, regularity and appearance of pupils, sclerae, conjunctivae), skin (including a description of presence or absence of petechiae); if fluids have been evacuated from facial orifices, their appearance, colour and odour;

f. inspection of the neck: checking for excessive mobility and/or creptiation, presence and absence of abrasions, other marks and bruising (including petechiae) over the entire circumference of the neck;

g. inspection of the thorax: shape and stability, breasts (aspect, nipples and pigmentation);

h. inspection of the abdomen: external bulging, pigmentation, scars, abnormalities and bruising;

i. inspection of the anus, perineum and genitals;

j. inspection of the extremities: shape and abnormal mobility, abnormalities; injection marks and scars; palmar surfaces, finger and toe nails;

k. material findings under fingernails.

4. Injuries must be described and documented.

INTERNAL EXAMINATION OF DECEASED BODY:

A. General

1. Relevant arte facts produced by sampling procedures must be documented.

2. The opening of the three body cavities is standard procedure.

3. Examination and description of body cavities includes: an examination regarding the possibility of gas being present (pneumothorax), measuring the volume of fluids and blood, regarding the appearance of internal surfaces, examining the intactness of anatomical boundaries, assessing the external appearance of organs and their location and detecting adhesions and cavity obliterations, injuries and haemorrhages.

4. Organs must be examined following the general principles of pathological anatomy.

5. All internal lesions and injuries must be precisely described by size and location. Injury tracks must be described in order to include their direction in relation to the organ anatomy.

B. Detailed

1. Head -The technique used for the examination of the cranium, must be adequate for the assessment of the scalp, temporal muscles, skull, meninges, cerebrospinal fluid, main blood vessels, and cranio-vertebral junction.

2. Thorax and neck the opening of the thorax must be performed using a technique which allows for the demonstration of the presence of a pneumothorax and the inspection of the thoracic walls, including the postero-lateral regions. In situ dissection of the neck must display the details of its anatomy.

3. Abdomen - The opening procedure of the abdomen must allow for an accurate examination of all layers of the walls. In situ dissection is necessary in certain cases. The whole intestine should be dissected and its contents described.

4. Skeleton - The examination of the thoracic cage, the spine and the pelvis is part of the autopsy procedure. b. Where appropriate traumatic (violent) deaths need a precise dissection of the limbs, possibly complemented by imaging procedures.

5. Special procedures

a. For the demonstration of particular injury patterns, deviations from the standard autopsy procedure are acceptable, provided that such procedures are specifically described in the autopsy report.

b. If there is any suspicion of neck trauma, the brain and thoracic organs are to be removed prior to the dissection of the neck, to enable detailed bloodless dissection.

c. In cases of suspected gas embolism, pre-autopsy imaging procedures should be performed. The autopsy technique must enable the forensic pathologist to diagnose an eventual gas embolism, to estimate the gas volume in the heart and to sample the intravascular gas.

d. Where appropriate, the examination should include a full exposure of the soft tissues and musculature on the back of the body. The same procedure must be applied to the extremities (so called "peel-of" procedure).

e. In suspected or obvious sexual assault, the sexual organs are to be removed "en bloc" together with the external genitalia, rectum and anus, before they are dissected. Relevant swabs of orifices and cavities must be taken prior to this procedure.

C. Sampling

This is to be performed in accordance to the SGRM/SSML/SSLM Guidelines for preserving autopsy material for forensic-toxicological analyses.

D. Release of the body

After a medico-legal autopsy has been carried out the body has to be returned in a dignified condition. This includes suturing of all incisions, and, if necessary and as far as possible, of facial injuries. The body should contain all internal organs. If whole organs are retained, this must be documented in the autopsy protocol.

Time Since Death

Sometimes there are witnesses who can state with certainty when a person died; other times there may be evidence such as a closed circuit video recording or a broken watch that can provide the information about the time of death. In the absence of direct evidence, it is up to the coroner to estimate time of death (T.O.D.). To do this, the coroner will consider the findings of experts such as identification officers, forensic pathologists and sometimes even forensic entomologists. "The time of death is sometimes extremely important. Determining the time of death is extremely difficult, and accuracy is impossible". No problem in forensic medicine has been investigated as thoroughly as that of determining the time of death on the basis of post mortem findings. Repeated experience teaches the investigator to be wary of relying on any single observation for

estimating the time of death (or "duration of the post mortem interval") and he wisely avoids making dogmatic statements based on an isolated observation.

Evidence for estimating the time of death may come from three sources:

1. Corporal evidence, i.e. that present in the body.

2. Environmental and associated evidence, i.e. that present in the vicinity of the body,

3. Anamnestic evidence, i.e. that based on the deceased's ordinary habits, movements, and day to day activities.

All three sources of evidence should be explored and assessed before offering an opinion on when death or a fatal injury occurred.

Death

Death is the irreversible cession of life.

Somatic death is the state of body that shows complete loss of sensibility and the ability to move and complete cession of the function of brain, heart and the lungs.

Moleculer or celluler death means the death of the tissues and the cells individually, which take place about 3-4 hr. after the stoppage of the vital functions. Signs of death:

The signs of death may be classified as early and late.

Early: Changes in the eyes, Changes in the skin, Cooling of body , Cadaveric lividity, hypostasis, post mortem staining

- Late: Cadaveric changes in the muscles, Putrefaction or decomposition, Mummification.

COMMON ESTIMATORS OF TIME SINCE DEATH

There are two methods for estimating the time of death:

1. Measuring the change produced by a process which takes place at a known rate which was either initiated or stopped by the event under investigation, i.e. death. Examples include the amount and distribution of rigor mortis, the change in body temperature, and the degree of putrefaction of the body.

2. Comparing the occurrence of events which took place at known times with the time of occurrence of the event under investigation, i.e. death. For example, a wrist watch stopped by a blow during an assault, the extent of digestion of the last known meal.

Postmortem changes and time of death

Many physico-chemical changes begin to take place in the body immediately or shortly after death and progress in a fairly orderly fashion until the body disintegrates. Each change has its own time factor or rate. Unfortunately, these rates of development of post mortem changes are strongly influenced by unpredictable endogenous and environmental factors. Consequently, the longer the post mortem interval, the wider is the range of estimate as to when death probably occurred. In other words, the longer the post mortem interval, the less precise is the estimate of the time of death.

CHANGES IN THE EYE:

Loss of corneal reflex: Soon after the death the eye loses its luster. The corneal reflex is lost; the cornea became opaque and looked like dimmed glasses.

Opacity of cornea: The cornea became dry, cloudy and opaque due to failure of production of tears. The speed with which the cornea became opaque after death is due to drying and is retarded if the lids are closed after death.

Pupil dilation: Pupil usually at the time of death and later became constricted through the development of rigor mortis. After death the blood stream in retinal vessels rapidly became segmented it appears within the minutes after death.

CHANGES IN SKIN:

After death the skin of whole body due to the draining of the blood from the small blood vessels of the skin, assume a pale and an ashy white appearance especially in fair bodies and loss of its elasticity. Wounds caused during life retain their red or blue color after death.

ALGOR MORTIS (BODY COOLING)

This is the most useful single indicator of the time of death during the first 24 hours post mortem. It is of some importance to note that the use of body temperature estimations to assess time of death applies only to cool and temperate climates since in tropical regions there may be a minimal fall in body temperature post mortem and in some extreme climates, the body temperature may even rise after death. The assessment is made on the basis of measurement of the body core temperature which, post mortem, requires a direct measurement of the intraabdominal temperature. In practice either the temperature is measured per rectum or the intra-hepatic/sub-hepatic temperature is measured via an abdominal stab. Oral and auxiliary temperatures should not be used. An ordinary clinical thermometer is useless because its range is too small and the thermometer is too short. A chemical thermometer 10-12" long with a range from 0-50^{o} Celsius is ideal. Alternatively a thermo-couple probe may be used and this has the advantage of a digital readout or a printed record. Whether the temperature is measured via an abdominal stab or per rectum is a matter of professional judgment in each case. If there is easy

access to the rectum without the need to seriously disturb the position of the body and if there is no reason to suspect sexual assault, then the temperature can be measured per rectum. It may be necessary to make small slits in the clothing to gain access to the rectum, if the body is clothed and the garments cannot be pushed to one side. The chemical thermometer must be inserted about 3-4" into the rectum and read in situ. The alternative is to make an abdominal stab wound after displacing or slitting any overlying clothing. The stab may be over the lower ribs and the thermometer inserted within the substance of the liver or alternatively a sub costal stab will allow insertion of the thermometer onto the undersurface of the liver. The body temperature should be recorded as early as conveniently possible. The environmental temperature should also be recorded and a note made of the environmental conditions (see below) at the time the body was first discovered and any subsequent variation in these conditions. If a method of sequential measurement of body temperature is use then the thermometer should be left in situ during this time period. This latter method is much easier to undertake when using a thermo-couple with an attached print-out device. Temperature readings of the body and observations made at the scene by one physician are always available for evaluation by an expert at a later time. The normal oral temperature fluctuates between 35.9ºC (96.7ºF) and 37.2ºC (99ºF). The rectal temperature is from 0.3-0.4ºC (0.5º-0.75ºF) higher (cited in reference 19 at p. 12). Since heat production ceases soon after death but loss of heat continues, the body cools.

During life the human body loses heat by radiation, convection, and evaporation. Heat loss by conduction is not an important factor during life, but after death it may be considerable if the body is lying on a cold surface. The fall in body temperature after death mainly depends upon a loss of heat through radiation and convection, but evaporation may be a significant factor if the body or clothing is wet.

It is usually assumed that the body temperature at the time of death is normal, but in individual cases it may be subnormal or markedly raised. As well as in deaths from hypothermia, the body temperature at death may be sub-normal in cases of congestive cardiac failure, massive haemorrhage, and shock. However, the claim that severe agonal bleeding lowers the body temperature is said to be without foundation. The body temperature may be raised at the time of death in heat stroke, some infections, and pontine hemorrhage.

The linear rate of post mortem cooling is affected by environmental factors and cadaveric factors other than the environmental temperature and the body temperature at the time of death.

1. **The "size" of the body.**

   The greater the surface area of the body relative to its mass, he more rapid will be its cooling. Consequently, the heavier the physique and the greater the obesity of the body, the slower will be the heat loss. In obese individuals the fat acts as an insulator, but for practical purposes body mass, whether from muscle mass or adipose tissue, is the most important factor. Children lose heat more quickly than adults because their surface area/mass ratio is

much greater. Prominent oedema in individuals with congestive cardiac failure is said to retard cooling because of the large volume of water present with a high specific heat whilst dehydration has the opposite effect. The effect of oedema fluid is said to be more potent than body fat. The exposed surface area of the body radiating heat to the environment will vary with the body position. If the body is supine and extended, only 80% of the total surface area effectively loses heat, and in the foetal position the proportion is only 60%.

2. Clothing and coverings.

These insulate the body from the environment and therefore cooling is slower. Simpson states that cooling of a naked body is half again as fast as when clothed. Henssge has graded the effect of clothing by the number of layers and thickness. He states that only the clothing or covering of the lower trunk is relevant.

3. Movement and humidity of the air.

Air movement accelerates cooling by promoting convection and even the slightest sustained air movement is significant. Cooling is said to be more rapid in a humid rather than dry atmosphere because moist air is a better conductor of heat. The humidity of the atmosphere will affect cooling by evaporation where the body or its clothing is wet.

4. Immersion in water.

A cadaver cools more rapidly in water than in air because water is a far better conductor of heat. For a given environmental temperature, cooling in still water is about twice as fast as in air, and in flowing water, about three times as fast. Clearly the body will cool more rapidly in cold water than warm water. It has been said that bodies will cool more slowly in water containing sewage effluent or other putrefying organic matter than in fresh water or sea water.

RIGOR MORTIS

Ordinarily, death is followed immediately by total muscular relaxation - primary muscular flaccidity - succeeded in turn by generalized muscular stiffening - rigor mortis. After a variable period of time rigor mortis passes off spontaneously to be followed by secondary muscular flaccidity. No measurable shortening of muscle occurs during rigor mortis unless the muscles are subjected to tension. When rigor is fully developed, the joints of the body become fixed, and the state of flexion or extension of these joints depends upon the position of the trunk and limbs at the time of death. If the body is supine then the large joints of the limbs become slightly flexed during the development of rigor. The joints of the fingers and toes are often markedly flexed due to the shortening of the muscles of the forearms and legs. Since significant muscle shortening is not a normal concomitant of rigor, it is unlikely that rigor mortis would cause any significant change in the attitude adopted by the corpse at death. It is now accepted that movements of a

corpse due to the development of rigor mortis can only occur in special circumstances, such as an extreme position of the body at the moment of death. If a body is moved before the onset of rigor then the joints will become fixed in the new position in which the body is placed. For this reason, when a body is found in a certain position with rigor mortis fully developed, it cannot be assumed that the deceased necessarily died in that position. Conversely, if the body is maintained by rigor in a position not obviously associated with support of the body, then it can be concluded that the body was moved after rigor mortis had developed. Rigor involves voluntary and involuntary muscles. Rigor of the myocardium should not be mistaken for myocardial hypertrophy. Likewise secondary muscular flaccidity of the atria and ventricles should not be mistaken for ante-mortem dilatation or interpreted as evidence of myocardial dysfunction. Involvement of the iris muscles means that the state of the pupils after death is not an indication of their ante-mortem appearance. Different degrees of rigor development may give rise to irregularity and inequality of the pupils. Contraction of the arrectores pilorum muscles during rigor may result in "goose-flesh" or "cutis anserina". The phenomenon is commonly seen in cases of drowning where it is thought to result from an agonal contraction of the muscles. Involvement of the walls of the seminal vesicles by rigor may lead to discharge of seminal fluid at the glans penis. Rigor mortis results from a physico-chemical change in muscle protein, the precisenature of which is unknown. When the muscle tissue becomes anoxic and all oxygen dependent processes cease to function, then the level of ATP is maintained by anaerobic glycolysis which results in increasing levels of pyruvic and lactic acids. Eventually, the muscle glycogen is depleted, the cellular pH falls to around 6, and the level of ATP falls below a critical level beyond which rigor rapidly develops. Normally ATP inhibits the activation of the linkages between actin and myosin; a fall in the level of ATP allows the irreversible development of these linkages. In individuals who have been exhausted or starved before death, the glycogen stores in muscle are low, so that rigor may develop rapidly. Classically, rigor is said to develop sequentially, but this is by no means constant, symmetrical or regular. Ante-mortem exertion usually causes rigor to develop first in the muscles used in the activity. Typically, rigor is first apparent in the small muscles of the eyelids, lower jaw and neck, followed by the limbs, involving first the small distal joints of the hands and feet and then the larger proximal joints of the elbows, knees and the shoulders and hips. Consequently, differences in the sizes of the joints, and in the muscles which control them, determine the development of joint fixation by rigor and produce the observed pattern of progression in the body. It is generally accepted that rigor mortis passes off in the same order in which it develops. The forcible bending of a joint against the force of rigor results in tearing of the muscles and the rigor is said to have been "broken". Provided the rigor had been fully established, it will not reappear once broken down by force. In temperate climates rigor will typically start to disappear at about 36-48 hours after death. However, if the environmental temperature is high then the development of putrefaction may completely displace rigor within 9-12 hours of death. The intensity of rigor mortis depends upon the decedent's muscular development; consequently, the intensity of rigor should not be confused with its degree of development. In examining a body both the degree (complete, partial, or

absent) and distribution of rigor should be assessed after establishing that no artefact has been introduced by previous manipulation of the body by other observers. Attempted flexion of the different joints will indicate the amount and location of rigor. As a general rule when the onset of rigor is rapid, then its duration is relatively short.

The two main factors which influence the onset and duration of rigor are

(a). The environmental temperature

(b) . The degree of muscular activity before death.

Onset of rigor is accelerated and its duration shortened when the environmental temperature is high. If the temperature is below 10°C it is said to be exceptional for rigor mortis to develop, but if the environmental temperature is then raised, rigor mortis is said to develop in a normal manner. In addition to these two principal factors, other endogenous and environmental factors are claimed to influence the onset of rigor. Onset is relatively more rapid in children and the aged than in muscular young adults. It develops early and passes quickly in deaths from septicaemia or from wasting diseases. It is delayed in asphyxial deaths, notably by hanging or carbon monoxide poisoning, and also when death has been immediately preceded by severe haemorrhage. Exposure of a body to intense heat results in heat stiffening due to coagulation of the muscle proteins. Unlike rigor mortis, heat stiffening is associated with muscle shortening resulting in the characteristic pugilistic posture of burned bodies. Heat stiffening obscures rigor mortis with which it should not be confused. Freezing of a body will cause stiffening of the muscles, postponing the development of rigor which is said to develop as soon as thawing of the body permits. Cadaveric spasm (synonyms: instantaneous rigor, instantaneous rigidity, cataleptic rigidity) is a form of muscular stiffening which occurs at the moment of death and which persists into the period of rigor mortis. Its cause is unknown but it is usually associated with violent deaths in circumstances of intense emotion. It has medico-legal importance because it records the last act of life. Cadaveric spasm may affect all the muscles of the body but it most commonly involves groups of muscles only, such as the muscles of the forearms and hands. Should an object be held in the hand, then cadaveric spasm should only be diagnosed if the object is firmly held and considerable force is required to break the grip. Cadaveric spasm involving all the muscles of the body is exceedingly rare and most often described in battle situations. Cadaveric spasm is seen in a small proportion of suicidal deaths from firearms, incised wounds, and stab wounds, when the weapon is firmly grasped in the hand at the moment of death. In such circumstances the gripping of the weapon creates a presumption of self infliction of the injuries. This state cannot be reproduced after death by placing a weapon in the hands. It is also seen in cases of drowning when grass, weeds, or other materials are clutched by the deceased. In this circumstance, it provides proof of life at the time of entry into the water. Similarly, in mountain fatalities, branches of shrubs or trees may be seized. In some homicides, hair or clothing of the assailant may be found in the hands of the deceased.

LIVOR MORTIS (HYPOSTASIS, POST MORTEM LIVIDITY, POSTMORTEM SUGGILLATIONS)

Lividity is a dark purple discolouration of the skin resulting from the gravitational pooling of blood in the veins and capillary beds of the dependent parts of the body following cessation of the circulation. The process begins immediately after the circulation stops, and in a person dying slowly with circulatory failure, it may be pronounced very shortly after death. Lividity is present in all bodies, although it may be inconspicuous in some and thus escape notice.

Lividity is able to develop post mortem under the influence of gravity because the blood remains liquid rather than coagulating throughout the vascular system. Within about 30- 60 minutes of death the blood in most corpses, dead from natural or non-natural causes, becomes permanently incoagulable. This is due to the release of fibrinolysins, especially from small calibre vessels, e.g. capillaries, and from serous surfaces, e.g. the pleura.

Clots may persist when the mass of clot is too large to be liquified by the fibrinolysin available at the site of clot formation. In some deaths associated with infection and cachexia, this fibrinolytic effect may fail to develop, explaining the presence of abundant clot in the heart and large calibre vessels.

Typically, lividity has a purple or reddish-purple colouration. Lividity in bodies exposed to the air may acquire a pink colour at the sides, but not, as rule, at the back or other areas which are close to the ground. In deaths from carbon monoxide poisoning, it is classically described as "cherry red"; in cases where methaemoglobin is formed in the blood during life (e.g. potassium chlorate, nitrates, and aniline poisoning) it appears chocolate brown; in deaths from exposure to cold, it is bright pink, and a similar coloration is seen in bodies refrigerated very soon after death. Refrigeration of a body already displaying typical purple Lividity will cause it to turn pink. Similarly, Lividity in parts of the body covered with moist clothes appears pink, whereas it is the usual purple color in other areas.

Lividity is first apparent about 20-30 minutes after death as dull red patches or blotches which deepen in intensity and coalesce over the succeeding hours to form extensive areas of reddish-purple discoloration. Slight Lividity may appear shortly before death in individuals with terminal circulatory failure. Conversely, the development of lividity may be delayed in persons with chronic anemia or massive terminal hemorrhage. After about 10-12 hours the lividity becomes "fixed" and repositioning the body, e.g. from the prone to the supine position, will result in a dual pattern of lividity since the primary distribution will not fade completely. Fixation of lividity is a relative, rather than an absolute, phenomenon, but nevertheless, well developed lividity fades very slowly and only incompletely. Fading of the primary pattern of lividity and development of a secondary pattern of lividity will be quicker and more complete if the body is moved within, say, the first six hours after death, than at a later period.

Even after 24 hours, moving the body will result in a secondary pattern of lividity developing. Duality of the distibution of lividity is important because it shows that the body had been moved after death. Pressure of even a mild degree is sufficient to prevent gravitational filling of the vessels and this is so in the compressed areas of skin in contact with the underlying supporting surface. The result is that these compressed areas of "contact flattening" also show "contact pallor" (or "pressure pallor"). A supine corpse will display contact pallor over the shoulder blades, buttocks, calves and heels. Other areas of contact pallor will correspond with the location of firm fitting clothing, e.g. elasticated underwear, belts and collars, and any firm object lying beneath the body, e.g. the arm of the decedent. Thus, the distribution of lividity depends upon the position of the body after death. Within intense areas of lividity, the accumulated blood may rupture small vessels to produce a scattering of punctate purple-black haemorrhages between one and several millimetres in diameter. These haemorrhages are seen most commonly over the lower legs of victims of suicidal hanging with complete suspension. These haemorrhagic loci should be distinguished from ante-mortem petechial haemorrhages. Lividity is usually well marked in the earlobes and in the fingernail beds. In a supine corpse there may be isolated areas of lividity over the front and sides of the neck resulting from incomplete emptying of superficial veins. If the head is slightly flexed on the neck, then lividity may have a linear distribution corresponding to the skin folds. Isolated patches of hypostasis may be due to blood in the deeper veins being squeezed, against gravity, to the skin surface by the action of muscles developing rigor mortis Differentiation of lividity from bruising can be made by incising the skin. In areas of lividity the blood is confined to the dilated blood vessels whilst, in areas of bruising, the blood infiltrates the tissues and cannot be readily washed away under running tap water. Microscopic examination will resolve any doubts and provide a permanent record. In a decomposing body it may be impossible to definitively distinguish between livid staining of the tissues and a putrefying area of bruising. Areas of lividity are overtaken early in the putrefactive process. The red cells haemolyse and the haemoglobin diffuse into the surrounding tissues where it may undergo secondary changes such as sulphaemoglobin formation. In bruised areas similar putrefactive changes occur and it may be impossible to determine whether the pigment in the stained putrefied area originated from an originally intravascular or extravascular collection of blood, i.e. from a patch of congestion or from a bruise. Lividity occurs in the viscera as well as the skin and this provides some confirmation of the external observations. In the myocardium lividity may be mistaken for an acute myocardial infarction, and in the lungs may be misdiagnosed as pneumonia. Livid coils of intestine may falsely suggest haemorrhagic infarction. Lividity developing in the viscera of a body lying prone and resulting in a purplish congestion of organs usually found pale at autopsy can be disconcerting to those unaccustomed to these changes. Lividity attains its maximum intensity at around 12 hours post mortem, but there is some variation in descriptions of when it first appears, and when it is well developed. Lividity "ordinarily becomes perceptible within 1/2 to 4 hours after death, is well developed within the next 3 or 4 hours, and attains its maximum degree between 8 and 12 hours post mortem". it varies in its time of onset, is ordinarily apparent within 1/2 to 2 hours after death, and

its complete development is attained in from 6 to 12 hours". It is usually well developed within 4 hours and reaches a maximum between 8 and 12 hours. After 8 to 12 hours lividity becomes "fixed" and will remain where it originally formed". It commences to develop within an hour or so of death, becoming marked in 5 or 6 hours.

POSTMORTEM DECOMPOSITION (PUTREFACTION)

Putrefaction is the post mortem destruction of the soft tissues of the body by the action of bacteria and enzymes (both bacterial and endogenous). Tissue breakdown resulting from the action of endogenous enzymes alone is known as autolysis. Putrefaction results in the gradual dissolution of the tissues into gases, liquids and salts. The main changes which can be recognized in the tissues undergoing putrefaction are changes in colour, the evolution of gases, and liquefaction. Bacteria are essential to putrefaction and commensal bacteria soon invade the tissues after death. The organisms most commonly found are those normally present in the respiratory and intestinal tracts, namely anaerobic spore-bearing bacilli, coliform organisms, micrococci, diphtheroids and proteus organisms. The marked increase in hydrogen-ion concentration and the rapid loss of oxygen in the tissues after death favour the growth of anaerobic organism. The majority of the bacteria come from the bowel and *Clostridium welchii* predominates. Any ante-mortem bacterial infection of the body, particularly scepticaemia, will hasten the onset and evolution of putrefaction. Environmental temperature has a very great influence on the rate of development of putrefaction so that rapid cooling of the body following a sudden death will markedly delay its onset. In the temperate climate the degree of putrefaction reached after 24 hours in the height of summer may require 10 to 14 days in the depth of winter. A high environmental humidity will enhance putrefaction. Putrefaction is optimal at temperatures ranging between 70-100oF (21-38oC) and is retarded when the temperature falls below 50oF (10oC) or when it exceeds 100oF (38oC). The rate of putrefaction is influenced by the bodily habitus of the decedent; obese individuals putrefy more rapidly than those who are lean. Putrefaction will be delayed in deaths from examinations because blood provides a channel for the spread of putrefactive organisms within the body. Conversely, putrefaction is more rapid in persons dying with widespread infection, congestive cardiac failure. Putrefaction is accelerated when the tissues are oedematous, e.g. in deaths from congestive cardiac failure, and delayed when the tissues are dehydrated. It tends to be more rapid in children than in adults, but the onset is relatively slow in unfed new-born infants because of the lack of commensal bacteria. Whereas warm temperatures enhance putrefaction, intense heat produces "heat fixation" of tissues and inactivates autolytic enzymes with a resultant delay in the onset and course of decomposition. Heavy clothing and other coverings, by retaining body heat, will speed up putrefaction. Rapid putrefactive changes may be seen in corpses left in a room which is well heated, or in a bed with an electric blanket. Injuries to the body surface promote putrefaction by providing portals of entry for bacteria and the associated blood provides an excellent medium for bacterial growth. After normal burial, the rate at which the body decomposes will depend to a

large extent on the depth of the grave, the warmth of the soil, the efficiency of the drainage, and the permeability of the coffin. The restriction of air, in deep burials, particularly in clay soil, will retard decomposition, but never prevent it altogether. Buried in well drained soil, an adult body is reduced to a skeleton in about 10 years, and a child's body in about 5 years. Immersion of the body in faeces-contaminated water, such as sewage effluent will enhance putrefaction; however, it is generally accepted that in the first 48 hours after death changes are in the main due to organisms already present in the body. Typically, the first visible sign of putrefaction is a greenish discolouration of the skin of the anterior abdominal wall. This most commonly begins in the right iliac fossa, i.e. over the area of the caecum, (where the contents of the bowel are more fluid and full of bacteria), but occasionally, the first changes are peri-umbilical, or in the left iliac fossa. The discolouration, due to sulph-haemoglobin formation, spreads to involve the entire anterior abdominal wall, and then the flanks, chest, limbs and face. As this colour change evolves, the superficial veins of the skin become visible as a purple-brown network of arborescent markings, which tend to be most prominent around the shoulders and upper chest, abdomen and groins. This change, owing to its characteristic appearance, is often described as "marbling". The skin, which now has a glistening, dusky, reddish-green to purple-black appearance, displays slippage of large sheets of epidermis after any light contact with the body, e.g. during its removal from the scene of death. Beneath the shed epidermis is a shiny, moist, pink base which dries, if environmental conditions permit, to give a yellow parchmented appearance. This putrefactive "skin-slip" superficially resembles ante-mortem abrasions and scalds. Indeed, post mortem scalding of a body with water at 65oC (149oF) produces skin slip of the same type as in putrefaction. Subsequently, skin blisters varying in size from less than 1 cm to between 10 and 20 cm in diameter develop. These blisters are filled with dusky, sanguineous fluid and putrid gases. They burst on the slightest contact leaving the same slippery, pink base which underlies skin-slip. Putrid gas formation also occurs in the stomach and intestines causing the abdomen to distend and become tense. The increased pressure within the torso causes a purge of putrid, blood-stained fluid from the nose, mouth and vagina, and expulsion from the rectum of similar fluid admixed with faeces. Gas formation within the tissues causes generalized swelling of the body which is crepitant on palpation. The distention is greatest where the tissues are loose, particularly involving scrotum, penis, labia majora, breasts, and face. The gases produced include hydrogen sulphide, methane, carbon dioxide, ammonia and hydrogen. The dusky, greenish-purple face appears bloated with the eyelids swollen and tightly closed, the lips swollen and pouting, the cheeks puffed out, and the distended tongue protruding from the mouth. The head hair and other body hair is loose at its roots and can be easily pulled out in large clumps. The finger and toenails detach, often with large sheets of contiguous epidermis forming complete "gloves" or "socks" - a process described as "degloving". The neck, trunk and limbs are massively swollen, giving a false impression of gross obesity. Finally, the putrid gases, which are under considerable pressure, find an escape and the whole mass of decomposing soft tissues collapses. Putrefaction progresses internally beginning with the stomach and intestine. The gastric mucosa and the intestines are discoloured a brownish-purple. The mucosa of the airways

is a deep red and there is haemolytic plum-coloured staining of the endocardium and the vascular intima which is most readily appreciated in the aorta and its major branches. Small white granules - so-called "miliary plaques" - are seen rarely over the endocardium and epicardium. The heart becomes flabby, the wall thinned, and the myocardium a deep dirty red. A similar discolouration is seen in the liver and kidneys. The spleen becomes mushy and friable. The liver develops a honey-comb pattern resulting from gas formation and similar changes may be seen in the brain, most readily if it is fixed in formaldehyde prior to cutting. Subsequently the brain becomes semiliquid. The lungs, loaded with sanguinous fluid, appear dark red and are friable. Gradually a great part of this sanguinous fluid is lost by diffusion into the pleural cavities. Diffusion of bile pigments from the gall bladder discolours the adjacent liver, duodenum and transverse colon. The capsules of the liver, spleen and kidneys resist putrefaction longer than their parenchymatous tissues with the result that these organs are often converted into bags of thick, turbid, diffluent material. Progression of decomposition is associated with organ shrinkage. The more dense fibro-muscular organs such as the prostate and uterus remain recognisable until late in the process, thus aiding in the identification of sex. Perforation of the fundus of the stomach or lower oesophagus into the left pleural cavity or the abdomen may occur within a few hours of death. This is the result of autolysis rather than bacterial putrefaction. An uncommon finding, it is most frequently associated with cerebral injuries and terminal pyrexias. It is occasionally characterized as "neurogenic perforation of the oesophagus". There is considerable variation in the time of onset and the rate of progression of putrefaction. As a general rule, when the onset of putrefaction is rapid then the progress is accelerated. Under average conditions in a temperate climate the earliest putrefactive changes involving the anterior abdominal wall occur between 36 and 72 hours after death. Progression to gas formation occurs after about one week. The temperature of the body after death is the most important factor generally determining the rate of putrefaction. If it is maintained above 26oC (80oF) after death then putrefactive changes become obvious within 24 hours and gas formation will be seen in about 2-3 days. The putrefactive changes which have taken place up to this time are relatively rapid when contrasted with the terminal decay of the body. When the putrefactive juices have drained away and the soft tissues have shrunk, the speed of decay is appreciably reduced.

ADIPOCERE

Saponification or adipocere formation is a modification of putrefaction characterized by the transformation of fatty tissues into a yellowish-white, greasy, (but friable when dry), wax-like substance, with a sweetish rancid odour. Meant states that when its formation is complete it has a sweetish smell, but during the early stages of its production a penetrating ammoniacal odour is emitted and the smell is very persistent. It floats on water, and dissolves in hot alcohol and ether. When heated it melts and then burns with a yellow flame. Ordinarily it will remain unchanged for years. Adipocere develops as the result of hydrolysis of fat with the release of fatty acids which, being acidic, then inhibit putrefactive bacteria. The low (0.5%) level of free fatty acids in fat at the time of death may rise to 70% or more by the time adipocere is obvious to the naked

eye. However, fat and water alone do not produce adipocere. Putrefactive organisms, of which Clostridium welchii is most active, are important, and aipocere formation is facilitated by post mortem invasion of the tissues by endogenous bacteria. A warm, moist, anaerobic environment thus favours adipocere formation. It was once thought that adipocere required immersion in water or damp conditions for its development. However, the water content of a body may be sufficient in itself to induce adipocere formation in corpses buried in well sealed coffins. Adipocere develops first in the subcutaneous tissues, most commonly involving the cheeks, breasts and buttocks. Rarely, it may involve the viscera such as the liver. The adipocere is admixed with the mummified remains of muscles, fibrous tissues and nerves. The final product is of a larger bulk than the original fat with the result that external wounds may become closed and the pattern of clothing or ligatures may be imprinted on the body surface. Under ideal warm, damp conditions, adipocere may be apparent to the naked eye after 3- 4 weeks. Ordinarily, adipocere formation requires some months and extensive adipocere is usually not seen before 5 or 6 months after death. The medico-legal importance of adipocere lies not in establishing time of death but rather in its ability to preserve the body to an extent which can aid in personal identification and the recognition of injuries. The presence of adipocere indicates that the post mortem interval is at least weeks and probably several months.

MUMMIFICATION

Mummification is a modification of putrefaction characterized by the dehydration or desiccation of the tissues. The body shrivels and is converted into a leathery or parchment-like mass of skin and tendons surrounding the bone. The internal organs are often decomposed but may be preserved. Skin shrinkage may produce large artefactual splits mimmicking injuries. These are particularly seen in the groins, around the neck, and the armpits. Mummification develops in conditions of dry heat, especially when there are air currents, e.g. in a desert or inside a chimney. New-born infants, being small and sterile, commonly mummify. Mummification of bodies of adults in temperate climates is unusual unless associated with forced air heating in buildings or other man-made favorable conditions. The forensic importance of mummification lays primarily in the preservation of tissues which aids in personal identification and the recognition of injuries. The time required for complete mummification of a body cannot be precisely stated, but in ideal conditions mummification may be well advanced by the end of a few weeks.

VITREOUS HUMOUR POTASSIUM

Vitreous humer is the transparent gelatinous substance that fills the posterior chamber of eye. During life potassium conc. is low in vitreous humour but much higher in the peripheral tissue of eye .This electrolytic imbalance result from energy consuming vital activities. The postmortem cessation of which leads to progressive reversal of potassium gradient with the consequence of rise in vitreous conc. With the help of these equation we can determine the time of death: Sturner’s formula -: TSD = 7.14[K]-39.1 Madea’s formula-: TSD= 5.26[K]-30.9 TSD - Time since death [K] – Pot. Conc.(m.mol/ l) The Digestive System The digestive system and gut

contents of a victim can provide important clues to the time of death of a victim. Chewed food will firstly pass through the oesophagus and then down into the stomach within seconds of the initial swallowing. After 3 hours, the food then leaves the stomach and heads toward the small intestines. 6 hours after eating a meal, the food will have traveled half way through the small intestines and begin moving through the large intestine. Where the victim's small intestine is empty, it suggests that the victim ate his or her last meal approximately 8 hours before death. The digestive process usually takes a bit more than a day, but it can be affected by sickness, liquid intake, and fear or drug intake. Pathologists also briefly note that correct level of food digestion corresponds to its location in the digestive system. In the rare case that a clever murderer wishes to delude investigators by attempting to bring forward the time of the victim's last meal (giving them an explanation for where they were at the victim's time of death), he/she may manually feed processed food (resembling that of chewed food) into the victim's stomach. If this is so, the food collected in the stomach will be much less digested than abnormal, since the periodic motion of the stomach stops after death. The food may indeed appear slightly broken down, due to the presence of the stomach acids, but any abnormalities are otherwise detectable. In older people or in those affected by the effects mentioned earlier (sickness, fear, drug/liquid intake), the efficiency of food digestion alters and it is left to pathologists to determine if the extent of the undigested food is great enough to suggest the mentioned scenario.

ENTOMOLOGY

After death, the tissues of animals become attractive to a large variety of insects and other invertebrates. These may be classified into four groups:

• Necrophagous species: invertebrates that feed on the corpse itself.

• Predators and parasites of the necrophagous species: these species do not feed directly on the corpse.

• Omnivorous species: invertebrates that feed both on the corpse and on the other arthropods present.

• Adventives or opportunistic species: invertebrates present on the corpse by chance, or using it as an extension of their usual environment (e.g. as a shelter, nest) without feeding on it. The use of entomologic markers for determining the time of death is based upon the long - established observation that insects and other arthropods feeding on a corpse follow a specific faunal succession associated with the various stages of decay. Estimations of time of death require an accurate recognition of the sometimes numerous species present on a corpse, or its surroundings, in their different immature (eggs, larvae, puparia) or adult stages of growth, together with an extensive knowledge of their specific rates of development according to environmental parameters (season, temperature, humidity, etc.). Such investigations are difficult and can be undertaken only by experienced, full -time specialists in forensic entomology. Provided this is the case, this technique currently constitutes the only approach for estimating the time of death of

a putrefied cadaver with some accuracy - in some cases, to within a few days, even in deaths obviously dating back for months.

Injuries – Classification – Medico-legal aspects of injuries

Definition : An injury is defined as the termination of the natural continuity of any of the tissues of the living body.

The term hurt, injury and wound have almost the same meaning and some differences as well. They usually mean any damage to any part of the body or bodily harm caused by application of violence. Injury also includes any harm to the mind, reputation and property, and hurt includes bodily pain, disease or infirmity.

Production of Injuries:

A wound is produced when the intensity of the applied force to the body exceeds the capability of the tissue to adapt or resist the force. The capability of the tissue to adapt or resist the force depends on mass, velocity, exact area of the weapon pressing the body, structure of the tissue, mode of application of the applied force like compression, traction, torsion, tangent and leverage of the stress. Type of damage of the tissue depends on the degree of transfer of the kinetic energy from the relative movement of the weapon and/or the body.

Type of Injuries:

Injuries are typed on the basis of their appearance and the method of causation. Abrasion- It is the damage occurring in the epidermis; some dermal papillae may also be damaged. It is caused by rough hard blunt objects or surfaces or by drawing the tip of the pointed objects against the skin or mucous membrane. Most abrasions are caused by rubbing effect and others are caused by vertical pressure. Pattern of the impacting object may be retained at the abraded area.

Bruise: It is the extravascular collection of blood in the tissue, viz, dermis of the skin, subcutaneous tissue and other deeper tissues due to rupture of blood vessels caused by application of blunt force with overlying tissue remaining intact so that blood cannot escape outside. When the lesion is visible from outside it is called bruise and if not visible from outside it is called contusion. Bruises may be of different sizes, viz, petechial haemorrhage of pin head size, purpura of size 2 to 5 mm, echymosis of more than 5 mm, haematoma causing local swelling. Laceration- it is the tearing or splitting of the skin, mucous membrane and surfaces of any internal organs caused by application of blunt force. Blood escapes from the wound to the exterior or any body cavity.

Fracture: It is the breach of continuity of bone or tooth caused by application of blunt force. Clinical features and X-ray findings are considered to diagnose this type of lesion. In dislocation of joint the bone ends get displaced completely from their normal anatomical positions with damage to the adjacent tissues.

Incised wound: It is caused by the sharp edge of the weapon applied to the skin perpendicularly or obliquely making regular clean edges of the wound. It is called a slash wound when the length of the wound is greater than its depth. when the depth of wound is greater than its length and usually caused by pointed knife or dagger. A puncture wound is produced when a pointed thin bodied weapon is pushed into the body. Its depth is also greater than its diameter. A penetrating wound is produced when a bigger type of pointed weapon like teta is pushed into the body making a greater depth. When puncture or penetrating wound is such that it has an entry and an exit and a tract through the tissue it is called perforating wound. Legally puncture and penetrating wounds are also considered as stab wounds.

Firearm wounds: These wounds are produced by bullet or pellets fired from guns. Bullet usually causes perforation and pellets cause penetrating wounds. Firearm entry wounds are associated with burning, blackening, tattooing of the surrounding skin.

Blast wounds: This are produced by explosion of bombs and are due to blast pressure wave (shock wave), blast winds, heat, splinters, shrapnel, surrounding small fragments propelled by blast winds.

To produce wounds the weapons are applied to the body or the body is applied to the weapon with various velocities. Different wounds have different characteristics and can easily be identified.

Medical documentary evidences: Medicolegal reports on hurts prepared by the medical practitioners are very important for the courts in making their legal judgments. The type of wounds and weapons, legal categories of hurts and their ages must be specifically noted in the injury reports. Medicolegal training and experiences strengthen the abilities of the medical expert witnesses. Punishment in term of fine fixed about 20 years ago now needs to be reconstituted at a higher level.

Post- mortem changes -

A body undergoes complex and intricate changes after death. These post mortem changes depend on a diverse range of variables. Factors such as the ambient temperature, season, and geographical location at which the body is found, the fat content of the body, sepsis/injuries, intoxication, presence of clothes/insulation over the body, etc. Understanding the post mortem changes is imperative to estimate the time since death (TSD) or the post mortem interval (PMI).

Classification of Post Mortem Changes

Based on the order of their appearance, the post mortem changes classify as immediate changes, early changes, and late changes.

Immediate changes:

Immediate changes after death relate to the 'somatic death' or 'systemic death.' Somatic death deals with the irreversible cessation of the vital functions of the brain, heart, and lungs. Thus, immediate post-mortem changes are dubbed as the "signs or indications of death." Immediate changes include insensibility, loss of voluntary movements, cessation of respiration, cessation of circulation, and cessation of nervous system functions. During this time, primary relaxation of muscles occurs.

Cessation of respiration is checked by placing a stethoscope over the upper parts of the lungs where the slightest sound of breathing, if any, can be detected. Another alternative to using a stethoscope is the use of electrocardiograph (ECG). A flat ECG is indicative of cessation of circulation. Nervous system function cessation is detected using an electroencephalograph (EEG). Brain stem reflexes require checking, as well. Absence of respiratory sounds and movements, heartbeats, brain stem reflexes, ECG, and EEG activity signifies death and are noted as the signs observed immediately after death.

Early post mortem changes:

Early post mortem changes are associated with cellular death. They include changes in the skin, eyes, post mortem cooling (algor mortis), post mortem rigidity (rigor mortis), and post mortem staining (livor mortis).

After death, the skin of an individual becomes pale, ashen, and it loses elasticity within a few minutes of death.The lips become dry and hard. Numerous ocular changes are observable after death, which includes corneal opacity, loss of pupillary and corneal reflex, and loss of intraocular tension that leads to ocular flaccidity. If the eyes are left open after death, there is a deposition of dust in the exposed part of the eye.

Changes in temperature:

After death and cessation of circulation, the convectional transference of heat inside the body comes to a halt. Since no heat is being produced within the cadaver, the body starts losing heat due to the temperature difference between the body and the surroundings. This decrease in body temperature after death is termed as 'algor mortis,' and is used to estimate the post mortem interval (PMI).For estimating the PMI, the temperature of the body is measured using a 'thanatometer,' which is a 25 cm long thermometer with a range of 0 to 50 degrees C. The thanatometer gets inserted inside the rectum and records the temperature. The rate of fall of temperature is measured by recording the rectal temperature at regular intervals.

Changes in the muscles:

Immediately after death, the muscles undergo primary relaxation, as mentioned above, which is followed by stiffening of muscles known as rigor mortis. With the onset of putrefaction, rigor mortis passes off, and secondary relaxation occurs. Secondary relaxation occurs at around 36 hours after death due to the breakdown of the contracted muscles due to decomposition.

Rigor mortis is the post mortem stiffening/ rigidity of the body. It results from a decrease in levels of adenosinetriphosphate (ATP) beyond critical levels. When a person dies, calcium ions flood muscle fibers due to the loss of integrity of the muscle cells. These ions cause the binding of actin and myosin filaments, causing contraction. Relaxation of muscles is achieved by ATP driven pumping of the calcium ions back into the sarcoplasmic reticulum of the muscle cells. As a result of the lack of ATP, the muscles fail to relax, and the actinomyosin complex created

during the contraction stays intact; this causes the muscles to become hard and rigid. Rigor mortis first appears in the involuntary muscles of the heart, and apparently follows proximal to distal progression. It is observed in eyelids, followed by the neck, lower jaw, chest, upper limbs, abdomen, lower limbs, and then finally in the fingers and toes. Rigor mortis appears in 1 to 2 hours after death, is completely formed 12 hours after death, is sustained for the next 12 hours, and vanishes over the next 12 hours, sometimes referred to as the 'march of rigor.'

Livor mortis:

Circulation of blood is a continuous process carried out by the pumping action of the heart in a living individual. However, once the person dies, the circulation comes to a halt, and the blood starts moving towards the dependant regions of the body due to gravity. This effect results in reddish-blue staining of those low-lying dependent regions of the body, known as the livor mortis, post mortem staining, post mortem lividity, or post mortem hypostasis.During the initial phases, patches of discoloration start appearing in the dependent regions in 1 to 3 hours after death. These increase in size and spread all over the dependent regions in 4 to 6 hours and are fully developed within 6 to 8 hours. So, in case of the body of an individual lying on the floor of a room, the back of the individual will show postmortem staining.

Late post mortem changes:

Autolysis:

Autolysis (self-destruction) is an intrinsic activity brought about by the breakdown of cells and tissues of the human body because of the constituents of the said cells. Just after death, the cell membranes breakdown and release enzymes that start self-digestion. The first external sign of autolysis is the whitish appearance of the cornea.

Putrefaction:

Putrefaction is the decomposition of the body carried out by the microbial action. After cessation of homeostasis, the natural flora of the body migrates from the gut to the blood vessels and spreads all over the body. External micro-organisms enter the body through the alimentary canal, respiratory tract, and open wounds. In the absence of body defenses/immune mechanisms, the microbes keep growing, as they feed upon the proteins and carbohydrates of the blood and body parts. The principal bacterial agent causing putrefaction is the gram-positive, anaerobic, and rod-shaped *Clostridium welchii*.It releases lecithinase, which causes hydrolysis of lecithin present in the blood cells, causing their lysis. Putrefaction begins within an hour of death, but the peak activity of the microbes occurs around the 24-hour timeframe.

Putrefaction of erythrocytes within the superficial blood vessels leads to the formation of greenish-blue discoloration, which is observable through the skin. This outlining of the superficial blood vessels is known as 'marbling' of the skin.

By the end of 24 or 48 hours, maggots can be seen near the external orifices and/or the open wounds. These maggots, depending on the species of the fly, pupate and become adults by 6 to 8 days and can be used to estimate the PMI. By 5 to 10 days, there is liquefaction of most of the internal organs, the abdomen may burst due to the pressure exerted by the putrefactive gases, and the ligaments become softer by this stage.

Adipocere formation:

adipocere formation is a modification of the putrefaction process, which involves hydrolysis and hydrogenation of fatty tissues into a yellowish, greasy, rancid, wax-like substance called adipocere.This adipocere consists of mainly palmitic, oleic, and stearic fatty acids, and contains glycerol in smaller amounts. Adipocere formation most commonly presents in fatty regions such as the cheeks, chin, abdomen, and buttocks.

Mummification:

Mummification is a modification of the putrefaction process characterized by the desiccation or dehydration of the cadaveric tissues. The skin of the deceased becomes brown, hard, and brittle and has a stretched appearance over prominence like the zygomatic bones, mandible, etc. The body shrivels and shrinks in size, but the facial features and the injuries are preserved, as in the case of adipocere formation.

Collection of post mortem samples and preservation

Retention of Biological Tissues for Laboratory Testing Certain precautions should be taken during the selection and preservation of specimens to be analyzed.

Labeling containers: A specimen should never be placed in an unlabeled or even a partially labeled container. The container should always be fully labeled before the specimen is placed in it, and the label should contain the following information: official name of the medical examiner's or coroner's office , name of the deceased, case number, date and time, name of the medical examiner and a description of the contents.

Tissues for microscopic examination: Retaining tissues from all organs, even from normal-appearing organs, for microscopic examination should be an integral part of all autopsies and should be routinely done. In the investigation of drug-involved deaths, microscopic examination of tissues is often neglected. There is a preoccupation with and a tendency to rely solely on chemical and toxicological tests, the results from which are not always clear cut. Microscopic studies can provide supporting or confirmatory evidence.

Tissues for chemical analysis: Each specimen taken for drug or poison analysis should be placed in its own separate container, fully and legibly identified with the proper information. Each specimen should also be accompanied by a test request sheet indicating the specific drug or group of drugs to be tested for. Selection of the tissues and the amounts will vary with the tests to be performed.

Blood: Blood should always be saved for testing whenever possible in an medical examiner/coroner's cases. Submission of blood alone, however, for toxicological examination in drug-related deaths is fraught with danger. Many drugs are rapidly removed from circulating. Unless .highly sophisticated technological Skills and ll1struments are available, some drugs may not be detectable in blood within as short a time as 5-10 minutes after oral or parenteral administration.

Skin and subcutaneous tissues: Dissect out and retain for microscopic examination and chemical analyses the skin and Subcutaneous tissues at sites of drug injection, injuries, and other areas of significance. The skin and subcutaneous tissues at the site of a recent drug injection may show foreign body reaction to the extenders mixed with the drug. In suspected heroin deaths,double refractile materials may be seen in the tissue at the injection site under a phase contrast microscope.

Nails: Two or three whole nails should be retained for chemical analysis in cases of suspected arsenic or heavy metal poisonings.Remove nail from toe with a forceps by inserting one of its blades under the nail plate to obtain a good grip and pulling with a twist. This can be done with little damage to the nail bed. Place the nails in a properly and legibly labeled envelope.

Hair: Hair samples should be retained for analysis in suspected arsenic and various heavy metal poisonings.Retain samples from head and pubic areas. Remove by plucking to Qbtain whole lengths of hair including roots.

Bone: For bone samples, remove 1-2 inch lengths of half of the body of the vertebrae with an electric bone saw.

Brain: The head is often forgotten in medicolegal examinations unless there is ex- ternally obvious injury. No postmortem examination is complete without dissection of the head. Absence of externally obvious signs of injury does not rule out the possibility of brain injury. Even in cases of drug deaths with confirmed high blood levels of drugs, unless the head has been opened and the brain examined, possible brain injury as an alternative direct cause of death cannot be disputed with certainty.

Neck organs: Examination of the neck organs is essential for excluding other possible causes of death. The lumina of the neck organs cannot be inspected in situ adequately. Remove by a block dissection the tongue, pharynx, larynx, and the trachea. Dissect and examine for:

1. Evidence of trauma to the soft tissues

2. Fractures of the hyoid or other bony or cartilaginous structures

Cardiovacular system: The heart and the aorta should be carefully examined, especially in older persons, to rule out deaths due to cardiovascular diseases. Many drugs, including those used for the treatment of heart diseases, will cause changes in the heart tissues.

Lungs: In cases where inhalation of gas or chemical vapor is the suspected cause of death, sections of the lung tissue should be retained for chemical and microscopic analyses.

Stomach: Tie or clamp off the stomach at both ends, above the diaphragm and at the duodenal junction, and remove intact from body. Dissect and collect entire contents into a large chemically clean; properly labeled jar.

Intestines: Tie off the intestine into easily handled segments (1-2 ft) and remove the whole length from the body. Open each segment separately and retain contents of each segment in separate containers which are properly identified with the location of the segment as well as the information identifying the case. Carefully examine contents and the mucosal lining. Record all significant findings and retain tissues for microscopic examination.

Liver: Liver should be retained in drug related deaths, for many drugs are rapidly concentrated in the liver for detoxification, and appreciable quantities may be found in the liver in acute drug deaths while blood concentration may be minimal. Dissect out the whole liver; examine for gross pathological changes; retain appropriate areas for microscopic examinations and for drug analyses as needed.

Gallbladder and bile: Many drugs are concentrated and retained in the gallbladder for as long as 48 hours and high concentrations in the bile are usually indicative of chronic drug usage. Remove the whole gallbladder with contents and retain bile in properly labeled jar for chemical analyses as needed.

Kidneys and urine: Some portion of practically all of the drugs and chemicals taken into the body will be excreted or resorbed into the body through the kidneys. Many of these substances are nephrotoxic and damage the kidneys. High concentration of a drug in the urine is usually indicative of chronic usage. Remove both kidneys. Dissect and examine for abnormalities.