Reference Manual

On

Injury Examination

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Table of Contents

Introduction

Any doctor may be asked to examine an individual with an injury, resulting from an act of an accident, self-harm or intentional harm by others. It is essential for the doctor to examine and document the entire process carefully, completely and correctly. The documentation and reporting can have far-reaching medico-legal implications, civil or criminal, and may become evident only after months or even years.

These days the ligations for physical assault have greatly increased. As a result, it is important for doctors to document all procedures with the utmost care, as one can never predict which cases may be examined in a court of law.

Documentation should include the date, time and place of the examination. In addition, the doctor should also record the history as narrated by the patient. Following detailed history taking, a full examination of the patient should be performed followed by examination of the injury(s) present on the body. Documentation of the injury should include Type, Size, Shape, Site, Age and Location of the injury(s) with reference to fixed anatomical landmarks.

In addition to these factors, all medicolegal reports also require an opinion on the gravity of the injury e.g. grievous or simple; fatal or non-fatal.

Hence, it is evident that the diction used to describe injuries are extremely important, especially in a legal context. As a result, the specialized medical meaning of words must only be used correctly and not in the ‘lay’ sense; for example, the term ‘incision’ must only be used to describe a break in the continuity of the skin or tissue, caused by a sharp edged object. When in doubt, it is preferable to use ‘lay’ terms like cut, bruise, graze etc. when describing the type of the wounds than to use medical terminology incorrectly. The courts, reasonably, expects doctors to use specific medical terminology when writing a professional report, correctly and accurately.

A ‘wound’ and an ‘injury’ are not distinguished in law, except in a charge of wounding where the skin must be ‘breached’; such a definition would exclude rupture of the liver by a kick if the skin was not ‘breached’.

Legally, an assault refers simply to the act of threat and could include a number of form including threatening to injure another person, verbally abusing and threatening the safety of a loved one etc.

There does not need to have been any physical contact for someone to be charged with the offence of assault.

On the other hand, an actual attack involving physical contact is termed ‘battery’. While battery requires actual physical contact to take place between the assailant and the victim, there does not need to be any injury as a result of the contact.

Muluki Ain, 2020 B.S. defines hurt or battery as “causing bloodshed, wound, injury, grievous hurt or any pain or harm to the body of another person”.

In general parlance, however, the word ‘assault’ is commonly used to describe incidents with and without physical contact.

The documentation and analysis of injuries following the examination of an injured, does not require any specialist knowledge and falls within the competencies required of any medical doctor who has completed a full M.B.B.S. degree. However, a doctor with no specialist forensic training may, correctly, feel insufficiently qualified to interpret the wounds. In such a scenario, the doctor should resist any pressure to perform such examinations and should refer the patient to someone with greater experience in the field for an opinion. If however, the doctor does not accept his/her limitations at this stage, the lack of experience will not be accepted as an excuse for negligent documentation or interpretation of findings.

This guideline is therefore intended to provide medical practitioners who examine medicolegal injury cases with valuable references for consulting during examination. As a result, we intend this guideline to help bring uniformity in the reporting of injuries in medicolegal cases, civil or criminal, and thereby aid in the delivery of justice.

Rationale

Medical Officers and all physicians examine injuries regularly during the course of the treatment of patients. While live saving procedures and treatment always remains the priority, the legal aspect of medical examination and treatment cannot be forgotten. This is an important principle of medical ethics, the deliverance of justice.

Medical procedures, by their very nature, are destructive and this make the timely and accurate documentation of the injuries even more important from a medico-legal perspective. While it may not be evident at the time, many cases have the potential to be pursued in a court of law, and this would require the attending physician to present his findings before court in a professional report as well as in person.

The hesitancy of the medical personnel to appear in court has been evident in meetings with the medical fraternity. This can only be attributed to the lack of knowledge of the legal procedure as well as legal provisions and requirements, in the process of examination of injuries.

There is also a lack of competence among the medical fraternity regarding the descriptive parameters of injury. This has resulted in widely varied reporting of injuries from medical personnel all over the country.

The proper interpretation of injuries is only possible following correct and accurate documentation. This usually starts any interventional procedures are performed on the person and should be accompanied by a detailed account of the event causing the injury, as described by the injured. A photographic recording is an ideal tool that can assist not only after destructive procedures have been performed but also after recovery, at a much later date.

The interpretation of the mechanism of causation of the injury is an imporatant aspect of medico-legal reporting and includes the gravity of injury – serious or simple as well as to the perceived fatality of the injury. Once again, development of this guidelines assists medical officers and all physicians, in increasing their knowledge on the causation of injury, the causative force, the types of injuries resulting from the force as well as the relevance of the region of the body that is injured.

Terminology

Injury is derived from the Latin word ‘injuria’ which literally means not lawful.

Medically: ‘‘Injury’’ refers to any damage to the body produced by energy exchanges that have relatively sudden discernible effects. Though diseases also cause damage to the body, it is not termed as injury because the injury produced by disease lacks suddenness.

However, this disregards injuries that are caused due to sustained pressure rather than forceful impact, for example the laceration caused by sustained pressure against the skin, from a thin wire.

Knight defines a wound or injury as ‘damage to any part of the body due to the application of mechanical force’. This definition also disregards various injuries caused due to non-mechanical force, for example, burn injuries.

Pounder states “wound can have a wider meaning and encompass not only damage produced by physical force, but also damage produced by heat, cold, chemicals, electricity and radiation.”

Vij defines injury as “solution of natural continuity of any tissue of the living body”. This also includes burns from fire/heat, electricity, all lacerations and bruises of internal organs/tissues and the effects of any corrosive or solid upon the body. It also prevents any possible criticism about the skin being or not being severed.

No definition of injury and wound, however, gives credence to recent advances in mental health. In addition the lack of a legal definition for injury has necessitated a medical definition that is easily understood and widely accepted.

the Indian Penal Code, which encompasses more than just damage not to the body. Injury is hereby defined as “Any harm illegally caused to a person, resulting in damage to his body, mind, reputation or property.”

For the purpose of this guideline, as a reference to assisting medical officers in the examination of medico-legal cases, we shall confine to analysis the bodily damages for the purpose of documentation and reporting.

Bio-mechanics of injury

The severity, extent, and appearance of injuries depend on:

- The amount of force delivered to the body
- The time over which the force is delivered
- The region struck
- The extent of body surface over which the force is delivered
- The nature of the weapon

The body is subjected to external mechanical forces, one of the most popular being gravity, as well as to other forces that come into contact with the body during the course of life. The body deals with these forces by absorbing the forces using either the elasticity of the soft tissue or the strength of the skeletal system. When the force applied exceeds the ability of the tissue to adapt or resist, when the force exceeds the breaking point, this result in an injury or wound.

The intensity of the force obeys the usual laws of physics,

$K.E = ½ \times mass \times (velocity\ of\ impact)^2$

in that the force varies directly with the mass of the object and directly with the 'square' of velocity of impact.

This means that a cotton ball, weighing 10 g thrown at the head would not cause any injury, while a brick, weighing 1 kg, thrown would cause fatal injuries. This also means that a brick pressed against the scalp will cause no injury, but the same brick thrown against the head at a velocity of 10 m/s may smash the skull.

This principle is relevant not only in relation to blunt injury and firearm missiles, but also to stab wounds. In addition, the object may be the deceased's body itself, as is often seen in the violent injuries sustained during deceleration seen in death due to fall from height or in traffic accidents.

Another importance factor in the effect of the force is the area over which the force acts. For any given amount of force, the greater the area over which it is delivered, the less severe the wound. The size of the area affected by a blow depends on the nature of the weapon as well as the region of body.

For example, if a plank of wood is struck against the skin, the injury cause will be far greater if the striking surface is the narrow edge, than if the wide flat surface was used to strike. The same mass and velocity, resulting in the same force, applied to a smaller area results in greater impact and more injury.

Similarly, a blow delivered to the thigh, will cause more severe injury than if the same force is delivered to a flat portion of the body, such as the back, where there will be a greater area of contact and more dissipation of force.

If an object projects from the surface of the weapon, then all of the force will be delivered to the end of the projection and a much more severe wound will be produced. This is relevant when analyzing both blunt trauma as well as sharp for injuries, especially the latter, where the kinetic energy of a moving knife gets concentration on a sharp, very tiny area, of the blade and even more so on the tip. The same energy delivered by a wide surface of a cricket bat may not result in any evident injury. The force is usually a form of compression, traction, torsion, as well as tangential and leverage stress.

The type of force dictates injury not only base on the energy transferred but also on the nature of target tissue. For example, violent compression may not cause much damage to muscles over the legs, but will cause significant damage to abdominal organs and may even cause rupture. Similarly, torsion of the soft tissue, especially adipose, may not result is severe injury, yet, it may cause a spiral fracture in the femur.

It is evident that the transfer of kinetic energy from the relative motions of the body and the object is the source of the injury.

The principles of slowing down and dissipating energy transfer to decrease the effect is part of the principle for function of a seatbelt in a vehicle crash. The stretching of the belt fabric extends the time of energy exchange and the considerable area of the belt surface is a preferable alternative to transferring all the kinetic energy via a few square centimeters of forehead against the windscreen, during collision.

This may also be experience in the transfer of kinetic energy by moving the body in the direction of the force. When a car strikes a pedestrian, part of the energy may be transferred to the pedestrian, resulting in him being projected along the road, this energy is not then available for damaging the

tissues. Similarly, if a boxer ‘rides the punch’, moves in the same direction as the force, he is reducing the differential velocity between his head and his opponent’s fist, as well as extending the time over which energy transfer occurs.

If the object comes to rest in the tissue, all its kinetic energy must have been transferred to the tissue. Also, if a weapon deforms or breaks on impact, less energy is delivered to the body to produce injury, because some of the energy is lost. Thus, the resultant injury is less severe than one would have if the weapon did not deform or break.

With a high-speed impact, such as a bullet and to a lesser extent a stab or violent blow, the inertia of the victim’s body prevents any significant loss of energy transfer. The relative velocity of the bullet is so great that a bullet that does not leave the body causes more damage. This is because a bullet which passes clean through the tissues fails to exchange all its energy into trauma production. This is the reason for the banning of explosive-tipped bullets, the object of which is to expand the missile and prevent its exit from the body, so increasing its damage.

If the force does not strike in a linear fashion, but leads to a tangential impact, as in a glancing blow causing a ‘graze’, then only part of the kinetic energy is transferred and the damage will be significantly less.

Forensic anatomy of the skin

As most wounds involve the outer body surface, in some capacity, a review of the structure of the skin and subcutaneous tissues seem appropriate.

The skin forms the external covering of the body and is its largest organ, constituting 15% to 20% of its total mass. The skin consists of two main layers:

- The epidermis is composed of a keratinized stratified squamous epithelium that grows continuously but maintains its normal thickness by the process of desquamation. Epidermis is derived from ectoderm.
- The dermis is composed of a dense connective tissue that imparts mechanical support, strength, and thickness to the skin. Dermis is derived from mesoderm. The hypodermis contains variable amounts of adipose tissue arranged into lobules separated by connective tissue septa. It lies deep to the dermis and is equivalent to the subcutaneous fascia described in gross anatomy. In well-nourished individuals and in individuals living in cold climates, the adipose tissue can be quite thick.

The epidermal derivatives of the skin (epithelial skin appendages) include the following structures and integumentary products:

- Hair follicles and hair
- Sweat (sudoriferous) glands
- Sebaceous glands
- Nails

The integumentary system performs essential functions related to its external surface location. Skin and its derivatives constitute a complex organ composed of many different cell types. The diversity of these cells and their ability to work together provide many functions that allow the individual to cope with the external environment.

Major functions of the skin include the following:

- It acts as a barrier that protects against physical, chemical, and biologic agents in the external environment (i.e., mechanical barrier, permeability barrier, ultraviolet barrier).

- It provides immunologic information obtained during antigen processing to the appropriate effector cells in the lymphatic tissue.
- It participates in homeostasis by regulating body temperature and water loss.
- It conveys sensory information about the external environment to the nervous system. It performs endocrine functions by secreting hormones, cytokines, and growth factors and converting precursor molecules into hormonally active molecules (vitamin D).
- It functions in excretion through the exocrine secretion of sweat, sebaceous, and apocrine glands.

In addition, certain lipid-soluble substances may be absorbed through the skin. Although not a function of skin, this property is frequently used to deliver therapeutic agents. For example, nicotine, steroid hormones, and seasickness medications are frequently delivered through the skin in the form of small sticking plasters or patches. To reduce nicotine withdrawal symptoms during smoking cessation, nicotine patches are often used to provide a small constant dose of nicotine without the dangerous effects of tobacco smoke.

Skin is categorized as thick or thin, a reflection of thickness and location. The thickness of the skin varies over the surface of the body, from less than 1 mm to more than 5 mm. However, the skin is obviously both grossly and histologically different at two locations:

The palms of the hands and the soles of the feet. These areas are subject to the most abrasion, are hairless, and have a much thicker epidermal layer than skin in any other location. This hairless skin is referred to as thick skin.

Elsewhere, the skin possesses a much thinner epidermis and is called thin skin. It contains hair follicles in all but a few locations. The terms thick skin and thin skin, as used in histologic description, are misnomers and refer only to the thickness of the epidermal layer.

Anatomically, the thickest skin is found on the upper portion of the back where the dermis is exceedingly thick. The epidermis of the upper back, however, is comparable to that of thin skin found elsewhere on the body. In contrast, in certain other sites such as the eyelid, the skin is extremely thin.

Layers of the Skin

Epidermis

The epidermis is composed of stratified squamous epithelium in which four distinct layers can be identified. In the case of thick skin, a fifth layer is observed. Beginning with the deepest layer, these are as follow:

- Stratum basale, also called stratum germinativum, because of presence of mitotically active cells, the stem cells of the epidermis;
- Stratum spinosum, also called prickle cell layer, because of appearance of short processes extending from cell to cell;
- Stratum granulosum, which contains numerous intensely staining granules;
- Stratum lucidum, (limited to thick skin and considered a subdivision of stratum corneum)
- Stratum corneum, which is composed of keratinized cells.

Stratum basale

The stratum basale is represented by a single layer of cells that rests on the basal lamina. It contains the stem cells from which new cells, the keratinocytes, arise by mitotic division. For this reason, the stratum basale is also called the stratum germinativum.

The basal cells also contain various amounts of melanin in their cytoplasm that is transferred from neighboring melanocytes interspersed in this layer. Basal cells exhibit extensive cell junctions; they are connected to each other and to keratinocytes by desmosomes and to the underlying basal lamina by hemidesmosomes.

As new keratinocytes arise in this layer by mitotic division, they move into the next layer, thus beginning their process of upward migration. This process terminates when the cell becomes a mature keratinized cell, which is eventually sloughed off at the skin surface.

stratum spinosum

The stratum spinosum is at least several cells thick. Keratinocytes in this layer are larger than those of the stratum basale. They exhibit numerous cytoplasmic processes or spines, which gives this layer its name. The processes are attached to similar processes of adjacent cells by desmosomes.

Because of their appearance, the cells that constitute this layer are often referred to as prickle cells. As the cells mature and move to the surface, they increase in size and become flattened in a plane parallel to the surface. This arrangement is particularly notable in the most superficial spinous cells, where the nuclei also become elongate instead of ovoid, matching the acquired squamous shape of the cells.

Stratum granulosum

The stratum granulosum is the most superficial layer of the non-keratinized portion of the epidermis. This layer varies from one to three cells thick. Keratinocytes in this layer contain numerous keratohyalin granules, hence the name of the layer.

These granules contain cystine-rich and histidine-rich proteins, which are the precursors of the protein filaggrin, which aggregates the keratin filaments present within the cornified cells of the stratum corneum. Keratohyalin granules are irregular in shape and variable in size.

Stratum corneum

Usually, an abrupt transition occurs between the nucleated cells of the stratum granulosum and the flattened, desiccated, anucleate cells of the stratum corneum. The cells in the stratum corneum are the most differentiated cells in the skin. They lose their nucleus and cytoplasmic organelles and become filled almost entirely with keratin filaments. The thick plasma membrane of these cornified, keratinized cells is coated from the outside, in the deeper portion of this layer, with an extracellular layer of lipids that form the major constituent of the water barrier in the epidermis.

The stratum corneum is the layer that varies most in thickness, being thickest in thick skin. The thickness of this layer constitutes the principal difference between the epidermis of thick and thin skin. This cornified layer will become even thicker at sites subjected to unusual amounts of friction, as in the formation of calluses on the palms of the hand and on the fingertips.

The **stratum lucidum**, considered a subdivision of the stratum corneum by some histologists, is normally only well seen in thick skin. The nucleus and cytoplasmic organelles become disrupted and disappear as the cell gradually fills with keratin.

Dermis

Attachment of epidermis to dermis is enhanced by an increased interface between the two tissues.

Sections of skin cut perpendicular to the surface reveal numerous fingerlike connective tissue protrusions, dermal papillae, which project into the undersurface of the epidermis. The papillae are complemented by what appear to be similar epidermal protrusions, called epidermal ridges or rete ridges, that project into the dermis.

At sites where increased mechanical stress is placed on the skin, the epidermal ridges are much deeper (the epithelium is thicker), and the dermal papillae are much longer and more closely spaced, creating a more extensive interface between the dermis and epidermis. This phenomenon is particularly well demonstrated in histologic sections that show both palmar and dorsal surfaces of the hand, as in a section of a finger.

True dermal ridges

Dermal ridges tend to have a parallel arrangement, with the dermal papillae located between them. These ridges form a distinctive pattern that is genetically unique to each individual and is reflected in the appearance of epidermal grooves and ridges on the surface of the skin. These patterns are the basis of the science of dermatoglyphics, or fingerprint and footprint identification. The dermal ridges and papillae are most prominent in the thick skin of the palmar and plantar surfaces.

Hemidesmosomes

When studied with the transmission electron microscope (TEM), the basal surface of the basal epidermal cells exhibits a pattern of irregular cytoplasmic protrusions that increase the attachment surface between the epithelial cell and its subjacent basal lamina. A series of hemidesmosomes link the intermediate filaments of the cytoskeleton into the basal lamina. In addition, focal adhesions that anchor actin filaments into the basal lamina are also present.

Layers of Dermis

Examination of the full thickness of the dermis at the light microscope level reveals two structurally distinct layers.

- The papillary layer, the more superficial layer, consists of loose connective tissue immediately beneath the epidermis. The collagen fibers located in this part of the dermis are not as thick as those in the deeper portion. This delicate collagen network contains predominately type I and type III collagen molecules. Similarly, the elastic fibers are threadlike and form an irregular network. The papillary layer is relatively thin and includes the substance of the dermal papillae and dermal ridges. It contains blood vessels that serve but do not enter the epidermis. It also contains nerve processes that either terminate in the dermis or penetrate the basal lamina to enter the epithelial compartment. Because the blood vessels and sensory nerve endings are concentrated in this layer, they are particularly apparent in the dermal papillae.
- The reticular layer lies deep to the papillary layer. Although its thickness varies in different parts of the body, it is always considerably thicker and less cellular than the papillary layer. It is characterized by thick, irregular bundles of mostly type I collagen and by coarser elastic fibers.

The collagen and elastic fibers are not randomly oriented but form regular lines of tension in the skin called Langer's lines. Skin incisions made parallel to Langer's lines heal with the least scarring.

In the skin of the areolae, penis, scrotum, and perineum, smooth muscle cells form a loose plexus in the deepest parts of the reticular layer. This arrangement accounts for the puckering of the skin at these sites, particularly in erectile organs.

Deep to the reticular layer is a layer of adipose tissue, the panniculus adiposus, which varies in thickness. This layer serves as a major energy storage site and also provides insulation. It is particularly thick in individuals who live in cold climates. This layer and its associated loose connective tissue constitute the hypodermis or subcutaneous fascia.

Individual smooth muscle cells or small bundles of smooth muscle cells that originate in this layer form the arrector pili muscles that connect the deep part of hair follicles to the more superficial dermis. Contraction of these muscles in humans produces the erection of hairs and puckering of skin called "goose flesh." In other animals, the erection of hairs serves in both thermal regulation and fright reactions.

A thin layer of striated muscle, the panniculus carnosus, lies deep to the subcutaneous fascia in many animals. Although largely vestigial in humans, it remains well defined in the skin of the neck, face, and scalp, where it constitutes the platysma muscle and the other muscles of facial expression.

The skin is the largest organ of the body. It consists of the epidermis and the dermis. The epidermis is the outer cellular layer of stratified squamous epithelium, which is avascular. Most superficial is the keratinized dead layer of cells, the stratum corneum, which varies greatly in thickness from one part of the body to another. That on the soles and palms is the thickest, while that on protected areas such as the scrotum and eyelids measures only a fraction of a millimetre. This has forensic relevance in the amount of injury that is needed to penetrate the skin and allow bleeding from the underlying tissues.

The epidermis is generally corrugated, the under surface by papillae that dip into the dermis. The degree of undulation also varies greatly from place to place, the thinner skin tending to have a flatter junction between dermis and epidermis.

The dermis or corium, consists of mixed connective tissue carrying the skin adnexae, such as hair follicles, sweat glands and sebaceous glands. It has a rich network of blood vessels, nerves and lymphatics, and has numerous nerve endings of various types for tactile, pressure and heat sensing. The lower zone of the dermis has adipose tissue and – depending on the site in the body – deep fascia, fatty tissue and muscle will form strata below the skin itself.

The skin functions as a mechanical and permeability barrier and as a sensory and thermoregulatory organ. It also can initiate primary immune responses.

What is injury?

Medically: ‘‘Injury’’ refers to any damage to the body produced by energy exchanges that have relatively sudden discernible effects. Though diseases also cause damage to the body, it is not termed as injury because the injury produced by disease lacks suddenness.

Legally: Injury is defined as any harm caused illegally to a person i.e. to his body, mind, reputation or property.

Injury can be classified in the following ways:

1) **Kinetic injury also known as Mechanical Injury:**
   - **a) Blunt force injury**
     - i) Abrasion
     - ii) Contusion
     - iii) Laceration
     - iv) Fracture and dislocation
   - **b) Sharp force injury**
     - i) Incised wound
     - ii) Stab wound
     - iii) Chop wound
     - iv) Cut fracture
   - **c) Firearm injury**
   - **d) Blast injury**
2) **Non kinetic injury (Non-mechanical injury)**
   - **a) Thermal injuries**
     - i) Heat
     - ii) Cold
   - **b) Chemical injuries**
   - **c) Electrical injuries**
     - i) Low voltage
     - ii) High voltage
   - **d) Radiation**
3) Legally
   - a) Simple injury
   - b) Grievous injury

4) Ante-mortem and Post-mortem injuries

5) Fatal and non-fatal injuries

Kinetic injury (Mechanical Injury)

Body can withstand remarkable amount of force due to its elastic nature of the soft tissue or the rigid strength of the bone. Body gets injured only when the force exceeds the capability of the tissues to adapt or resist.

The intensity of the force obeys the law of physics. Force varies directly to the mass and square of velocity of impacting object (Force=1/2 mass x Velocity^2). The weapon can be anything including the body itself. The effect of mechanical force can cause compression, traction, torsion, tangential and leverage stresses. The resultant damage depends upon the type of the weapon and the nature of the target tissue. For example violent compression due to explosion may do little harm to muscle but may rupture lung and intestine, while torsion may leave adipose tissue unaffected, yet cause a spiral fracture in a femur. Similarly, minimum amount of force is enough to pierce the body with a sharp knife but the same amount of force may not be enough to do any harm with a cricket bat.

Blunt force injuries

Blunt force injuries are one of the commonest injuries encountered in medical practice. Road traffic accidents, physical assaults and falls are the major circumstances of injury infliction.

Abrasion (scratches, grazes)

It is the most superficial type of injury which affects only epidermis of the skin. It is usually caused by tangential glancing impacts but vertically downward force on to the skin can also cause abrasion e.g. ligature mark of hanging. Usually abrasion does not bleed but because of the folded nature of the junction between the dermis and epidermis, it often bleeds slightly.

Types of abrasion:

a) **Scratches**
b) **Grazes**
c) **Pressure abrasions**
d) **Impact abrasions**

Scratches:

Scratches are very important in medicolegal cases because nail scratches, which may be semicircular or linear, over the neck may suggest strangulation. Likewise, scratches around the inner thighs and genitals may suggest possibility of sexual assault. Scratches over the fore-arms and legs may suggest resistance offered by the victim.

Grazes:

Grazes are common in Road traffic accidents where the skin skids over the road. Grazes can also be seen if the body has been dragged upon some rough surfaces.

Pressure abrasion:

It is due to the sustained direct pressure over the skin as seen in case of ligature mark of/ hanging and strangulation. The ligature mark of hanging tends to dry up and appear parchmentized. Parchmentization is less common in case of ligature strangulation. Pressure abrasions when parchmentized, tends to appear brownish and hard in comparison to the surrounding skin. Sometimes few blister filled with serous fluid may also be seen along the edge of the ligature mark which are termed as rope burns. Rope burns appear due to frictional force against the skin. Ligature binding of limbs impart imprint abrasion which may suggest use of restraint.

Impact abrasion:

Impact abrasions are due to direct impact of some object on skin which on crushing the cuticle casts a reproduction of the shape of the object e.g. radiator grill marks, tyre thread mark, muzzle mark in contact gunshot injury, bite mark etc. Usually, intradermal hemorrhage also occur simultaneously which gives even clearer imprint mark.

Though the exact time of age cannot be assessed, an approximate time range since injury can be estimated by noting the following changes in abrasion wound:

a) Bright red lesion – freshly produced
b) Blood and lymph which dries up and forms scab – 12 to 24 hours
c) Scab turns brownish – 2 to 3 days
d) Scab dries, shrinks and falls off – 7 days.

The direction of the application of the force can often be identified by inspecting the abraded wound with a hand lens if necessary. The torn fragments of the epidermis are pushed towards the end of the abrasion along the direction of application of the force.

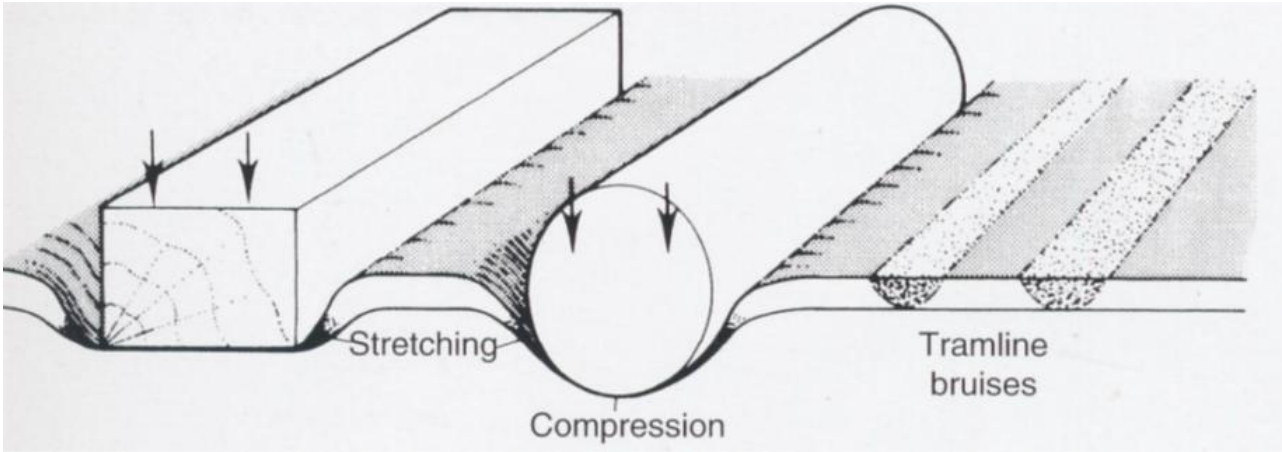
Note:

1) During postmortem examination, abrasion may look parchmentised, brownish in color and thinned out due to drying. Abrasion may be more evident after some time due to drying.
2) Ante-mortem abrasion has red base whereas postmortem abrasions has pale base. Abrasion might occur on the dead body while transporting which should be separated form ante-mortem abrasion.
3) Nappy rash in kids might look like abrasion giving false impression of child abuse. Nappy rash can be easily differentiated from true abrasio**No table of contents entries found.**n by it site of distribution of the former.
4) Abrasions caused by ants and cockroaches feeding on the epidermis of dead body may look like abrasion injury. Such abrasions due to ants and cockroaches can be easily differentiated from actual abrasion injury by their appearance where the former abrasions will be devoid of definite pattern and the margins will be irregular. Such 1abrasions by ants and cockroaches are often found in clusters.

Contusions (bruise)

Contusion is the bluish discoloration of the skin due to blunt force, which results in rupture of the small blood vessels, and leakage of blood from the site of rupture into the surrounding tissue, without puncturing the skin. Sometime the blood may move into the surrounding tissue along the facial planes and the contusion may appear away from the actual site of impact e.g. black eye. Black eye can be produced due to direct blow to the orbit as well as due to blunt trauma to the forehead. This type of contusion which appear away from the actual site of impact is also known as "*ectopic bruise*". Such ectopic bruise can also be seen around the knees in case of blunt impact around the thighs.

Contusion does not exactly represent the size and shape of the causative object because the blood leaks in wider surrounding areas and also the visible contusion may not be the actual site of impact as mentioned earlier. But an *intradermal contusion* is an exception where the contusion assumes the shape of the object because the leakage of the blood is small and is confined between the epidermis and upper strata of the dermis, e.g. tram line contusion due to cylindrical objects. A tram line contusion is caused by a moderate force impact with a cylindrical linear object on the skin. The major stretching and shearing of the skin occur at the edges of contact and not directly beneath the center of the object. This leads to intradermal bleeding at the edge and appearance of tram line contusion.



Mechanism of formation of tramline contusion.

Multiple small circular bruise around the neck might suggest throttling. Similar multiple small bruise elsewhere on the body might suggest grabbing. Elliptical ecchymosis over the neck, chest, thigh etc. may suggest possibility of sucking as seen in case of sexual assault.

Contusion is not only confined to the skin but can also occur in the deeper tissues, including muscles and internal organs as a result of rupture of blood vessels due to blunt force.

The age of the contusion can be documented depending upon the color changes that occur during the process of healing. The fresh contusion will be red to blue in color and will remain as it is for 2 days and gradually will change to purple, green, yellow and then to normal skin color. However, it should be remembered that the speed of these changes is variable and it is not always possible to use the colour changes as a 'clock' or 'timetable' of the bruise. Even in one individual, two bruises inflicted at the same time may differ in their appearances during resolution. The size of the bruise, the age of the individual (in older people bruises resolve more slowly), the presence of disease and many drugs may affect both the formation and the resolution of a bruise. As a very broad rule of thumb, a small bruises in a fit young adult will resolve in about 1 week. Research showed that if green color is identified the bruise is less than 18 hours old and if yellow color is identified, the bruise was over 18 hours old, whereas if no yellow color could be seen, the bruise could not be reliably aged. Again, it is essential to remember that the skin from Asians, Africans and blacks has a layer of melanin pigment of varying intensity which will reduce the visibility of a bruise and will mask the colour changes that occur.

Note:

1) **Delayed appearance of the bruise:** Sometime the bruise may not appear immediately after sustaining blunt injuries especially over the abdominal area. The extravagated blood in the internal organs may take one to two days to appear on the surface of the body which is also known as coming out bruise. On the other hand, Contusion may become more prominent or new contusion may surface later after death. This is because of well-known postmortem phenomenon of bruise becoming more prominent after death. The delayed appearance of contusion after death is mainly due to the hemolysed red blood cell which are more potent at staining the tissue compared to intact red blood cell. If this postmortem phenomenon of bruise becoming more prominent after death is not taken into consideration then there might be a dispute later if second autopsy is done.

2) **Contusion may not be visible over the soles and hands**. Because the skin over these areas are thick which may block the appearance of extravasation of blood. Sometimes, diffuse contusions over the back of the body may not be evident through the skin. Hence in custodial deaths, 4 to 5 parallel longitudinal incisions over the skin and underlying muscles of the back is advisable. This is very important to see the contusion at the back to rule out cause of death as blunt force. Diffuse blunt force injuries to the back have potential to cause death.
3) **Fabricated contusion (False bruise):** Sometime a person may fabricate contusion with help of local application of chemical (e.g. Semicarpus anacardium also known as Marking nut) on the skin. But such contusion can be easily differentiated from the true contusion as fabricated contusion often has multiple blisters over the contused area due to irritation caused by chemicals.
4) **Coup and Contre coup contusions:** Coup and contrecoup contusions of brain can occur individually or together. When a moving object impacts the stationary head, coup contusions are typical, while contrecoup contusions are produced when the moving head strikes a stationary object. Coup contusions are seen at the site of impact whereas contrecoup contusions are seen at 180 degree opposite of the coup contusion. Contrecoup contusion are formed when brain bounces up and strike the rough boney surface of the cranial cavity, when moving head strikes stationary object. E.g. if a man falls on his back and back of his head strikes the ground, at the site of impact i.e. at the occipital region of the brain coup contusion will be seen and contrecoup contusion will be seen at just 180 degree opposite of the occipital lobe i.e. at poles of the frontal lobes and temporal lobes.
5) **Postmortem collection of blood between the esophagus and cervical spine** is often seen during dissection of the neck, which can mimics contusion and might have wrong impression of death due to strangulation. This phenomenon is well described by Gordon and Prinsloo which is often known as Gordon and Prinsloo artifact. Hence, head and chest cavity should be open first to let the blood drain out before proceeding for special layer by layer dissection of the neck in case of death due to fatal pressure over the neck.
6) When in doubt, whether the suspicious injury is contusion or not, the alleged contused area should be incised and washed during autopsy examination. If it resist washing with plain water then it's a contusion. True contusion resist washing with water because true contusion

is an ante-mortem enzymatic process. Postmortem hypostasis (settling of blood at the most dependent part of the body after death) is often confused as contusion by a layman person.

7) **Extreme caution should be exercised while interpreting contusion in decomposed body.** Decomposing muscles may be dark brown in color which may mimicking contusion. Also, real contusion in decomposing body may look like decomposing muscle. Hence contusion looking lesion in decomposed body should be interpreted with great caution. Moreover, even microscopic examination of contused tissue after decomposition is of no help.

Laceration (tear)

A laceration is a disruption of the tissue due to stretching or crushing as a result of blunt force. Sometimes a split laceration (incised looking laceration) may look like incised wound over the boney prominences. Such split laceration occur due to application of blunt force over the skin overlying the bone for e.g. head, knee etc. Here the underlying bone acts as an anvil and the skin gets split which may look like sharp force injury. However, close inspection of such split laceration, with help of magnifying glass, can reveal tissue bridges and torn vessels which distinguish it from incised wound. When excessive amount of pulling force is applied to the limbs of a person the limb may get avulsed or there may be multiple parallel lacerations with near avulsion. Such kind of avulsion with formation of skin flap may be seen in road traffic accidents where tyre of a heavy vehicle rolls over the skin of limbs. Fracture of the long bones also cause laceration when it pokes out through skin. If the edge of the sharp weapon is blunt then it has the potential to cause laceration. Lacerated injury often cause less bleeding in comparison to incised wound because blood vessel are not completely cut. Moreover, foreign materials such as rust, dirt and splinters might get lodged in the lacerated wound which must be carefully preserved for forensic examination if the weapon has not been recovered

Artefactual lacerations might be introduced during faulty autopsy technique. For example, the liver might be inadvertently poked with finger while retrieving it form the abdominal cavity. So to know whether the injury seen in the liver is ante-mortem or post-mortem one should look for the contusion surrounding the injury. If the injury is surrounded with contusion then the injury is likely to be ante-mortem or peri-mortem and not postmortem. Similar contusions should be looked for around the suspicious injury inside the body to identify them as ante-mortem injury because contusion is an ante-mortem phenomenon.

Fracture

Various types of fracture of the bone can occur due to application of blunt force. Some of the major fractures are mentioned below:

1) **Fracture of the vault of the skull**
   a) Fissured fracture: Linear fracture of the skull. Sometime, if there are multiple linear fractures on the skull bone, the sequence of fracture can be established by Puppe's rule. It states that the fracture line due to the second impact does not cross the fracture line of the first fracture.
   b) Depressed fracture: Here the fractured bone fragment is driven inward or in case of children the skull may simply get depressed which is also known as pond fracture.
   c) Comminuted fracture: Here the bone is fragmented into multiple pieces.
   d) Diastatic fracture: Here fracture occurs along the skull suture.
2) **Fracture of the base of the skull:** Fracture of anterior, middle, and posterior cranial fossa can occur due to blunt force commonly seen in road traffic accidents. Ring fracture i.e. fracture around the foramen magnum which occurs due to intrusion of cervical spine into the cranial cavity in case of fall from height and landing on feet or buttocks. Another important fracture is hinge fracture of base of skull where fracture line runs from side to side across the floor of the middle cranial fossa. This type of fracture is commonly seen in motorcycle accidents and hence commonly referred as "motor cyclist fracture".
3) **Fracture dislocation of joints:** Fracture dislocation of joints can also occur due to blunt force injury. These types of injury commonly occur in road traffic accidents. Sometime the fractured fragment may also protrude through the skin which is also known as open fracture.
4) **Fracture of long bones:** Fracture of long bones are also one of the important injuries seen in road traffic accident, physical assault and falls.
5) **Cut fracture of the bone:** This type of cut fracture is seen in chop wound where heavy sharp weapons is used. The bone is cut to the variable thickness corresponding to force applied.

Sharp force injuries

INCISED WOUND

An incised wound is a clean cut injury to the tissue by a sharp cutting object and appears spindle in shape. Incised wound has clean cut margins whose length is greater than its depth. It has head end and tail end. The head end is deeper and signifies the initiation of the wound whereas tail end is shallower and represent the direction of the drawing of the sharp object. The breadth of the wound should be taken in both approximating and not approximating the cut margins.

Hesitation cuts or tentative cuts are superficial cuts suggestive of suicidal motive which are usually inflicted over neck, wrist and arms.

Defense cuts are incised wounds suggestive of homicidal motive commonly present over the hand and arms of the victim while defending perpetrator with sharp weapon.

The wound should be measured twice. First without approximating the margins and then with approximating the margins.

Stab wound

Stab wound has depth greater than its length and breath. Size and shape of the wound greatly vary according to nature of weapon, site of the body injured and amount of force applied. Depth of the wound is not advisable to record by probing as probing may create false track. So the depth of the wound is recorded by careful layer by layer dissection of the wound to find out the layers of tissues pierced. If the knife has pierced tissues e.g. muscle then it is known as penetrating wound but if it has pierced through and through it is known as perforating wound. Likewise, if the knife has reached body cavity it is termed as penetrating wound but if it has pierced through and through the body cavity then it is termed as perforating wound.

In the stab wound due to single edged weapon, one end of the wound will be ragged and the other end will be clean. The clean end of the wound is due to the sharp edge of the weapon. Sometime the sharp edge may impart fish tail like appearance if slight alteration of the path occurs while withdrawing the weapon. Double edged weapon will cause clean top and bottom ends. Sometime hilt or guard of the knife may cause contusion around the entry wound. Sometime, the entry wound of the stab wound be may nonspecific if the knife is twisted while drawing out of the body.

Note: Non pointed object such as a rod can also cause stab wound.

Chop wound

It is caused by sharp heavy cutting weapon for e.g. Khukuri, Axe etc. which is characterized by deep tissue cut along with beveling of the tissue. Chop wound usually present with cut of the underlying bone.

Cut throat injuries: Cut throat injuries are produced by sharp edge weapons. This type of injury can be seen in both homicidal as well as suicidal cases. Suicidal cut throat injuries are characterized by superficial parallel hesitation cut marks around the major fatal incised wound. Hesitation cut marks are rare in homicidal cut throat injuries.

The direction of the drawing of the weapon over the neck can be known by tailing of the incised wound. Tailing is the distal portion of the incised wound which is inflicted due to drawing of the weapon on the skin. It is relatively shallower in comparison to the head end of the wound.

Fire arm injury

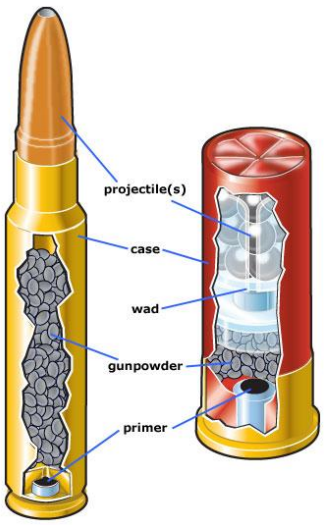
To understand fire arm injury it is necessary to understand the basic types of fire arms and their cartridges. Basically there are two types of fire arms:

1) Rifled firearms (Rifle, Pistol, Revolvers)
2) Smooth bore firearms (Shot guns)

Rifled firearms:

Rifled firearms are those firearms where spiral grooves (rifling) are cut inside a gun barrel that gives the bullet a spinning motion. These types of firearm use rifle cartridge as ammunition which has single bullet as a projectile. These bullets when fired carry a distinct mark (rifling marks) while travelling through the barrel and hence, when a bullet is found in the body it is advisable not to retrieve it with metallic forceps as the forceps might introduce new marks on the surface of the bullet which may impair the identification of the firearm from which the bullet was actually fired.

To understand the injury on the body due to rifled firearm one has to understand the rifle cartridge and its firing mechanism. A rifle cartridge consist of two segments namely cartridge case and a bullet. A cartridge case is an elongated metallic cylinder made up of cupronickel alloy which is closed at one side and a bullet tightly fitted at another side. The closed side, which is also known as base, accommodates the primer mixture (detonator). Gunpowder is placed in between the detonator and bullet within the cartridge case.



Ammunition of a rifle (rifled firearm) and a shot gun (smooth bore firearm).

When the gun is fired, the hammer of the gun strikes the base of the cartridge which ignites the detonator. The detonator intern ignites the gunpowder producing enormous amount of gas inside the cartridge which pushes the bullet out of the barrel. The cartridge either remains inside the gun which then is removed manually or gets ejected out automatically through another port in case of automatic or semiautomatic guns.

When gun is fired bullet, burnt and unburnt gun powder, shoot particles and fire are ejected out of the barrel. Hence depending upon the distance between firearm and target the entry wound will possess the substances that eject out from the muzzle of the gun as mentioned above.

Wound of entry:

Contact shot

Contact shot wound from a rifled firearm is usually circular in shape. But it may be stellate in shape at areas where skin overlay the bone (e.g. forehead). It is because of the propellant gas that splits the skin while escaping out backward through the wound of entry due to resistance offered by bone. Such phenomenon is not seen over the soft area e.g. abdomen because the propellant gas does not escapes out backward but instead intrudes inside due to lower resistance offered by the soft tissue, hence all the materials that eject out of the muzzle are present inside the wound. If the barrel is tightly held against the skin muzzle imprint can be seen around the entry wound whereas, abrasion collar, burnt hair and tissue may be seen if the barrel is loosely held against the skin.

Close range shot

At close range, say up to 20cm, there will be some smoke soiling, tattooing, powder burns, abrasion collar and burnt skin and hair which may be very variable and depend upon both the gun and the ammunition used. Tattooing is the deposition of fine and coarse gunpowder particles, which become truly impregnated into and mark the skin, and cannot be washed or scrubbed off. The shape of the entry wound can tell us the angle that the bullet made with that area of skin while entering. A circular hole indicates that the discharge was at right angles to the skin, whereas an oval hole indicates a more acute angle. Examination of the entry wound will show that the skin is inverted; the defect is commonly slightly smaller than the diameter of the missile due to the elasticity of the skin. An ‘abrasion collar’ or ‘abrasion rim’ around the hole is caused by the friction, heating and dirt effect of the missile when it indents the skin during penetration.

Intermediate range shot

At intermediate range, say from 20 cm to about one meter, there will be no smoke soiling around and no burnt hair and skin. Tattooing will be present and will be more dispersed and abrasion rim (collar) will be present.

Distant range shot

In distant range shot, say more than one meter, only the abrasion rim is evident. Smoke soiling, tattooing, burnt hair and skin are not seen because they do not travel beyond one meter.

Wound of exit:

The exit wound of a bullet is usually everted with split flaps, often resulting in a stellate appearance. There can be no burning, smoke or powder soiling. If the bullet has been distorted or fragmented or if it has fractured bone, the exit wound may be considerably larger and more irregular and those fragments of bullet or bone may be represented by multiple exit wounds. Where skin is fiappe supported, as by a belt, tight clothing or even leaning against a partition wall, the exit wound may be as small as the entrance and may fail to show the typical eversion. To increase the confusion, it may also show a rim of abrasion, although this is commonly broader than that of an entry wound.

Smooth bored firearms:

These type of firearms usually have long barrels (either double or single) with smooth inner surface. They use shot gun cartridge as ammunition which usually use multiple pellets (made up of Lead also known as shots) as projectile. When gun is fired multiple pellets eject out of the barrel which start to disperse after travelling for some distance. In some instances, instead of multiple pellets, a single shot known as slung is used.

A shot gun cartridge is about 4 cm long (can vary according to the weapon), cylindrical in shape and has diameter similar to that of the barrel of the gun. It is made up of plastic or a cardboard with one end closed with metallic cap and other end closed with a cardboard or a plastic lid. The metallic end houses the percussion cap and primer (detonator) above which gunpowder is placed. Above the gun powder wad (compressed paper or plastic disc which separates pellets from gunpowder) is placed and above the wad pellets are placed and is then closed with a paper or plastic disc. When gun is fired wad, pellets, some fire, some smoke and burnt and unburnt gun powder are ejected out of the barrel, but cartridge case remain insides the barrel or get ejected out from another port and not from the muzzle. Sometime the pellets may be packed in separate plastic container inside the cartridge which after ejecting out of the barrel get burst and impact around the wound of entry giving petal shaped contusion around the entry wound.

Wound of entry:

Contact shot:

In contact shot the wound of entry is usually circular and the margin is regular and often has a clean-cut appearance and muzzle imprint mark. But if entry wound is above the tissue supported by the underlying bone (skull, shin etc.) the margin of entry wound may tear and appear stellate shaped. It is due to the rapid expansion of the gas which cannot disperse as quickly as they would in soft areas such as the abdomen, and the greater ballooning of the skin results in splits of the skin, which often has a radial pattern. There may be smoke soiling of at least some of the margin of the wound. There may be a narrow circular rim of abrasion (abrasion collar) around the entry wound. The wads will usually be found along the wound track. The tissue around the wound tract will be blackened.

Near range shot:

In near shot range say from few cm to 20 cm, a similar entry wound resembling contact shot may be seen, but it will be devoid of muzzle imprint. Moreover, the entry wound will be surrounded with smoke soiling, burnt hair and skin. Powder tattooing of the skin around the wound of entry are also seen. These powder tattooing are due to the impregnation of the burnt and unburnt gun powder in the skin which cannot be washed off.

Intermediate range shot:

Intermediate range shot from 20 cm to 1 meter the wound of entry will be circular but there will be no smoke soiling, no burning of hair and skin. Tattooing of the surrounding skin and abrasion collar will be present.

Distant range shot:

Beyond 1 meter, the pellets usually start to disperse. The margin of wound of entry will first appear rat bitten in appearance (rat hole) and then a single large entry wound along with multiple satellite pellet entry wound may be seen around the main entry wound as the distance increases. Normally at a distance of 20 to 50 meter there is a uniform dispersion of pellets on the skin which are seldom fatal.

Exit wound:

Usually, shotguns rarely produce an exit wound when fired into the chest or abdomen, although single pellet exit wounds can occasionally be seen. Exit wounds can be seen when a shotgun is fired into the head, neck or mouth. The exit wound in these cases may be a huge ragged aperture, especially in the head, where the skull may virtually explode with the gas pressure from a contact wound, ejecting part or even all of the brain from the cranial cavity.

Note:

1. While interpreting the wound of entry and shot range, one has to make sure whether the bullet has pierced through the garments or not. In such cases smoke soiling and burning are usually present on the garment. The fabric of the garment may be found inside the wound of entry.

2. There may be multiple entry and multiple exit wounds with one bullet in case of perfect alignment of body parts.
3. The bullet entering the skull will cause beveling of the inner table of the skull signifying exit at the inner table. Likewise, beveling at outer table of the skull signifies exit wound.
4. The bullet after entering the body may strike the bone and the fractured bone spicules and bullet may create multiple exit sounds.
5. A bullet after entering the body cavity may pierce the gut and the bullet might be vomited out or pass with the stool.
6. Any projectiles, foreign bodies such as wads, bullets or shot should be carefully preserved for the police. The skin around the wounds may be swabbed for powder residues if this is considered to be necessary, but the retention of wounds themselves is no longer considered to be essential. Swabs of the hands of the victim should be taken.
7. The autopsy doctor must ensure that accurate drawings and measurements of the site, size and appearance of the wound are obtained and photographs are taken of each injury with an appropriate scale in view.

Blast injury:

Injuries in blast usually depend upon the type of bomb. In case of explosives used by military the release of high energy may result in death and disruption from blast effects over a wide area. In contrast, terrorist devices, unless they contain very large amounts of explosive, the pure blast effects are far more limited.

When an explosion occurs, the chemical interaction results in the generation of huge volumes of gas, which are further expanded by the great heat that is also generated. This sudden generation of gas causes a compression wave to sweep outwards from the epicenter. The pure blast effects can cause either physical fragmentation or disruption of the victim solely from the effects of the wave of high pressure and hot gases striking the body. There will also be pressure effects upon the viscera and these effects are far more damaging where there is an air/flThi interface, such as in the air passages, the lungs, the gut and eardrums. Ruptures and hemorrhage of these areas represent the classical blast lesion. This blast effect is usually low in lower- powered terrorist bombs. Injuries form projectiles and shrapnel ejecting out of the terrorist bomb constitute major cause of death and injury. Apart from blast injury secondary effect of blast also play major role in causing death and injury. These effects are:

- Burns – directly from the near effects of the explosion and secondarily from fires started by the bomb
- Missile injuries from parts of the bomb casing or shrapnel or from adjacent objects
- All types of injury due to collapse of structures caused by the explosion;
- Injuries and death from vehicular damage or destruction

  Examination of the dead following an explosion is essential, with careful documentation of the sites and sizes of the abrasions, bruises, lacerations, burns and any other injuries. If a near or massive explosion disrupts one or more bodies, identification of the fragments is essential. This task can be extremely difficult and resembles the response to a mass disaster such as an air crash in which many bodies may be severely disrupted and burnt. The autopsy doctor has an additional task to assist in the identification and recovery of material that might have formed part of the bomb.

Non kinetic injury (Non-mechanical injury)

Thermal injuries

Heat

Burns and scalds.

Burns can be classified by severity and extent. There are several systems of classification of the severity of burns but the most common one is as below:

- 1 first degree – erythema and blistering
- 2 second degree – burning of the whole thickness of the epidermis and exposure of the dermis;
- 3 third degree – destruction down to subdermal tis- sues, sometimes with carbonization and exposure of muscle and bone.

Wallace's Rule of Nine can be used to represent the percentage of burn area over the body. Which are depicted as below:

- Head and neck: 9 %
- Chest: 9 %
- Abdomen: 9 %
- Right upper limb: 9 %
- Left upper limb: 9 %
- Upper back: 9 %
- Lower Back; 9 %
- Anterior aspects of right lower limb: 9 %
- Posterior aspects of right lower limb: 9 %
- Anterior aspects of left lower limb: 9 %
- Posterior aspects of left lower limb: 9 %
- Perineum 1 %

Usually 30 to 50 % of the total body surface area burn is fatal but it may vary according to age e.g. elderly people may die even with only 20 % body surface area burn. The severity of the burn not only depends upon the body surface are but also site and depth of burn on the body. For example even smaller area burn over the nose and mouth may cause a person to suffocate and eventually die.

A person recovered from burnt house may not have died as a result of burn. Because fire is one of the most common ways of concealing homicide, which should be kept in mind while performing autopsy examination. Soot in trachea, bronchus and terminal bronchioles and carbon monoxide in blood signifies that the victim was still alive when fire started. But the converse is often not true. Absence of soot in the airways and absence of carbon monoxide in the blood might be observed in case of rapid conflagration or where accelerants (petrol) were used where very less amount of soot and carbon monoxide are produced.

The actual cause of death from burns is complex and results from the interplay of many factors. In rapid deaths, the directly destructive effects of heat on the respiratory tract leading to asphyxia, the combined toxic effects of carbon monoxide, cyanide and the multitude of other noxious gases (oxides of nitrogen, phosgene etc.) that are inhaled, the release of toxic material from the extensive tissue destruction, and 'shock' due to pain all cause or contribute to death. Most of these factors do not apply to scalds, unless the victim falls into the hot liquid. Where death from burns or scalds is delayed, dehydration and electrolyte disturbances as a result of plasma loss from the damaged skin surfaces are early causes of death. Later, renal failure, toxaemia from substances absorbed from the burned area and infection of widespread burns may be responsible.

A careful search must be made for any ante-mortem injuries that may have caused or contributed to death. These injuries can be of almost any type, but strangulation and shooting appear to be most common in cases of homicide concealed by fire. It is always advisable to x-ray a burned body, especially if the fire damage is so extensive as to make examination of the skin surface impossible. However, not all injuries on burned bodies are sinister, and where skin has been subjected to severe burning or charring, it will often split. These splits can also be caused by post-mortem movement during recovery of the body when charred and brittle skin is moved. Another misinterpreted injury is the 'heat hematoma' inside the skull. This spurious 'extradural hematoma' lies between the bone and dura and is caused when severe heat has been applied to the scalp, resulting in expansion of the blood in the skull diplöe and the intracranial venous sinuses, which rupture, resulting in the formation of a collection of blood outside the meninges. The blood is brown and spongy, unlike a true hematoma. A simple presence of this type of collection of blood may mislead the unexperienced doctor into thinking that this is the result of a trauma. In true extradural hematoma

carboxyhemoglobin is absent but in heat hematoma carboxyhemoglobin. In true extradural hematoma carboxyhemoglobin is absent because it had developed before the fire started.

The general features of scalds are similar to those of burns, with erythema and blistering, but charring of the skin is absent. Ante-mortem scalds can be distinguished from postmortem blisters by presence of erythema and signs of inflammation in the former. The postmortem blisters usually contain decomposition gas or decomposition fluid.

Note:

When a dead body is found outside in the sun, the exposed body parts may develop sunburns often with blisters and slippage of epidermis which may mimic scalds.

Cold

Body temperature below 35.0 °C (95.0 °F) is hypothermia. Prolong exposure to cold may cause chill blains, frost bite, trench foot, paradoxical undressing or even death. Hypothermia below 28 °C is fatal. Paradoxical undressing is the phenomenon seen in severe hypothermic individual where he/she removes his or her clothing despite extreme cold. It is due to the fatigue of the vasoconstrictor muscles of the periphery which lead to gushing of warm blood to the surface of the body from the core resulting in false sense of warmth.

In a person dying of hypothermia the surface of the joint may be pinkish brown in color. It is due to the inability of the cold tissue to extract oxygen. But this sign is dubious if body is refrigerated because similar features are seen in such refrigerated body. Sluggishness of the blood flow may cause micro-infarct of the organs which is evident in the pancreatic tissue as hemorrhagic pancreatitis and surrounding fact necrosis.

Chemical injuries

Several chemical which are corrosive in nature may cause tissue damage. For example: Concentrated acid and alkalies.

Concentrated acid such as Sulfuric acid caused tissue damage by coagulative necrosis. The areas of contact become blackish brown formation of coagulum which prevents further penetration of acid. In contrast, alkali (Sodium hydroxide) causes liquefactive necrosis which cause extensive penetrating damage.

Electrical injuries

Injury and death from the passage of an electric current through the body are common in both industrial and domestic circumstances.

Usually, the entry point is a hand that touches an electrical appliance or live conductor, and the exit is to earth (or 'ground'), often via the other hand or the feet. In either case, the current will cross the thorax, the most dangerous area for a shock because of the risks of cardiac arrest or respiratory paralysis.

Usually in case of household current, when a live metal conductor is gripped by the hand, the muscles will go into spasm, which cannot be voluntarily released because the flexor muscles are stronger than the extensor which result in sustained 'hold on'. This 'hold on' effect is very dangerous as it may allow the circuit to be maintained for long enough to cause cardiac arrhythmia.

The focal electrical lesion (Joule burn) is usually a blister, which occurs when the conductor is in firm contact with the skin and which usually collapses soon after infliction, forming a raised rim with a concave center. The skin is pale, often white, and an areola of pallor (due to local vasoconstriction) is a characteristic feature. The blister may vary from a few millimeters to several centimeters. The skin often peels off the large blisters leaving a red base. The other type of electrical mark is a 'spark burn', where there is an air gap between metal and skin. Here, a central nodule of fused keratin, brown or yellow in colour, is surrounded by the typical areola of pale skin. Both types of lesion often lie adjacent to each other.

In high-voltage (High tension electrical grids) burns, multiple sparks may crackle onto the victim and cause large areas of damage, sometimes called ‘crocodile skin’ due to its appearance. High voltage may also cause extensive burns and charring if contact period is large.

Lightening deaths can be due to direct thunder strike on the body or it may be indirectly due to electric current surging through the wet ground to the body. A fern like pattern also known as Lichtenberg marks can be seen on the body. These marks are believed to be as a result of current spreading through the blood vessels. But these findings may not be found in all lightening deaths. Sometimes the cloths of the lightening victim may be tattered at multiple places giving false impression of physical assault.

Radiation

In general radiation exposure does not present with any immediate side effects unless exposure is severe. Severe exposure may present with burns, severe illness and death. Moreover, radiation may be a part of high energy bomb blasts.

Cranio-cerebral injury

Head injury is the most common region of the body to be injured. It is the most commonly targeted part of the body in cases of physical assault. In addition, severe injuries following falls, especially those requiring forensic investigation, tend to involve the head. This is because brain and its coverings are vulnerable to trauma that would rarely be lethal to other body parts.

Injury to the scalp

The usual range of abrasions, contusions and lacerations may be seen on the scalp. Lacerations over the scalp may look extremely similar to incised wound, as the underlying skull acts as an anvil against which the skin gets split. Closer observation of the wound, however, will reveal tissue bridges and crushed hair follicles, which are characteristic features of lacerated wound.

Injury to the scalp may be concealed by hair. Skull should be palpated for any swelling or injuries. If necessary, hair should be shaved with the help of a scalpel, for proper visualization.

Scalp injury tend to bleed profusely even after death. A postmortem injury to the head may cause considerable bleeding, especially if inflicted soon after death. These factors should be investigated and caution should be exercised when interpreting findings to answer questions about the ante mortem or postmortem nature of the wound or about the length of time of survival following the injury. There is no reliable way of resolving this difficulty and experience is the best teacher in this regard.

Head injuries, present over the vertex of the head should always raise suspicion regarding the manner of death. It is highly unusual for an individual unusual to fall upon the top of the head, even from a considerable height.

Accidental head injuries present over the vertex of can be occasionally seen in a backward fall that just happens to reach a vertical surface, such as a wall or piece of furniture. In such cases, there is usually grazed abrasion accompanying the laceration or contusion.

Injury to the face

Facial injuries are also found to be extremely common in physical assault and accidents. The boney prominences of the faces such and eyebrows, nose, chin and zygomatic prominence often strike first due to the protruded arrangement.

A blow to the orbit may cause laceration of eye brow and fracture of the orbit. This fracture of the orbit may cause black eye but one should also remember that black eye can also be as a result of ectopic contusions that percolated down from the scalp.

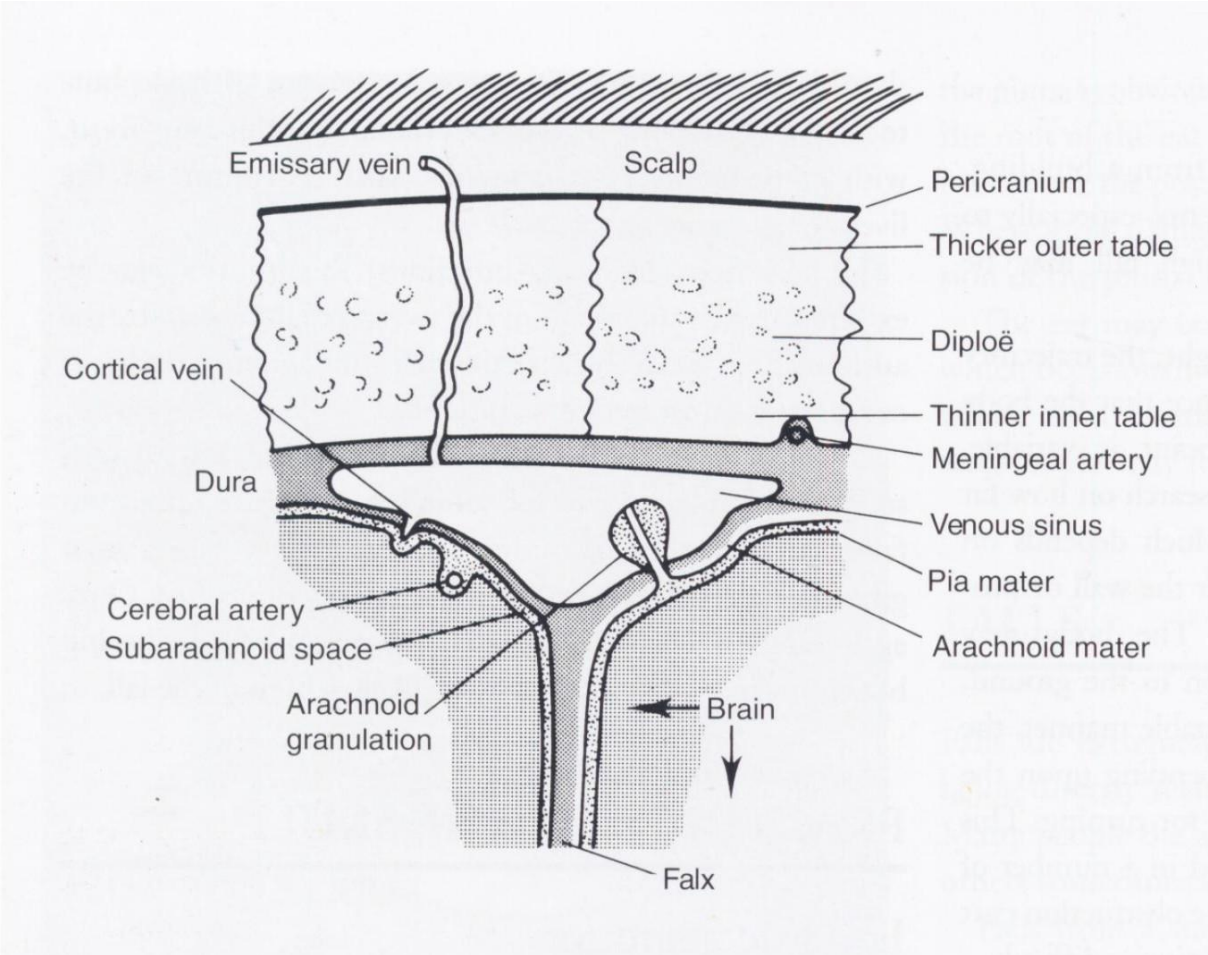
Nose is frequently seen to be severely injured, resulting in massive epistaxis. This is true even in cases with no nasal fractures. An unconscious person may die due to the negligence of health professionals in ensuring that the airways are protected from profuse posterior nasal bleeds that may pass back through the posterior nares into the throat and cause fatal airway obstruction.

The maxilla and mandible may be fractured by direct blows and ay also m cause dangerous intra-oral bleeding from associated soft tissue damage.

At autopsy the facial skeleton may be exposed by dissecting the whole facial skin upwards from the neck incision and reflecting it as far as the orbits, if necessary. Good restoration can be achieved as long as the skin is not penetrated.

Injuries to the mouth and lips are also extremely common in physical assault and is the diagnostic finding in cases of smothering. Bruised and lacerated lips along with tear of the frenulum should assist in suspecting the cause of death as smothering or due to foul play.

Forensic Anatomy of Skull



Forensic anatomy of the skull.

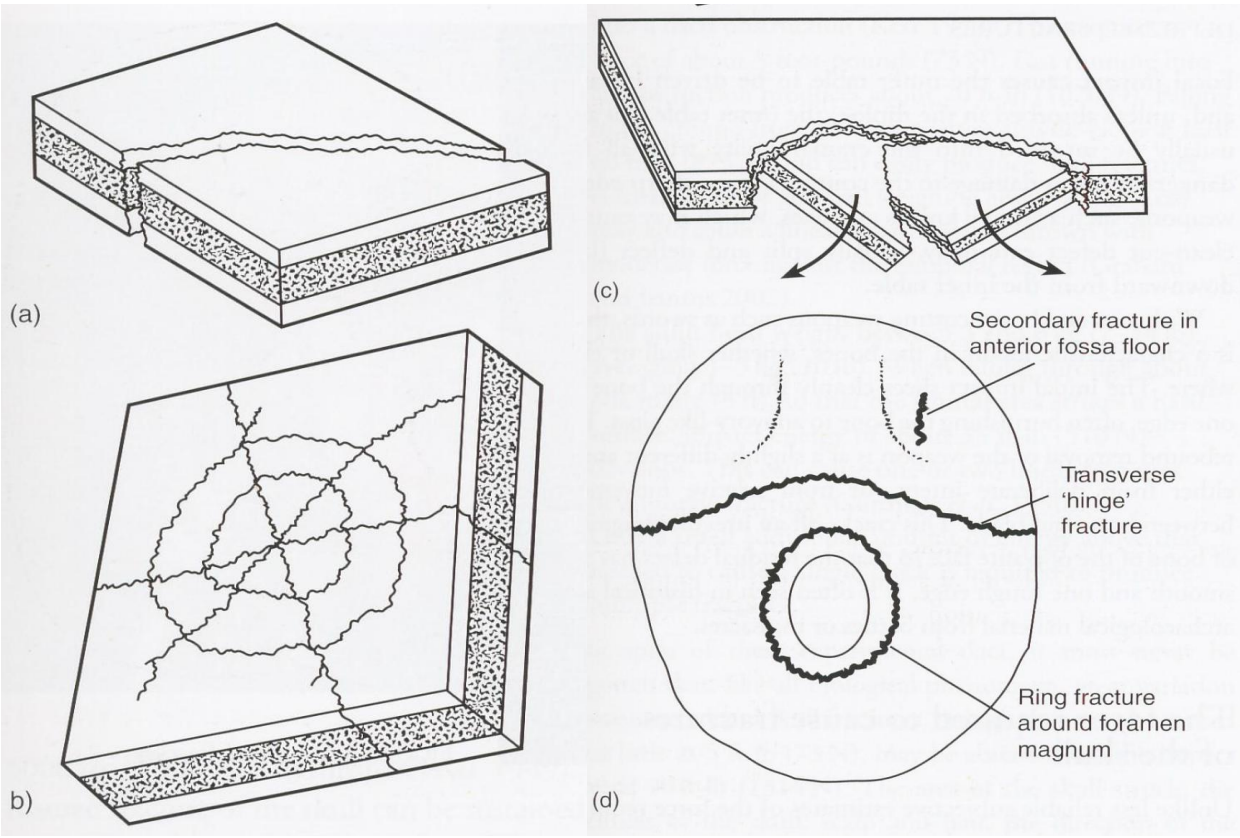
Adult human skull consists of two parallel layers of cortical bone surrounding compact bone in the areas surrounding the brain. The compact bone is interrupted at suture lines and vanishes where the bone becomes particularly thin, especially in the floor of the skull. The most vulnerable parts of the skull are in the parieto-temporal, lateral-frontal and lateral-occipital regions.

The presence of skull fracture is an indication of the severity of the force applied to the head. While it is not uncommon for a force sufficient to fracture the skull to also cause some intracranial injuries, even if it is only transient concussion, extreme forces to the head may not result in external injuries, including fractures but may show extensive intra-cranial injuries.

Types of skull fracture

Skull fractures can be classified as:

a) Linear fractures – straight or curved fracture lines, which either radiate out from a depressed fracture or arise separately, at distance from the site of impact
b) Ring fracture – fracture of posterior fossa, around the foramen magnum. most often caused by a fall from height, with impact on the feet, buttock or head, resulting in cranial invagination of the cervical spine
c) Pond fracture – shallow depressed fracture, forming a concave pond, often occurring in pliable infant skull bones, without fracture of the skull
d) Mosaic or spider-web fracture – comminuted fracture of skull, which forms a spider's web or mosaic pattern
e) Depressed fracture - comminuted fracture that is depressed inwards due to focal impact. Traumatic epilepsy has been show to occur as a late sequelae of depressed skull fractures
f) Diastatic fracture – separation of skull bones along the suture lines, commonly seen over the lambdoid suture, which fuses late, around 60 years



Types of skull fracture (a) linear fracture (b) spider's web fracture (c) depressed skull fracture (d) base fracture

Skull fracture is very susceptiple to infection and a person may survive from skull fracture but may later succumb to infection, of the meninges, or from development of brain abscess.

Force required to fracture the skull

Simulation experiments have shown that one can suffer a simple fissured fracture of the skull simply by walking into a fixed obstruction. This requires a force of about 5 foot–pounds (73 N).

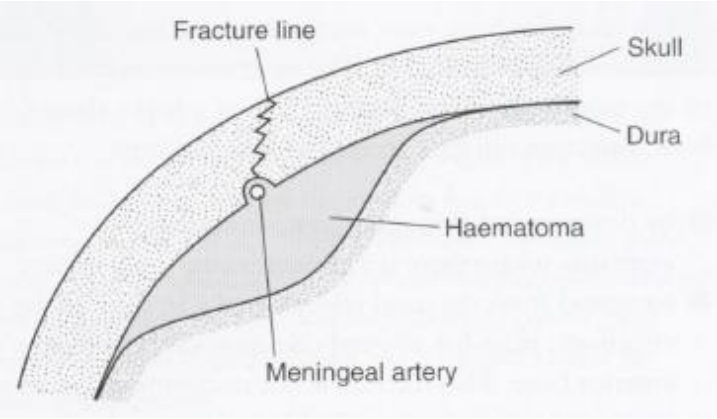
Running into an obstruction produces about 70 foot-pounds (1020 N) leading to multiple linear fractures. Falling from a sitting position on a concrete floor of 1 meter generates a force of about 35 foot-pounds (510 N). This is sufficient to cause one or two linear fractures on the skull.

A case has been reported, where an intoxicated person was lifted by his two fiends, by grabbing the hands and feet. The head of the person fell back and struck the floor, resulting in skull fracture.

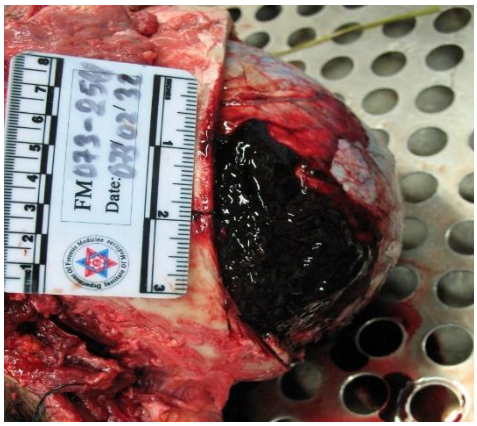
Intracranial Injuries

Trauma to the head may result in damage to the contents of the cranial cavity. Damage may occur either to the neural tissue or the rich vasculature that surrounds and penetrates these tissues. The content of the skull are the most fragile of all the vital organs which can get injured even in absence of fracture of the skull.

Extradural hemorrhages



Formation of extradural hemorrhage.



Left sided extradural hematoma

Rupture of posterior branch of middle meningeal artery which lies at the lateral aspect of the parieto temporal region of the skull is the commonest site of origin on extra-dural hemorrhage. The vessel ruptures as a result of fracture of squamous temporal bone on the lateral skull wall.

Leakage of the arterial blood strips off the underlying dura with progressive spurts and pooling in the space formed. It has been suggested that 35 – 100 ml of blood is needed for clinical signs to

appear and about 100 ml of blood is fatal. In rare cases, extradural hemorrhage may occur without fracture of the skull. In such instances the bleeding might have occurred from torn venous sinuses or from other smaller meningeal arteries.

Extradural hemorrhage is classically associated with “lucid interval”. The sign, however, it must be remembered, is not invariably present in all cases of extradural hemorrhages. Lucid interval is the temporary improvement in a patient's condition after which the condition deteriorates again, in a patient who had previously been unconscious following traumatic brain injury. In this period of temporary improvement, the patient may regain consciousness and feel better. However, the initial loss of consciousness was caused by cerebral concussion, which subsided, followed by a second loss of consciousness which could be due to increase intra cranial tension resulting from an expanding epidural hematoma. **A medical practitioner is liable for medical negligence, if he discharges a patient during the lucid interval and the patient later dies due to re-bleeding.**

Subdural hemorrhage

Subdural hemorrhage is much more common than extradural haemorrhage and is less often associated with a fractured skull. The lesion is traditionally classified into three types: acute, subacute and chronic. It is unhelpful to subdivide the acute type, however, and only acute and chronic haemorrhages need be considered.

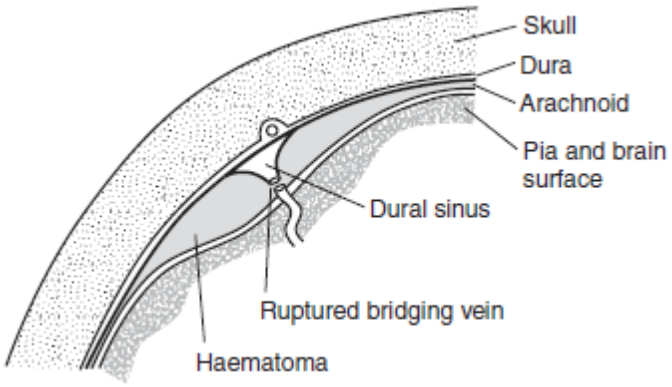
Subdural haemorrhage can occur at any age, but is common at the two extremes of life. It is one of the major causes of death in fatal child abuse. Caffey stated that the association of subdural haemorrhage with long bone fractures constituted the basis for diagnosis of child abuse.

While subdural hemorrhage is often associated with vigorous shaking of the baby, as seen in shaken baby syndrome, it is often argued that blunt injury, which might have gone unnoticed, might be the real cause.

Subdural hemorrhage is invariably associated with trauma and there is probably no such entity called ‘spontaneous subdural haematoma’. This is more commonly seen in conditions of vascular fragility, such as senility and bleeding diatheses, where minimal trauma, too trivial to be elicited in history taking, precipitates the bleeding.

Minor subdural bleeds, insufficient to give rise to any neurological or clinical symptoms or signs other than a transient headache, invariabley occur with the regular knocks of everyday life. Only when the bleeding causes cortical irritation or increases intra-cranial tension (35 - 100ml), does it become clinically apparent.

Subdural haemorrhages are frequently associated with both subarachnoid bleeding and with cerebral damage, making its individual contribution to symptomatology impossible to assess.



Formation of a subdural hemorrhage.

Acute subdural haemorrhage

The presence or absence of a fracture is immaterial, except as an indicator of severity of trauma to head. Subdural hemorrhage arise from from torn communicating veins (bridging veins) crossing the subdural space, between cortical vessels and dural sinuses. Subdural bleeding occur from shear stresses in the upper layers of the cerebrum, moving communicating veins laterally to cause rupture at their junctions with cortical veins or sinus surfaces. It is very rarely possible to identify the bleeding points.

Subdural bleeding is most often seen over the lateral cerebral surface, around the parasagittal area. As with most intracranial damage, the mechanical cause is a change of velocity of the head, either acceleration or deceleration, with relation to the velocity of the brain, and almost always associated with a rotational component.

Unlike epidural hemorrhage, subdural bleed need not be situated directly under the impact area nor be on the same side of the head. A subdural haemorrhage, unlike an epidural, is quite mobile.

Injuries originating high on the parietal area commonly drain down due to gravity and cover the entire hemisphere. The haemorrhage may remain fluid or may clot into a firm mass and both modes are commonly present. Again, as in any intracranial space occupying lesion, a minimum volume of about 35ml is required to cause neurological signs, though some writers prefer a larger volume, such as 100ml.

Lucid interval can also be seen in acute subdural hemorrhage however, the time taken for development is much longer. This is due to the venous origin of subdural hemorrhage, which takes longer to accumulate.

Chronic subdural hemorrhage

Chronic subdural hemorrhage may occasionally be seen as an incidental finding at autopsy in the elderly, where death was caused by some unrelated condition, with appearance changing with time. Recent lesions up to several weeks old are reddish-brown with a gelatinous membrane covering the surface. The contents are thick but liquid and may have areas of redder, more recent bleeding. An older haematoma, up to months or even a year old, is firmer, with a tough membrane around both surfaces, resembling a rubber hot water bottle filled with jelly or oil. The contents are liquid and may be brown or even straw coloured. Sometimes the interior may be much firmer and may have multiple colours due to the presence of varying stages and metabolites of hemoglobin. Also, the dura as well as brain matter is often stained brown or yellow. A small subdural lesion found at autopsy should not be used in isolation to determine the cause of death. In such situations, other findings should be explored elsewhere in the body.

There may be pressure changes visible on the surface of the brian, if the haematoma is large enough (more than 50–100ml). A chronic haematoma may become large and press down on the cerebral hemisphere sufficiently to dent and distort the surface. This may progress, to cause signs of hippocampal and cerebellar tonsillar herniation, and lead to all the attendant dangers to the vital centers in the brainstem.

The chronic haematoma arises from multiple episodes of the acute lesion, which, after an interval, become sheathed in a capsule of connective tissue. The haematoma may eventually be reabsorbed, it may remain dormant at the same size, or it may enlarge at a later date, due to re-bleeding.

Dating of a subdural haemorrhage

Estimation of age of subdural hematoma may have considerable forensic significance, especially if the lesion is significantly old, making association with the initial trauma that much more difficult. In addition, there may have been one or more episodes of trauma, any of which may have criminal or civil implications.

For example, an elderly man may survice for several days after bring struck on the head during a robbery. Autopsy may reveal only a large chronic subdural haematoma, which the defense may try to associate with the individuals's confused behavior over the previous year.

In accidents, the pre-existence of the subdural haematoma may cause unsteadiness that may have precipitated the accident. However, if this is missed at autopsy, it may confound the investigators as to the nature of cause of accident.

Unfortunately, in spite of several claims of reliable methods of dating subdural haematoma, such estimations are of doubtful value, partly because of the repetitive nature of bleeding, results in varying ages within the same haematoma.

In general, subdural haematoma gradually changes in colour from dark red to brown, first becoming apparent after 5 days and usually does not become obvious for 10–12 days. From gross appearances, brown colour changes occur between the first and second week, when a discrete surface membrane also becomes obvious. Crompton states that, the presence of a membrane firm enough to be picked with forceps makes the subdural haemorrhage at least 12 days old.

Inflammatory reaction to subdural bleeding begins within a few hours, resulting in cellular infiltration from the dural surface. A delicate 'neomembrane', made up of thin-walled capillaries and fibroblastic granulation tissue, grows from the periphery to cover the outer (dural) surface of the clot during the first few weeks. Around a month after the initial injury, a firm capsule develops, forming a cystic cavity containing dark brown, watery fluid. According to Munro, liquefaction of the contents does not occur before 3 weeks.

If no further enlargement occurs, this capsule becomes more and more fibrous, and rarely, completely absorb the haematoma by fusing with the outer capsule.

Some hematomas remain solid with an organised clot within, often with areas of fresher haemorrhage of different ages. When a substantial subdural bleed has occurred, the underlying cerebral cortex may be infarcted, caused by either a natural infarct having bled through the arachnoid into the adjacent subdural space or, more often due to the pressure of the haematoma on cortex and cortical blood vessels. Within the first couple of months large sinusoidal vessels appear in the newly formed connective tissue.

A number of later writers have pointed to marked variations in this chronological scheme. It should also be remembered that there is considerable personal variation in healing rates and also, because of the frequency of repeated subsequent fresh bleeding, attempts at estimating the date of the original bleed are shoulde attempted with caution.

Subarachnoid haemorrhage

Subarachnoid hemorrhage is more common than subdural haemorrhage and any injury resulting in cortical damage is invariably associated with sub-arachnoid haemorrhage. It has a mixed aetiology and whenever there is damage to the cortex, there will be some degree of subarachnoid bleeding. Traumatic subarachnoid bleeding may occur as a pure lesion where there is no cortical contusion, no neck injury, no deep brain lesion and no other membrane haemorrhage.

Subarachnoid bleeding frequently occurs as a result of natural disease, especially rupture of vascular malformations. When trauma is also present, the complex association of either the trauma precipitating the rupture or a rupture causing a fall, may need to be to be considered.

The mechanism of formation of subarachnoid hemorrhage varies greatly depending on the nature and extent of injury and as a consequence, so does the appearance. When secondary to laceration of the brain, its localization and severity depend upon the primary injury. Where it arises from a blunt impact, its position is not a good localizing sign as blood in the subarachnoid space mixes with cerebrospinal fluid, making it more difficult to clot and more mobie. Bleeding high over the cerebral hemispheres readily flows down to cover the entire brain. The sulci tend to collect more blood, especially in the insula.

Unlike with subdural bleeding, survivors rapidly dispose of the blood following pure subarachnoid hemorrhage. Haemolysis turns the cerebrospinal fluid a xanthrochromic yellow and gets reabsorbed within weeks. There may be residual brown or yellow staining of the pia or arachnoid.

Bleeding into the subarachnoid space is caused by shear stresses and rotational movements of the brain that rupture the bridging veins after they leave the cortex and before they penetrate the arachnoid. In addition, small cortical arteries may contribute some of the leakage as well.

When associated with cortical injury, bleeding will invariably come from cortical veins and small arteries directly into the subarachnoid space. It may be supplemented by intracerebral bleeding breaking out through the cortex.

While the role of subarachnoid hemorrhage in contributing to death in massive trans-meningeal injuries is doubted, its importance in cases with no other attributable cause cannot be ruled out. However, the presence of cerebral oedema and diffuse axonal injury should be excluded as far as possible.

Also, it importance in sequelae of natural disease, a ruptured aneurysm for example, is beyond reproach. Sudden death can occur from massive natural subarachnoid haemorrhage in the absence of any traumatic lesions.

Death can be remarkably rapid when a profuse haemorrhage occurs into the subarachnoid space. While the mechanism is not well understood, numberous cases of victims reporting of familiar signs of headache, neck stiffness, vomiting and progressive failure of consciousness, before dying suggest some role of meningeal irritation in these cases.

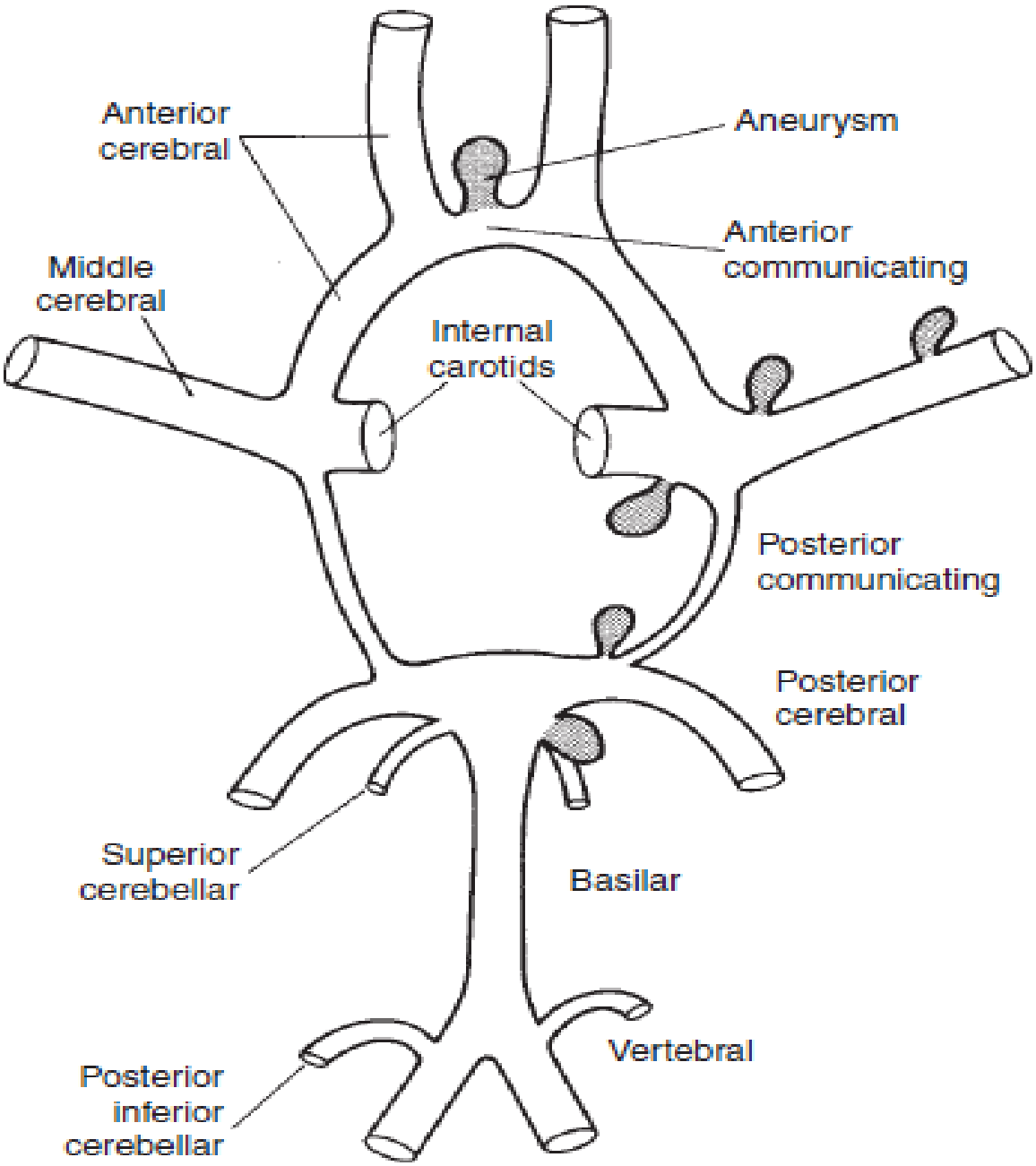
Death have also been reported where the individual just slump or collapses. In these instances, sudden intra-cerebral pressure increases tend to be the cause of death.

Ruptured berry aneurysm and trauma

Ruptured ‘berry’ aneurysm can cause a major medico-legal issues. A healthy young adult male found collapsed in the bathroom of his hostel would. However, the proper examination and detection of ruptured aneurysm(s) will go a long way in alleviating the misgivings in family members. This is further complicated when ruptured aneurysms are associated with an event of trauma.

The prosecution’s claim that the head injury mechanically ruptured the aneurysm or assisted in the event is not beyond absolute proof. Aneurysms of the circle of Willis are deeply located and lie protected under the mass of the brain. However, it would be hard to deny that a heavy blow to the

head, jaw or neck could rupture, split or weaken the fragile wall of a large, thin-walled aneurysm. This is further complicated by the elevated adrenaline levels, resulting in raised muscle tone, heart rate and blood pressure. As a result, it is more likely that raised internal blood pressure is a far probable reason, for rupture of a fragile aneurysm, than a blow on the head.



Common sites of berry aneurysm

Cerebral injuries

Cerebral injuries can themselves be fatal even in absence of skull fracture. A victim does not die of a simple fractured skull, but that the fracture is evidence only of a substantial head injury. This is only a 'symptom' or 'marker' for concomitant brain damage, which was the real lethal lesion.

The pathological and clinical manifestations of head injury may vary depending on the degree of force applied. A wide range of lesions may be caused by a variety of actions. The sequelae of clinical features is also extremely unpredictable – for example, severe head injuries have been known to be unassociated with concussion, while apparently insignificant damage has been followed by prolonged unconsciousness even ending in death.

Munro aptly states **'Any type of head injury can give rise to any type of intracranial damage.'**

The mechanism of brain damage

The brain may be injured in one of two ways:

- Direct intrusion, by a foreign object or fragments of skull
- Deformation of the brain in closed head injuries

The brain, being incompressible, is highly resistant to purely axial impacts. However, it is extremely rare for an impact to be completely devoid of any rotational movement and this is the primary culprit in causing damage in closed head injuries. Thus, it is possible to sustain severe and perhaps even fatal brain damage without an actual impact. It is the change in velocity, aceeleration or deceleration, with rotational component, that causes damage. It is of course obvious that the energy delivered by impact is far greater.

The majority of head injuries are seen in cases of traffic accidents or fall. In both these instances, there is a sudden deceleration of a moving head on contact with a fixed surface. In case of physical assault and combat injuries, the stationary head is accelerated by an external blow. The sudden change in velocity is transmitted to the brain, which can become damage against both the sharp edges and the flat surface of dura on violent movements.

Experiments have shown that there are marked pressure changes within the cranium on impact. Upon impact, pressure momentarily increases at the impact point but falls to a negative value diametrically opposite. This is good evidence supporting the theory of contrecoup damage being a vacuum effect.

The actual physical disruption of cerebral tissue is caused by one or more of three processes:

1) Compression of constituent units, by being forced together
2) Tension of units, pulling them apart
3) Sliding or shearing strains, moving adjacent layers of tissue laterally against each other

The area of the skull beneath an impact becomes momentarily depressed even if it does not fracture, causing compression. This is responsible for the typical cerebral contusions. At the same time, other areas of the skull must bulge outward to accommodate the deformation, this 'rarefaction' remote from the impact may cause tension damage. Laminar deformity or 'shear stress' caused by angular rotation of the head is perhaps the most important. The head is pivoted on the first cervical vertebra, and any impact on jaw, face or cranium will produce an angular momentum. Sudden deceleration of a moving head, causes deceleration of the skull while the momentum of the brain will cause it to continue in motion, again almost certainly with some rotatory component. This restraint will occur first – and with maximum effect – on the most superficial layers of the cortex. These in turn will drag on the next deepest layer and so on until the difference in velocity is equalized – but this will have been at the expense of laminar tearing of the cerebral tissue and its associated blood vessels.

In addition, the sharp edge of the tentorium and the lower edge of the falx may cause damge to the cerebrum, the corpus callosum and the brainstem. Impact against the side wall of the skull and against the falx may cause diffuse contusion of the cortex. The cerebellum being lighter and more tightly packed tends to suffer less damage

The configuration of cranial floor is partly responsible for the common places of localization of cerebral damage, at the tips and undersurface of the frontal and temporal lobes.

Cerebral contusion

Brain tissue may disrupt when either linear or, more often, laminar stresses are applied to the cortex. In the usual type of cortical contusion seen in a closed head injury, the cortex is blue or red from haemorrhage, though if survival has lasted for some time, there may be added discoloration from associated cortical infarction. The haemorrhage may be diffuse or may be punctate and is often a mottled purplish red when confined to the cortex. Extension into the underlying white matter tends to be pure red in fresh lesions. The lesion is often wedge-shaped, with the base on the surface, tapering away into the deeper layers.

Cerebral laceration

Laceration of the cortex is an extension in severity of mechanical separation of tissue. When relatively superficial, the cortex appears ‘red velvet’, becoming progressively more tattered as severity increases. The cerebral surface fissures, with fragments of cortex detaching and presence of deep lacerations, sometimes reaching even the deep ganglia or ventricles. In cerebral lacerations and possibly contusions, the pia mater and often arachnoid are torn, resulting in leakage of blood into the subarachnoid and even subdural spaces. The corpus callosum is commonly torn at its posterior end and may represent a guillotine effect of the free lower edge of the falx or differential lateral movement of the cerebral hemispheres.

Lacerations, like cerebral contusions are most often found in those areas of the brain where the cortex is most likely to come into contact with irregularities in the internal profile of the skull. The undersurface of temporal lobes and orbital surfaces of frontal lobes are most frequently injured.

Traumatic intracerebral haemorrhage

Substantial areas of haemorrhage are common in severe head injuries. Primary hemorrhages occur at the time of impact or soon afterwards; while secondary injuries are caused by changes in intracranial pressure or bleeding into infarcts caused by vascular damage.

Intracerebral hemorrhage may rupture into the ventricular system or through overlying cortex into the meningeal spaces.

Such haemorrhages sometimes occur in older subjects, with hypertension and cerebral atherosclerosis. When a scalp injury is present – and perhaps even a fractured skull – it may be difficult to decide if a head injury (such as a fall) was responsible for the cerebral haemorrhage, or whether a sudden ‘stroke’ caused by a natural cerebral haemorrhage resulted in the fall. The presence of left ventricular (cardiac) hypertrophy, a history of hypertension and the site of the (usually solitary) haemorrhage tends to point to a natural bleed. This is especially so if the large size of the lesion seems inconsistent with the degree of head injury sustained.

Hypertensive lesions tend to be predominant in the thalamus, external capsule, pons and cerebellum, and are more often occipital than frontal or temporal. However it may sometimes be absolutely confounding and impossible to differentiate the two conditions.

Brainstem haemorrhage

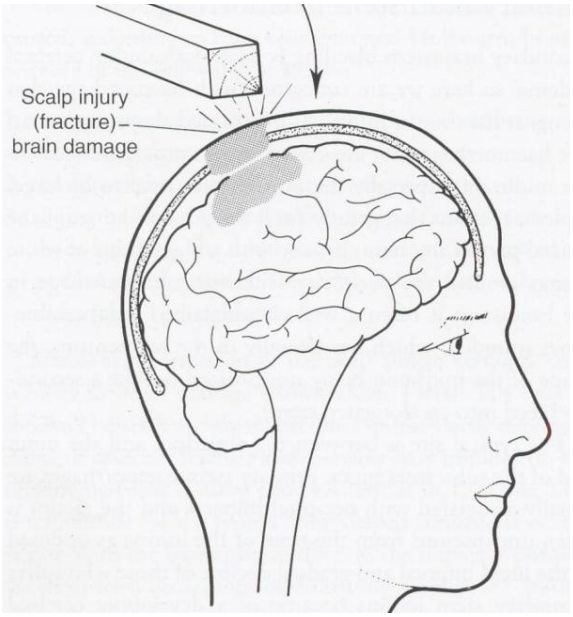
Hypertensive haemorrhages not associated with trauma can also occur in the midbrain, especially the pons. Spontaneous hypertensive hemorrhages are large, explosive lesions that cause the pons to swell, with disruption of central part of the stem and a ragged rim of white matter around the periphery.

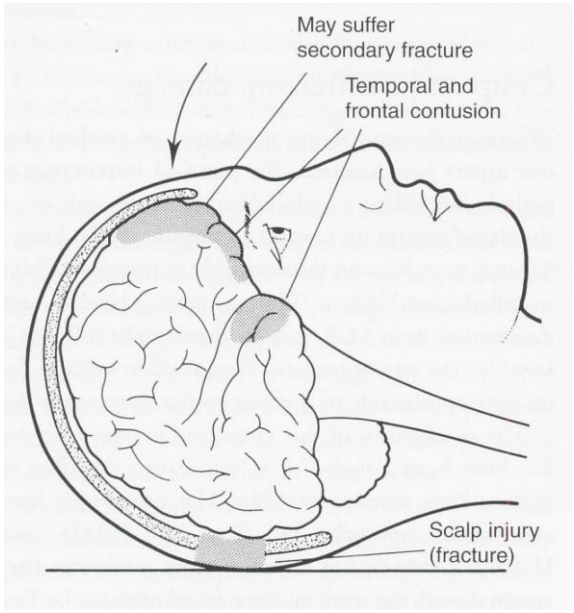
Traumatic haemorrhage in the brainstem is often a well-circumscribed lesion, sometimes rounded, which lies laterally in the tegmentum, the shape of the midbrain being undistorted. The typical site is between the aqueduct and the outer end of the substantia nigra.

Primary brainstem haemorrhages are usually associated with occipital impacts and the victim is often unconscious from the time of the injury, as opposed to the lucid interval and gradual decline of those who suffer secondary stem lesions because of a developing cerebral oedema or space-occupying meningeal haematoma.

Coup and contrecoup damage

When a mobile head is struck with an object, the site of maximum cortical contusion is most likely to be beneath or at least on the same side as the blow. This is the so-called ‘coup’ lesion. When a moving head is suddenly decelerated, as in a fall, though there might still be a ‘coup’ lesion at the site of impact, there is often cortical damage on the opposite side of the brain – the ‘contrecoup’ lesion.





Coup lesion to fixed head. **Contrecoup lesion to moving (decelerated) head.**

As already discussed, Yanagida et al. provided proof the deformity of the skull results in ‘rarefaction’ at a site away from the site of impact, causing ‘vacuum’ which acts along with tension forces to cause injury.

Note

- There may be no coup damage at all, only contrecoup.
- There need be no skull fractures, even in presence of severe coup and contrecoup lesions
- Contrecoup injury is most common in the the tips and undersurface of frontal and temporal lobes, and may rarely be symmetrical, in a fall on the occiput.
- Contrecoup lesions are likely to be diametrically opposite, on the contralateral surface of the brain, but exact geometrical correspondence may not necessarily be present.

- Contrecoup lesions on the occipital lobes are virtually unheard of, due to the anatomical configuration of the floor of the cranium, but the reasons are by no means understood.
- In temporal or parietal impact, contrecoup lesions may be on the opposite side of the ipsilateral hemisphere, from impact against the falx cerebri.
- Contrecoup lesions may be sufficiently severe to cause blood-filled cavitation in deep cortex and underlying white matter, especially in frontal lobes and tips of temporal lobes.
- Fall on the occiput may cause contrecoup lesions resulting in fracture of the thin bones of the floor of anterior fossa. Such cracks in the roofs of the orbits may allow meningeal haemorrhage to seep into the orbits and appear as 'black eyes'.
- Contrecoup lesions may be seen in fixed head, where shoring of the head may cause typical contrecoup lesions. Coup lesions are invariably present. Scalp may be free from injury.
- The interpretation of contrecoup lesions is most reliable in when examining contusions or lacerations. Meningeal haemorrhages, either subdural or subarachnoid, may also arise in association with contrecoup lesion, but cannot be relied on to determine the direction of impact.

Concussion

Concussion is a transient paralytic state following head injury which is instantaneous, with no evidence of structural cerebral injury and always followed by amnesia from the moment of injury.

Denny-Brown and Russell showed that the rate of change of velocity of the head was important in producing concussion, which rarely developed if the speed threshold was less than 8.5m/s (28ft/s).

Gross skull and brain damage have occurred with little or no apparent concussion, though concussion may be so transient that the subject may not even fall to the ground. Relatively minor head injuries have given rise to prolonged unconsciousness. True concussion may last for seconds or minutes. If prolonged unconsciousness extends into hours, days or longer, then there is likely to be some structural brain damage.

Occasionally what appears to be simple concussion proves to be fatal, causing respiratory paralysis, though at autopsy no significant lesions are found. Slight cerebral oedema and scattered non-specific petechial haemorrhages may be found.

Concussion has an association with rotational head movements. This is evident when fixation of the head before impact, decreases loss of consciousness after impact. The frequency of concussion in boxing contests proves the importance of shear stresses in causing neuronal damage.

Concussion may be followed by a 'post-concussion syndrome' characterized by headaches, unsteadiness and anxiety. Retrograde amnesia is almost inevitably associated with concussion, though, like concussion itself, it may be so transient as to escape notice. Commonly only of minutes' duration, it can extend to several days before the head injury. While there is often a recovery of much of the memory, the memory of events immediately before the incident rarely returns, which may be a protective mechanism. Concussion has been attributed to several causes, but the most acceptable hypothesis is the 'diffuse neuronal injury'.

Diffuse neuronal and axonal injury

Graham et al.describe four principal forms of diffuse brain injury:

- Diffuse vascular injury
- Diffuse axonal injury
- Hypoxic brain damage
- Diffuse brain swelling.

Diffuse vascular injury consists of multiple minute haemorrhages throughout the brain and the patients dies within 24h. Recent research have shown that traumatic axonal injury is much more common than previously realized also, diffuse axonal injury can be precipitated by processes other than head injury. Geddes et al. (2000) suggested new terminology:

- Axonal injury (AI) – a non-specific term referring to damage to axons due to any aetiology
- Traumatic axonal injury (TAI) – damage to axons caused by trauma, may vary from small foci of axons to diffuse brain damage
- Diffuse axonal damage (DAI) – clinicopathological syndrome of widespread axonal damage, with specific traumatic aetiology

Diffuse axonal injury is primarily a non-impact rotational acceleration–deceleration phenomenon, deformation by stretching probably being the most significant factor. A low level of injury causes transient changes in the permeability of the axolemma, gradually leading to ionic changes, accumulation of fluid and axonal swelling and eventually, with an increasing grade of injury, to intracellular Calcium accumulation, proteolysis and collapse of the cytoskeleton.

The disruption of axons leads to bulbous and clubbed 'retraction balls or globes' on the axons in the cerebral hemispheres, cerebellum and brainstem. Retraction globes are most numerous in the corpus callosum, the superior peduncles, the parasagittal white matter, the medial lemnisci and the corticospinal tracts. After some weeks in those who survive long enough, clusters of microglia, presumably part of a repair process, congregate at the site of ruptured axons.

Disturbance of axonal transport causes accumulation of substances in damaged fibres that can be demonstrated immunohistochemically. At present, β-amyloid precursor protein (β APP) is considered the most reliable indicator of axonal damage, revealing axonal injury within 2–3 hours of the insult, whereas it takes about 12–18 hours for axonal bulbs to become visible on routine or silver stains.

However, one should keep in mind that β APP is not specific to head injury but is an indicator of derangement of fast axonal transport, which has also been demonstrated in other pathological conditions. Similarly, retraction globes may also be seen around the periphery of natural lesions such as cerebral infarcts and haemorrhages and microglial clusters have been reported in viral/HIV encephalitis, previous global hypoxia and fat embolism.

Geddes et al. concluded **'the demonstration of traumatic axonal damage is likely to be of limited use in most forensic situations, except to confirm that there has been a head injury'.**

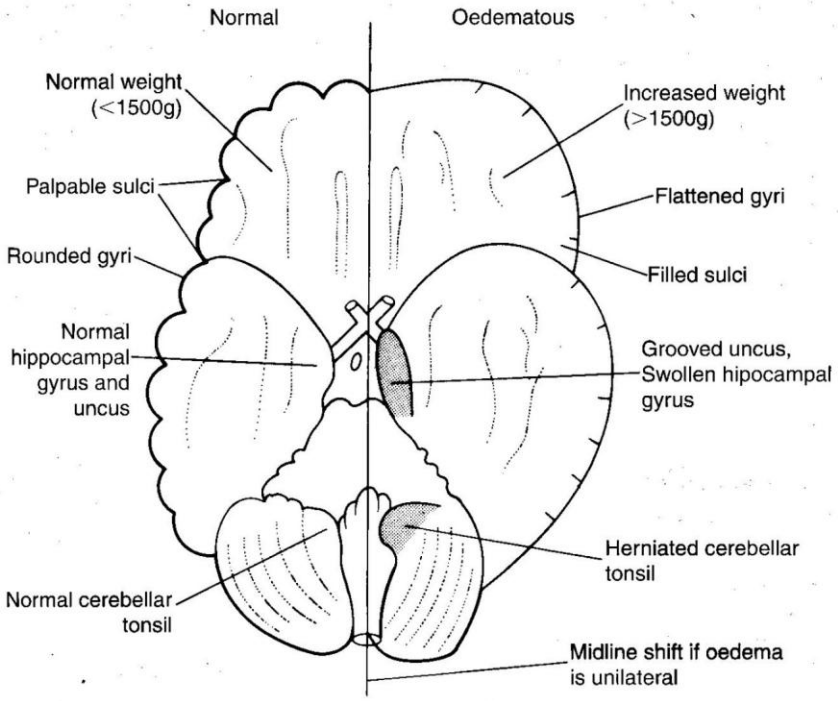
Cerebral oedema

Swelling of brain tissue may be a localized effected around any lesion, but we are concerned with generalized oedema of the brain. Swelling of the brain is extremely common after a substantial head injury, especially in children. Though commonly associated with other intracerebral damage, it can occur as the sole abnormality – and not infrequently – prove fatal, particularly in the young.

Cerebral Oedema is the most common cause of raised intracranial pressure, more common than than localized space-occupying lesions such as haematomas or tumours, though of course these often coexist with cerebral oedema. Oedema may well be related to diffuse neuronal injury and to concussion. Its cause is obscure, with fluid in the brain increasing total weight by at least 100 grams, mainly in the white matter. Electron microscopic studies have revealed an extracellular compartment, which is much wider in the white matter (up to 80nm) than in the grey matter (up to 20nm), which explains the preference of oedema for the white matter.

The autopsy features of cerebral oedema are readily recognized. Removal of the vault exposes tense, stretched dura. The brain is similarly turgid and bulges through dural incision. Gyri are pale and flattened, while sulci appear filled, smoothening the normally corrugated cerebral surface. The ventricles may be shrunken to slits by the swelling of the adjacent white matter.

Severe oedema causes the cerebral hemispheres to press down upon the tentorium and herniate through the opening. The hippocampal gyrus may press into the opening, causing uncal grooving. Similarly, cerebellar tonsils may be 'coned' into the foramen magnum. These pressure effects cause haemorrhage and necrosis at the sites of pressure. Normal anatomical grooving should be differentiated from tonsillar coning by associating with other signs of brain swelling.



Signs of Cerebral Oedema

Cerebral oedema may be the only intracranial abnormality found in children and, in the absence of any other demonstrable lesions, the cause of death has to be attributed to compression of the vital centres in the brainstem.

Cerebral oedema tend to be self-propagating, especially in children suffering from head injury, usually following road traffic accidents. It begins with oedema following brain trauma, the subsequent rise in intracranial pressure impairs the venous return from intracranial sinuses but is insufficient to restrict the arterial inflow, so further aggravating congestion and oedema. This leads to worsening cerebral hypoxia and oedema causing infarction and brain death.

In addition to mechanical damage, cerebral oedema can be caused or aggravated by hypoxia. Head injuries frequently associate with respiratory compromise, due to accompanying injury to thorax. Macroscopic evidence of brain swelling can be demonstrated at autopsy in cases where the interval between trauma (or onset of hypoxia) and death was less than one hour.

While consired severe and fatal in many instances, several methods are available to reverse oedema, including hyperventilation, which acts by providing full oxygenation and reducing the peripheral carbon dioxide tension, thus causing constriction of arterioles and a reduction in brain volume and transudation.

SPINAL INJURIES

The spine and head tend to be connected in more ways than one, for example, brainsteam neuronal chromatolysis has been associated with interruption of ascending fibres in the cervical cord. Spicer and Strich have shown association betweem spinal root ganglia haemorrhage and head injury. EEG changes have been shown to occur in victims with cervical spine whiplash injury.

The upper two cervical vertebrae provide most of the rotational movement of the head, whilst the lower neck allows for flexion and extension. Violent force applied to the head tends to damage those parts of the neck. Spinal damage are caused by

- **Compressional stress**
- **Hyperflexion and hyperextension stress.**

Compression damage

Fall on the head, feel or buttocks tend to be absorbed by the skull as well as other skeletal elements. However, when transmitted, it can cause impaction of the upper cervical spine on the base of the skull, resulting in a ‘ring fracture’. A vertical fall onto the head may cause the ‘burst atlas’, where impact of occipital condyles in an axial direction wedges the superior atlantal articulating facets apart and split the ring of the vertebra. The posterior arch can also be fractured in hyperextension by compression between the occiput and posterior spine of the axis. Compression fractures of vertebral bodies may occur, most commonly in the lower dorsal and upper lumbar zone, particularly T12 and L1. Spinal cord injury is infrequent compared with angulation injuries, unless posterior extrusion of a disc or backward displacement of fragments of a disrupted vertebral body predisposes to injury.

Hyperflexion and hyperextension injury

Hyperextension is much more dangerous than hyperflexion in causing spinal damage possibly because of the protection from the contraction of the strong posterior neck muscles, while the anterior longitudinal ligament is incapable of protecting the cervical spine during hyperextension.

Frontal or Rear collision accounting for 80% of all accidents, has a component of hyperflexion and hyperextension of the cervical neck. Death in these situations has been considerably decreased though implementation of policies on use of head restraints and seatbelts, restricting the range of movement.

During frontal collision, the subjects head swing down into hyperdlexion, and if unrestrained, will strike the fascia or windscreen of the car. The head will then rebound, causing hyperextension of the neck. The head flies into hyperextension first, in case of rear collision, under head rest is available. These accidents 'pile on', vehicles striking the one in front, resulting in deceleration hyperflexion.

Lesions are seen in cervical as well as in the thoracic and lumbar segments, and vary greatly resulting in hemorrhage in muscles, rupture of anterior longitudinal ligament, as well as tearing of intervertebral discs and annulus fibrosus. Nerve roots may be torn or compressed, and impingement of the spinal canal by vertebral fracture and dislocations. Compression, ischaemia and haemorrhage of the spinal cord are the most serious complications. The first two cervical vertebrae are commonly injured.

Hyperextension injuries tend to force the vertebral body forwards and impinge on the vertebral canal, possibly causing cord damage. Hyperextension can result in impingement of the cord by the the ligamentum flavum. Hyperflexion injuries result in dislocation of vertebra, causing it to tilt backwards, compromising the lumen of the spinal canal.

An anterior dislocation of the cervical vertebra can occur in a fall onto the back of the head, while unilateral dislocation is common when the head strikes the windscreen. Posterior dislocation may be caused by blows that jolt the head backwards with hyperextension of the neck. Gross injury to the spinal column. as in high-speed railway or road traffic accident, may result in transection of the cord due to the guillotine action of two displaced vertebral fragments. This is commonly seen in upper or mid-thoracic region, caused by direct impact or gross 'whiplash' effects. The thoracic aorta is commonly torn, where descending arch meets the spine, even without spinal fracture.

Damage to the cervical spine is rarely seen in suicidal hangings in which death is caused by compression of blood vessels or rarely, airway. Judicial hangings, with a long drop, cause fracture dislocation of the cervical spine, often with complete severance of the two fragments and transection of the spinal cord. Hanging with the knot of the suspension point beneath the chin may result in a violent hyperextension of the neck, that often fractures and dislocates the axis from third cervical vertebra. With a heavy person and a long drop, the head may be decapitated.

Spinal cord injury

Most spinal cord injuries arises from intrusion of part of spinal column into the canal. This may cause bleeding into the space outside the spinal dura, resulting in a space-occupying lesion in the canal compressing the cord, or within the dura, from either ruptured vessels or from haemorrhage in the cord itself.

Cord damage may also occur in the absence of any apparent intrusion into the canal, This is similar to injuries seen on the brain, in closed head injuries without a fracture. The cord may appear oedematous any obvious mechanical defect. Such injuries must be attributed to momentary collision of the cord against the canal or a transient deformity that did not exceed the threshold resulting in permanent damage. The damage in the cord may also extend for several segments above and below the point of impact. Central grey matter have a softer, more vascular organization and tend be favoured by haemorrhage than the white columns.

Infarction may occur in association with contusion, or due to damage to the local blood supply – often the anterior spinal artery. Cord may undergo liquefaction (‘myelomalacia’) over several days or longer, with progressive worsening of neurological symptoms.

CHEST AND ABDOMINAL INJURIES

Chest and abdominal injuries are the next frequently attributed to death in forensic practice. Sometimes, the injury may not be evident externally, hiding potentially fatal internal injuries.

Forensic anatomy

When considering the medico-legal significance of the anatomy and structure of the chest and abdomen, the most important aspect is the relation of the visceral contents. It should be appreciated that, positionally, the spleen, most of liver as well as stomach are thoracic organs, laying under the costal margin, vulnerable to chest injuries.

Penetrating injuries to the lower lateral thoracic wall may perforate both the peritoneal and pleural cavities. The diaphragm is invariably perforate as is the stomach, within the rib cage. Similarly, downward stabbing of the heart tend to perforate diaphragm, and cause penetrating injuries to liver.

It is therefore important to consider both thoracic and abdominal organs in one block, especially in penetrating injuries, to ensure that the complete tract is examined.

Injuries to chest wall

Respiration is dependent on the development of negative pressure in the chest cavity to draw air into the lungs. Loss of integrity of the rigid chest wall causes impairment of expansion and severely limits air entry. This can happen either by severe mechanical failure of the rib cage or by penetration of the pleural cavities.

Rib fractures are common occurrences, however, rib fractures do not impair respiration unless:

- Multiple ribs on either side are fractured, causing ‘flail chest’
- Rib fragments ends penetrate the pleura and lungs, causing ‘pneumo and hemo-thorax’
- Severe pleural and muscular pain limit respiratory effort

Frontal impact, classically seen in steering wheel impact injuries, causes fracture of multiple bilateral ribs leading to the development of ‘flail chest’. The loss of rigidity of the rib cage, impairs expansion of the lungs. The dislodged section gets sucked inwards during inspiration, showing the classical sign of ‘paradoxical respiration’. Dyspnoea and cyanosis develop, leading to progressive hypoxia.

The jagged edges of fractured ribs may puncture the parietal and visceral pleura as well as the lung parecbyma, causing a pneumothorax or a haemothorax, or both. These pneumothorax and haemothorax may communicate externally, in rib fractures resulting from penetrating wounds.

Rib fractures are most commonly seen on the anterior or posterior axillary lines, due to the mechanics of the shape of the ribs. The upper ribs are less often involved, except in focal direct violence from kicking, punching or in traffic accidents. Ante-mortem fracture sites almost always show sub-periosteal or sub-pleural bleeding, though rarely ante-mortem fractures may be totally bloodless, while post-mortem cracks may exhibit oozing from marrow cavity into adjacent tissue.

Resuscitation provide a common cause for extensive rib fractures, accounting for up to 40 per cent of all fractures. These fractures caused during attempts at revival are, by definition, peri-mortem in timing, and it is impossible to say if they were immediately ante-mortem or post-mortem.

Intercostal muscles may conceal mobility of ribs due to their bracing action and it is useful to slit all the intercostal muscles with a knife when examining the chest. The ribs in elderly may be extremely fragile due to osteoporosis and may be breakable by finger pressure.

Rib fractures are extremely common in children, especially infants and may even be extremely important in the diagnosis of child abuse. Transverse sqeezing of the chest in an infant causes hyperflexion, resulting fracture of the ribs in the posterior segments, usually near their necks. Callus starts developing within about 2 weeks, but this is extremely variable and so difficult to date.

While anterior rib fractures are rare in infancy, other than from child abuse; they may be the result of vigorous resuscitation. In very young infants, though rare, possibility of fractures dating back to birth cannot be dismissed. The sternum may also be fractured by stamping or other frontal impacts, including resuscitation, but requires far more force than to fracture ribs.

Haemothorax

Any injury to the chest wall or thoracic organs that breach blood vessels can lead to a haemothorax. Most fatal thoracic exsanguination arise from large vessels in the lung or mediastinum, followed by intercostal and mammary arteries to a lesser degree.

Another obvious source of a haemothorax is the heart, though rupture of heart should be accompanied by a defect in the pericardial sac for blood to be spilled into the pleural cavity. The pleural cavities can accommodate several litres of blood, either liquid or clot, or both.

Many intrathoracic haemorrhages may have virtually no external blood loss. As the knife passes obliquely into the chest through the intercostal muscles, forming a valve-like mechanism of tissues, that seals the external wound almost completely and prevents significant external bleeding. Internally however, great vessel or heart chamber may be punctured, resulting in a fatal cardiac tamponade or haemothorax. Post-mortem haemorrhage can add considerably to the volume found in the chest at autopsy and it is impossible to quantify this additional leakage in retrospect.

Infection following a chest wound is uncommon in forensic practice, as most chest trauma cause significant injury are usually fatal before infective sequelae have time to establish. However, cellulitis, pleural inflammation and even empyema may occur, especially when foreign material is embedded into the wound. Some common pathogens associated with chest infection are Staphylococci, Proteus, Coliforms and Clostridium Perfringens.

Pneumothorax

Pneumothorax is defined as collection of gas in the pleural cavity. They are of three types:

1) Simple type – pleural leak allows air to enter pleural cavity before rapidly closing. The lung partly collapses and the air is gets absorbed on recovery. The communication may rarely remain open, leading to a non-pressurised bronchopleural fistula
2) Tension pneumothorax – leak in the pleura (or rarely the chest wall) has a unidirectional valve-like action, resulting in air being sucked into the pleural cavity on inspiration, but cannot escape on expiration. This causes complete collapse of the lung onto its hilum and a shift of the mediastinum to the opposite side.
3) Sucking type – commonly seen in combat injuries, when an injury of the chest wall communicates with the pleural cavity, forming a direct passage of air from the exterior. These injuries are commonly complicated by haemorrhage and infection.

Pneumothorax are commonly traumatic in origin but natural disease can also cause a pneumothorax. Common causes include ruptured emphysematous bulla, tuberculous lesion at lung periphery, or a tear at the site of fibrous pleural adhesion, that communicate with pleural cavity and lead to development of pneumothorax.

Injuries of the lungs

Lungs may be injured in both open and closed chest injuries. Any substantial impact on the chest can contuse lungs beneath the area of impact or cause ‘contrecoup’ lesion on the opposite surface. Deceleration injuries, seen in fall and road accidents. occur along the posterolateral surfaces in the paravertebral gutter, as a vertical line of subpleural bruising or rib imprint. Impact may form subpleural blood blisters, which release blood or air into the pleural cavities.

In all severe chest injuries the central parts of the lung may show bleeding, sometimes forming haematomas, with breakdown of lung tissue. Osborn describes ‘pincer contusions’, where expanded lower margins of the lungs become trapped in the narrow costophrenic angles.

Laceration with detachment of lobes or even entire lungs from the hilum may occur in blunt injuries. Pulmonary ligament below the hilum is a frequent site of haemorrhage. Hilar as well as more peripheral vessels may rupture, causing intrapleural or mediastinal haemorrhage. Lungs are the most vulnerable organ to this blast injury due to the large tissue–air interface.

In children, greater elasticity of ribs and chest can result in lung injuries without fracturing of ribs.

Injuries of the heart

The heart is vulnerable to both penetrating and blunt injuries. The commonest form of fatal cardiac insult is a stab wound , most frequently entering via the intercostal space, less frequently from the abdomen and rarely penetrating the sternum.

The right ventricle is most commonly injured in a stab wound, due to its largest frontal area, but the anterior interventricular septum and the left ventricle are also susceptible. A shallow wound may not reach the lumen of the ventricle, and may cause death from cardiac arrhythmias, or as a result of coronary vessel rupture, resulting in myocardial insufficiency or cardiac tamponade. Frequently, the knife passes into the cavity, causing massive bleeding into the pericardial sac. In the right ventricle, muscular thickness is insufficient to close the defect despite low intraventricular pressure contraction of the thick left ventricle wall may seal the wound, and reduce bleeding. In general, wounds in the right ventricle are more dangerous because of the absence of this ‘self-sealing’ effect.

Blunt injuries of the heart seen in traffic accident, fall from a height or stamping assault, can cause fatal damage. There are usually multiple rib and sternal fractures, and may present with flail chest.

Cardiac injuries from blunt trauma are commonly present over the front of the organ, especially right ventricle, though posterior bruising and laceration can occur if the heart is compressed against thoracic spine. Myocardial rupture may be present, without external signs of injury to either the heart or the external chest wall. The interventricular septum may be ruptured as a sole lesion, the cause being obscure. In high impact injuries, such as aircraft crashes, the whole heart may be avulsed from its root, and may be found lying loose in the thorax.

Occasionally cardiac damage may be seen in an intact chest, especially in a child with pliable thorax. Fatal blunt damage of the heart may occur, however, without a mark on the skin of the thorax nor damage to the chest wall.

Haemopericardium and cardiac tamponade

Hemopericardium may result due to hemorrhage from the surface or cavity of the heart, or even from the intra-pericardial segments of the roots aorta and pulmonary artery. Most causes of haemopericardium are from natural disease, such as ruptured myocardial infarct or ruptured dissecting aneurysm of the aorta, but it is also a common sequel of injury to the chest.

When blood accumulates in the pericardial sac, as is seen following natural disease, or when this accumulation is faster than it can escape, when following trauma, this cause ‘cardiac tamponade’. Moritz states that 400-500 ml of blood cause sufficient pressure on the heart to prevent atrial filling during diastole. However, cases of sudden death with incidental autopsy finding of cardiac tamponade shows significantly less blood in the pericardium, amounting between 100 and 200 ml.

Injuries to great vessels

Aorta is the most vulnerable vessel, commonly getting injured due to deceleration trauma in road and air accidents, and in fall from a height. Sudden deceleration of the thorax causes severe traction on the root of the heart, resulting in complete or partial tear of aorta in the descending part of arch of Aorta. Fiddler states that this is almost constantly at a point 1.5cm distal to the attachment of the ligamentum arteriosum, the remnant of the ductus arteriosus, is annular and transverse to the axis of aorta. The aorta may have multiple parallel intimal tears, ‘ladder-rung tears’, which affect only the intima and inner media, leading to false aneurysms and dissections following recovery.

The pulmonary artery is less vulnerable, but may be damaged by depressed rib cage and sternal fractures, seen in stamping assaults and steering wheel impacts. Pulmonary vessles are more vulnerable to injury at the hilum of the lungs. Both Aorta and pulmonary vessels are often involved in penetrating injuries, notably stab wounds, of the chest. The rupture of blood vessels may result in a variety of conditions, including hemothorax, hemopericardium, etc

Abdominal injuries

Abdominal injuries are also varied in severity depending on the location of the injury, in relation to the location of abdominal structure. The large coverage of the anterior abdomen by the intestine frequently results in perforation with consequent chemical or infective peritonitis. The liver and spleen, bleed extensively, and may cause haemoperitoneum. The intestine and mesentery are other frequently injured structures, wounds often being multiple because of their overlapping structure. The stomach, partially protected by the rib margin, is less often penetrated from the abdomen, but may be more frequently associated with downward chest stabbings. The kidneys are frequently associated with a thrust in the back.

Closed injury to the abdomen following blunt trauma is common in both accidents and assault. The liver, intestine, spleen and mesentery are most vulnerable. In homicides, assaults and child abuse, kicking, stamping and heavy punching can also cause blunt injury to the abdomen.

Bruising of the abdominal wall, skin and underlying muscles, may tend to migrate track from the initial area of impact to become more diffuse. Blood may track down the inguinal canal and appear in scrotum or labia, confusing possible association with sexual assault. Fingertip bruises may be seen, especially in child abuse, and are called ‘penny contusion’.

Severe or fatal intra-abdominal injury may be present without any mark on the skin. This is common when clothing protects underlying structures or if the impact is diffusely applied, over a large surface. Liver, mesentery or intestine may show extensive contusions and injuries with no external sign whatsoever.

Intestine and mesentery are frequently damaged in abdominal trauma. Crushing of the abdomen may result in extensive bruising of the gut and its vascular mesentery, resulting in multiple microruptures of the mesentery, causing extensive haemoperitoneum.

The duodenum and jejunum are particularly vulnerable to transection, especially in children, where a heavy blow in the central or upper abdomen can compress the duodenum against the spine, transecting the third part, almost as cleanly as with a scalpel.

Injury to the mesentery may also damage arteries without causing severe bleeding, but rather causing occlusion or thrombosis, resulting in bowel infarction as a consequence. Perforation may be delayed for a day or two. The colon is rarely injured unless the abdominal injury is gross or in case of forcible insertion of foreign objects into the rectum, associated with sexual assault.

Rupture of the spleen is commonly undiagnosed and can occur from either impact on its surface or from traction on its pedicle. An enlarged spleen is more fragile, with malaria, glandular fever and other infections causing increased risk of rupture. The rupture may be immediate or may be delayed by large subcapsular haematoma with the tear protected by the capsule for days or even weeks.

Hepatic rupture is another common manifestation in serious abdominal trauma, following fall from height or in road accidents. Hepatic rupture will cause significant exravasation of blood into the peritoneal cavity even after death. The liver may show linear cracks, commonly on the convex antero-superior surface, ranging in severity from superficial subcapsular tears to complete transection. Subcapsular haematoma may form that is protected from rupture by the capsule, causing a delay in collapse and subsequent death.

Hepatic rupture is a well-recognized component of child abuse syndrome due to the frequency of presentation. Liver tears also occur during the process of birth, particularly in breech presentation.

Renal parenchymal injuries are much rarer due to the location at the back of the abdomen, in the paravertebral gutter. However, kidneys may be injured in kicks or heavy blows to the loin and is also seen in traffic accidents. Hemorrhage of the peri-renal pad of fat is much more common than damage to the actual organs themselves. As such, depending on the intensity and location of force, kidneys may show the entire range of injuries, from pulverization to shallow surface lacerations. Vascular injury may result in post-injury infarction. However, it is difficult to differentiate direct trauma to the suprarenal vessels from the common post-stress haemorrhages, often bilateral, of the Waterhouse–Frederichsen type. These occur quite commonly a few days after any trauma, surgical operation or infection, and are rarely diagnosed by clinicians.

Foreign bodies in the gut

Though not usually associated with trauma, foreign bodies are frequently found in the alimentary system, both clinically and at autopsy. Disorders of the mental state have precipitated ingestion of a large number of objects. Betz et al. described a death due to intestinal obstruction from ingestion of his own hair. Sexual perversions, and hyper-normal sexual activities also account for insertion of foreign objects into the rectum, urethra, and vagina.

The 'body-packing' phenomenon has seen a rise in drugs being concealed in condoms, either swallowed or, less often, inserted into the rectum or vagina. Deaths have occurred following rupture of a package within the gut, releasing a massive lethal dose of heroin or cocaine. Rarely, the mass may cause intestinal obstruction.

Injuries to the pelvis and pelvic organs

Pressure applied to the front of the abdomen or pubic area, in running over, may cause fracture dislocation of the symphysis and one or both sacroiliac joints. A lateral impact may shatter the superior and, less frequently, inferior pubic ramus as well as dislocate ipsilateral sacroiliac joint. A fall from a height onto the feet may transmit the force up the legs and cause either dislocation of the hip joint or even impaction of one or both femoral heads through the acetabulum. If the hip joints remain intact, the pelvic girdle may fracture and dislocate the sacroiliac joints. A kick or heavy fall onto the base of the spine may fracture the coccyx or sacrum.

Pelvic organs, except for the bladder, are well protected from trauma. Blows or kicks on the lower abdomen may cause rupture of the bladder, when full. The empty bladder is rarely injured. The male urethra is also vulnerable to trauma. The scrotum is vulnerable to severe bruising, especially from kicks – resulting in development of haematocele. Scrotal and vulval injuries can occur from falling astride an object, while cycling, or in traffic accidents.

Complications of abdominal injury

Hemorrhage is the commonest fatal sequel to intra-abdominal trauma, with spleen and mesentery accounting for copious and fast bleeding, while the liver tends to ooze more slowly. The mesentery frequently causes massive hemorrhage due to the difficulty in diagnosing the bleeding site. Perforation of the gastrointestinal canal is another serious complication of abdominal trauma,

though death often ensues from the combined effects of injuries, before infection gets established. Perforated peptic ulcer, penetration of the stomach or duodenum will cause chemical peritonitis that can cause severe and immediate shock. Rupture of the intestines will inevitably lead to generalized peritonitis. In addition to internal infection, open wounds introduce extraneous organisms into the peritoneal cavity that can cause infection. Trauma to the intestine may also cause an intractable ileus.Injury to the pancreas can cause leakage of proteolytic enzyme and may be cause widespread fat necrosis in the mesentery and omentum.

Medico-legal aspect of injuries:

1. Cause of injury.
2. Direction of injury.
3. Time of injury
4. Identification of the object inflicting injury.
5. Homicidal wound

   Homicidal wounds can be present over any part of the body, commonly on the neck, head and trunk. Wounds may be present at the inner side of forearm or hand of victim while defending or protecting. 'Defense Wounds'.
6. Suicidal wound

   Suicidal wounds are usually found in the accessible parts commonly by light weapons on the throat (cut throat wounds). Tail end of the wound indicates which hand has been used.
7. Accidental wounds

   Accidental wounds can be seen at any part of the body.
8. The depth of the wound may indicate the force of the penetration.
9. Direction and dimensions of the wound may help to indicate the positions of assailant and victim.
10. Age of the injury can be estimated depending upon the extent of healing.
11. Position, number and direction of wounds can give clue for manner of production-Suicidal, Accidental or Homicidal.

Legal Aspect of Injuries in Nepal

Muluki Ain, 2020 B.S.

Kutpitko Mahal

Section 1. If some body produces bleeding or wounds or pain and other harm on the body by means of weapon or by any way is physical assault.

Section 2. If any of the following conditions result from assault that amounts grievous hurt (Angabhanga)

Permanent privation of vision……...1

Permanent privation of smelling power of nose…….…….1

Permanent privation of hearing capacity of ears.....…...1

Permanent privation of talking power of tongue…….…….1

Cutting breasts of female to loss of function……..…1

Emasculation in male and infertility in female due to injuries .....……1

Fracture and dislocation of joints of spine, hands and legs leading to disability…….1

Section 9. If injuries are produced by simple weapons or sticks, stones, not resulting angabhanga, measurement of depth in penetrating injury and measurement of length in other injuries should be taken. The punishment will be Rs.500/- per inch fine from greatest dimension. Injuries produced by guns, Khukuri, Sward, Chopper, Axe, Khunda, Vala and like others, the punishment will be Rs. 500/- per inch fine and imprisonment of one month per inch up to maximum two years.

Section 10. Injuries less than one inch produced by sticks, stones or other weapons, the punishment will be up to Rs. 500/- fine with consideration of nature of weapon and nature of injury.

Section 11. If there are bruises or contusions due to assault, the punishment will be Rs. 100/- per bruise up to Rs 1200/- fine.

Section 12. If there is fracture or dislocation of teeth, the punishment will be Rs. 300/- per tooth fine.

Section 14. A Sec.14 A. If corrosives or alike substances splashed, pasted, smeared or applied by other ways resulting disfiguration to the face or any other parts of body, the punishment will be 5-8 years of imprisonment and Rs.100000/- to 300000/- fine in case of facial disfiguration, and 3-8 years of imprisonment and Rs. 50000/- to 150000/- fine in pain and disfiguration in other parts of body. The fine amount should be given to victim as treatment expenses.

Section 22. Sec.22. During the examination and documentation of injuries, if there is distortion of measurement of injuries or documented false injury deliberately which effects in punishment, the examiner will be fined up to Rs.250/- with consideration in differences of punishment.

Section 23. Sec.23. In case of malingering with false injuries or scars, the examinee will be punished with Rs. 100/- fine.

Materials and equipment required

1. Well lit room with examination table
2. Chairs and tables as requird
3. Stationeries
4. Six inch scale
5. Measuring tape
6. Height measuring scale
7. Magnifying glass
8. Digital Camera
9. Weighing machine
10. Stethoscope
11. Sphygmomanometer
12. Torch light
13. Gloves
14. Sterile swabs

Examination of injury

The examinee may:

1. Come to the emergency department with injury. You inform the police. Treatment and injury examination goes side by side.
2. Come with police with the requisition letter. You receive the requisition letter and proceed for examination. Sometime you may not be the initial treating physician. In such case you ask for previous treatment document and proceed for present examination.
3. Come with requisition later from police/Court whom you might have or might not have treated in the past.

Requisition letter:

Requisition letter is the document written to the physician by the police or court asking for performing medicolegal examination and preparing report of the case. Usually two copies are send by the police. Return one copy to the police after signing at the back of letter mentioning that you have acknowledged the case. If you have stamp of yours or your hospital then stamp over your signature. Also, write your full name, mobile number and date. Next copy, keep it for your record.

Rapport Building and unrestraining the examinee:

Rapport building is equally important in clinical as well as in medicolegal cases. Ask the police to remove restrains (usually hand cuffs) if any. Examination of subject with restrain is unethical. Perpetrator or victim both are patients to a physician so there should be no discrimination.

Be polite and empathic while dealing with the examinee.

Take the examinee to examination room. Police can stay in the same room but make sure that the police stays far enough so that the conversation between you and the examinee is not audible to him. Otherwise, separate room in presence of chaperon is preferable. Chaperon is especially needed during examination of a female examinee by a male doctor. Make sure that the examinee feels safe and maintain privacy as far as possible while examining.

Ask name, age, sex, address, occupation of the examinee. If code name is mentioned in the requisition letter, note down the code name. You can ask his/her real name but the real name should not come in the final report for the purpose of confidentiality.

Examinee are often allocated code names by the police, especially in sexual assault case. This is a good practice as it prevents disclosing the real identity of the victim and thereby helping to prevent future harassment and psychological impact to the victim.

History in the words of the examinee:

Before examining patient it is very much essential to note down the history of causation of the injury in the world of the examinee. WH QUESTIONS (When, how, what, who, where etc.) are the main source of deriving history relating with the injury.

Note down when, how, where and by whom injury was inflicted. Also ask what was done after sustaining injury. e.g. any treatment.

Regarding mild head injury always ask about the loss of consciousness and any amnesia before and after the injury. This is very important to know about the concussion of the brain.

Informed consent:

Informed consent is equally important not only in clinical cases but also in medicolegal cases. If a person is examined without prior informed consent then it amount to assault and is also against the medical ethics.

Adult person more than 16 years of age and with mental soundness can consent for examination. Minor and mentally unsound person cannot give consent for examination hence respective guardian is a must.

While taking consent it should be clearly explained regarding the examination and its subsequent disclosure to the court of law.

What if the examinee is a minor (less than 16 years of age) ?

When examinee is less than 16 years of age consent has to be obtained from the available guardian. If no guardians are available consent can be obtained from accompanying police personnel. Obtaining consent form police personnel is often not valid in court of law so this mode of obtaining consent should be taken only when other means of obtaining consent are unavailable, e.g. in case of street children. But you may often find non-government organizations working for the welfare of the society, who might come forward to take the responsibility. In such situation consent can be taken form the representative form that non-governmental organizations.

What if the examinee denies consent?

In such situation, forced examination of the examinee is not recommended. Try to find out the cause why the examinee is not consenting for examination and proceed accordingly. If the examinee still refuses to give consent, explain that it will go against him and document the situation on your report along with signature of any two witnesses (colleagues or hospital staffs). Also mention date, time and position of the witnesses.

Identification marks (हुलिया):

Identification marks are the integral part of any medicolegal report. These marks commonly help in positively identifying the examinee. Permanent scars, tattoos or any permanent peculiar feature on the body, preferably over the visible parts of the body, are recommended for recording in the report. If such features are not available over the visible parts, then features present at other areas of the body can also be taken. If no tattoos, scars or peculiar features are available you can describe the facial features e.g, complexion, shape of the face, eye brown, lips, baldness of hair etc. Record any two such features in the report.

General examination:

Measure body length with help of measuring scale. If measuring scale is not available, mark upto seven feet on the wall with help of measuring tape. Make the subject stand against the wall with his/her back facing the wall and with bare foot. Take a ruler and place it horizontally over the head. Mark this point on the wall and measure the length.

Take the body weight on a weighing machine.

Take pulse, Blood pressure, Respiratory rate and body temperature (armpit temprerature). If subject vitals are unstable refer the case to Emergency department or treat the case. Report can be prepared at later date when examinee becomes well.

Dental examination:

Make a dental chart as shown below

Right	Left
18 17 16 15 14 13 12 11	21 22 23 24 25 26 27 28
48 47 46 45 44 43 42 41	31 32 33 34 35 36 37 38

S = ___ Total Teeth (Permanent/Deciduous/Mixed)

The number given above is the standard representation of the teeth. For example tooth no. 11 is right upper central incisor, tooth no. 25 is left upper second pre molar, tooth no. 48 is right lower wisdom teeth and teeth no. 37 is left lower second molar teeth and so on.

Note down any peculiar dental findings, missing teeth or unerrupted teeth on the respective teeth number.

Past medical history:

Ask the examinee about the past medical history. Past medical history may include history of Diabetes mellitus, Hypertension, Tuberculosis, any history of trauma or any psychiatric illnesses. These past medical history often help to correlate the effect of previous disease on present injury fate.

Personal history:

History of alcoholism, tobacco and other drug of abuse are also important. Menstrual history is important particularly in menstruating women. Aslo take obstetrical history in relevant situation.

Injury reporting and opinion.

Report should be prepared as per the format available in Nepal Rajpatra.

Opinion regarding the injury is one of the most important part of the medicolegal report. Answer the questions asked by the police or the court as mentioned in the letter.

Grievous or non-grievous

Though the gravity of the injury, that is grievous or non-grievous, is the verdict of the court, the court often needs opinion of the physician to give the verdict. Hence you should also mention the grievousness of the injury as well. To tell whether the injury is grievous or non-grievous, follow the Muliki Ain 2020 in Kutpit ko Mahal as described earlier

Fatal or non-fatal

The court often need opinion whether the injury is fatal or not fatal. It might be challenging to tell whether the injury is fatal or non-fatal. Though fatal injuries usually land up in mortuary, the injuries present on the body can be opined as fatal or non-fatal by asking yourself a questions. Will the injury cause death in normal course of time and in absence of complication? If answer is yes then the injury is fatal otherwise it is non-fatal. For example a penetrating wound to the foot is non-fatal even though you might argue that it can be fatal if it is complicated by tetanus. But similar penetrating injury in the abdomen is always regarded as fatal though you can argue that there may not be complication of peritonitis. Penetrating injuries to head, chest and abdomen are always considered as fatal. Likewise contusion of internal organs are also considered as fatal injury eg, contusion of brain, heart, liver, lungs intestine, kidneys and so on.