HEAD INJURIES

TABLE OF CONTANTS

1. INTRODUCTION

Injuries involving the head and neck are the most common and most important injuries in forensic practice and are the most frequent causes of mortality and morbidity. The reason seems obvious; the head is the target of choice in great majority of assaults. The brain and its covering are vulnerable to degrees of blunt trauma that would rarely be lethal if applied to other areas.

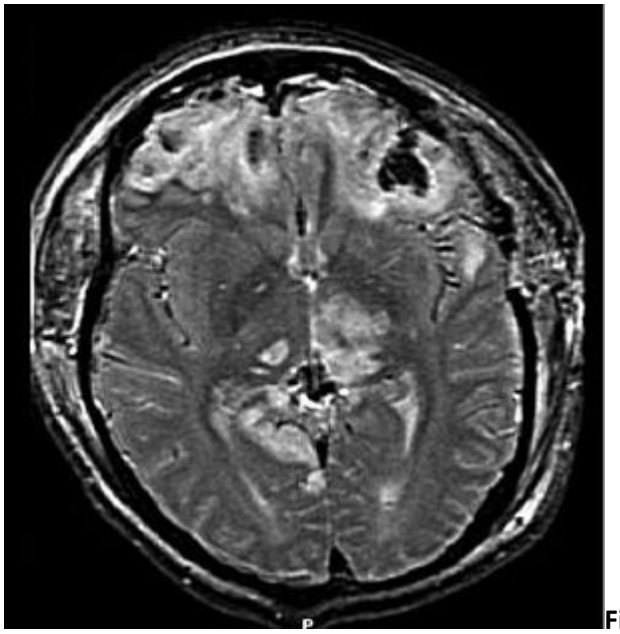


Fig.1.1

Sound knowledge of the anatomy of the head and neck including practical understanding of the neuropathology of trauma is non-negotiable. Correct interpretation of different lesion is of great medico-legal importance, because it assist investigators reconstruct the sequence of event which resulted in injury in-so doing therefore facilitate the administration of justice.

NB: Consult relevant text for anatomy of the head and neck!!

2. FORENSIC/ MEDICO-LEGAL CONSIDERATIONS IN HEAD INJURIES

SCALP INJURIES

- Anticipated injuries are often not found (protective features of hair or helmet etc)
- Scalp wounds bleed profusely
- Infections can spread through venous connection from the scalp to cranial cavities resulting in meningitis or brain abscesses.
- Scalp injuries caused by blunt force may simulate an incised wound.
- Subaponeurotic haemorrhages migrate and can gravitate from frontal or parieto-temporal regions towards peri-orbital and pre-auricular regions respectively causing confusion to the exact cause of the ecchymosis.
- In a newborn baby normal birth trauma must be distinguished from injuries caused by instrumentation, a fall or deliberately inflicted injuries.
- At post mortem examination careful removal of hair might reveal hidden injuries.
- Determine whether the object which caused a scalp wound also penetrated the skull and consider the possibility of a broken-off piece of the insulting object embedded in the brain.

SKULL INJURIES

- The amount of force which caused the fracture cannot be determined with certainty.
- Guard against empirical statement with regards to direction and type of weapon used, it is often impossible to deduce that information.
- Death is not caused by the skull fracture *per se* but by secondary lesion like haemorrhage, infection or brain tissue injury.
- Not having a skull fracture does not exclude severe head trauma.

EPIDURAL HAEMORRHAGE

- Always the result of trauma.
- Discrimination between traumatic epidural haematoma and epidural burn haematoma should be made.

SUBDURAL HAEMORRHAGE

- Venous bleed thus often symptoms will develop gradually but no always.
- Especially in the elderly the primary traumatic event is forgotten.

- Injury to a head in a fixed position will not cause a subdural haemorrhage unless there is some mobility of the intracranial content.
- Differentiate between subdural haemorrhages vs. subdural hygroma.
- Dating of subdural haemorrhages is an important part of the investigation.

SUBARACHNOID HAEMORRHAGE

- Natural vs. Complication of trauma
- Differentiate between post mortem haemolysis in putrefied bodies especially over the occipital region.

INTRACEREBRAL HAEMORRHAGE

- Natural vs. Complication of trauma.
- It is very important to conduct extensive histological examination of the brain surrounding an intracerebral haemorrhage, so that an underlying pathology can either be confirmed or excluded.

BRAIN CONTUSIONS

- Contusions follow injury.
- It is important to differentiate between a cortical contusion and a cortical infarction or necrosis.
- It is important to think in terms of coup and contrecoup injuries when determining the point of impact.
- In healing a focus can develop which could give rise to epileptic fits.

MENINGITIS

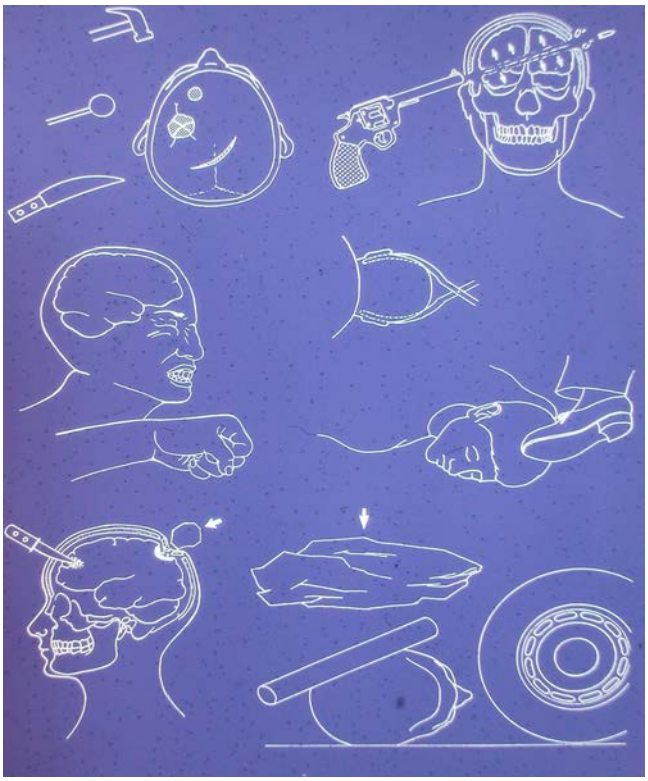
- Natural vs. Complication of trauma.
- Take a pus swab for microbiological examination in all cases.
- To look for a possible source of infection such as sinusitis, otitis media or mastoiditis.

BRAIN ABSCESS

- Complication of trauma vs. Natural origin**.**
- Swabs for microbiological examination.
- To look for a possible site of infection, such as sinusitis, otitis media or mastoiditis, or a site of injury. Pay special attention to the lungs because brain abscesses can follow upon bronchiectasis.

3. MECHANISMS OF HEAD INJURIES

Fig.3.1. Causes of head injuries



CONTACT (Blunt; Sharp) **vs. Non-CONTACT** (Acceleration; Deceleration; Rotational)

Analogy for head injury: Jelly (brain), wrapped in cling film (arachnoid), in a paper bag (dura), inside a cardboard box (skull), wrapped in brown paper (scalp).

Any layer maybe damaged by: Direct impact on the box (blow/stab), Dropping the box (fall) or Shaking the box (acceleration/deceleration).

CONTACT (object directly impacting on the head)

- **Local effects:** Laceration/ Incision of the scalp, Skull fracture, Extra-dural haematomas, Cortical contusions etc.
- **Distant effects:** Contrecoup contusions/ Contrecoup fractures, Intracerebral haemorrhages.

NON-CONTACT

- The brain mass is suspended in a fluid medium (CSF), different movements at different speeds of the head affects the position and shape of the brain.

- When the box (skull) suddenly moves (accelerate/decelerate/rotate) for a moment the skull moves faster than the brain, in other words the brain 'lags' behind the skull frame. This is one of the important mechanisms of brain injury.
- The different types of forces involved in head injuries are ***compressive, tensile and shearing*** forces.

NB: See also **BK, Forensic ABC, Forensic Pathology by Di Maio** and **Leestma's Forensic Neuropathology** for future reading on mechanisms of head injuries.

Table 3.1 Head injuries: pathology and mechanisms of death

Mechanisms / Pathology	Primary IMMEDIATE	Primary EVENTUAL	Secondary EVENTUAL
Contusion **Laceration**	Destruction of functional brain tissue		Mechanisms of complications such as: • Bronchopneumonia • Shock lung • PE
Haemorrhages **Oedema** **Tumour**		Suppression of vital functions	
Infarction **Haemorrhage**		Destruction of functional brain tissue	
Infection **Abscess**		Toxaemia; suppression of vital functions	

4. CLASSIFICATION OF HEAD INJURIES

The classification of head injuries is a vast and complex topic. There are many attempts at classifying head injuries but invariably all of them are insufficient. There is merit to each proposed classification and alas a product of the different proposed classifications could be a viable system. Regardless of the complexities a practical understanding of the neuropathology of trauma is essential knowledge.

Head injuries can be classified into 2 major categories of brain damage, **primary** and **secondary** damages. The primary damages are those that occur at the moment of the injury and include scalp lacerations, skull fractures, contusions and lacerations of the brain, shearing/tensile injury (traumatic diffuse axonal injury, concussion, pontomedullary rent and multiple petechial haemorrhages), and the intracranial haemorrhages. The secondary damages are those produced by complications arising from the primary damages and include the lesions produced by increased intracranial pressure, ischemia, brain swelling, and infection.

An alternative method of classifying the pathology of head injury is into **focal** and **diffuse** injuries. Focal injuries are those which can be seen by the naked eye or by imaging studies; these injuries result from direct impact to the head (e.g., scalp laceration and contusion, skull fracture, epidural haemorrhage, subdural haemorrhage, brain contusion).

Classification of changes following on a specific head:

a. Injury caused by a sharp or blunt force.

b. Area of the injury: widespread or localised injuries.

c. Open and closed injuries.

d. Injuries caused by direct or indirect force.

A practical approach might be to describe head injuries anatomically including pathologic fallout from the specific anatomical injury:

a. Extracranial (open or closed)

b. Intracranial (open or closed)

ANATOMICAL CONSIDERATIONS OF THE HEAD AND NECK

- The adult head weighs between 3kg and 6kg averaging 4-5kg.
- An average brain masses at autopsy of 19-20yr olds: M-1430g F-1294g
- The head is pivoted on the cervical spine (mystery of God!! small unstable joint supporting large mass)
- The scalp is thick, vascular and multi-layered.

- Be cognisant of the possible protective gear like a helmet or cap obscuring external injuries on the head.
- The skull is a well reinforce bone and internally has irregular ridges.
- The brain is almost incompressible, and purely axial impact may give rise to little or no cerebral damage.
- The well-known aphorism of Munro (1938) must be kept in mind that: 'Any type of head injury can give rise to any type of intracranial damage'.

5. EXTRACRANIAL (CLOSED OR OPENED)

A. SCALP

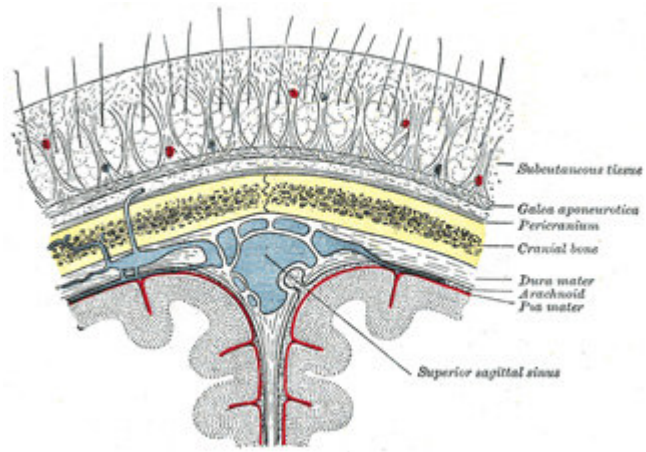


Fig.5.1

- The scalp is very vascular and after injury can bleed profusely.
- The absence of scalp injuries does not exclude skull or intracranial damage and the presence of scalp injuries does not always result in skull and intracranial damage.
- The presence of hair on the scalp is the modifying factor on the scalp which could deflect a tangential blow or partly cushion a direct impact.
- Injuries to the scalp include: Abrasions, Bruising, Lacerations, Burns and Incised wounds.

Scalp abrasions

- Abrasions on the scalp are less common and are less defined due to the protective effect of the hair. If not looked for, they are easy to miss.

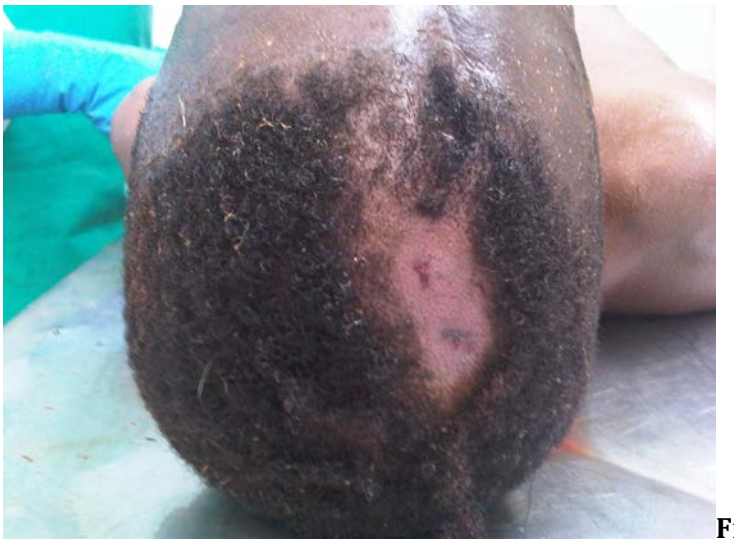


Fig.5.2. *Abrasions*

Scalp bruising

- Swelling usually subsides or diffuses after death. Bruising of the scalp is bleeding into the scalp layers commonly between the aponeurosis and the epidermis but could be present below the aponeurosis. Also bleeding could be present below the epicranium.

- Bruising in the frontal and temporal regions can be mobile under the influence of gravity toward the peri-orbital region (**peri-orbital haematoma**) and behind the ear (**battle sign**) respectively.

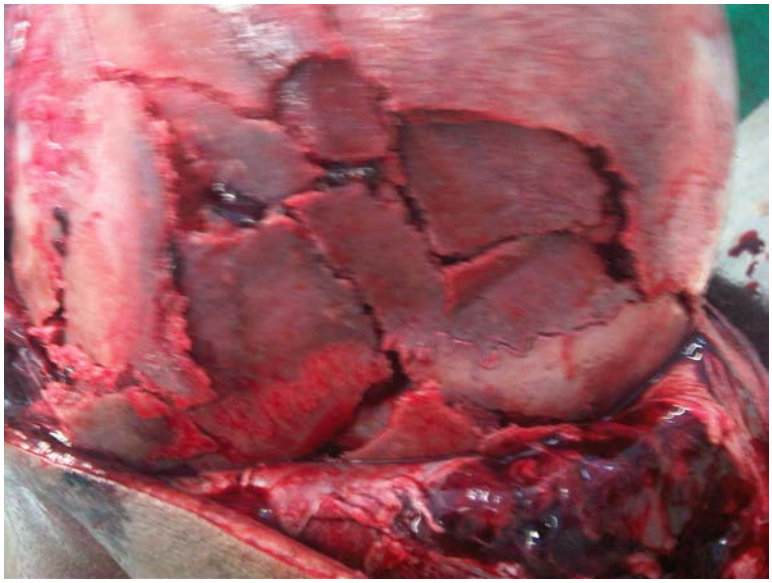


Fig.5.3. *Subaponeurotic haemorrhages and comminuted depressed skull fracture*

Scalp lacerations

- Lacerations on the scalp bleed and if not managed judicially can lead to death. The worse type of a scalp laceration is avulsion of the scalp.
- May reproduce the pattern of the inflicting object, even though a random splitting is common.
- It might be difficult to differentiate between a laceration (blunt force injury) and an incised wound (sharp force injury). Closer examination with a magnifying lens will show characteristic differences between the two types of injuries.
- Scalp injuries may bleed profusely even after death, especially under the influence of gravity.

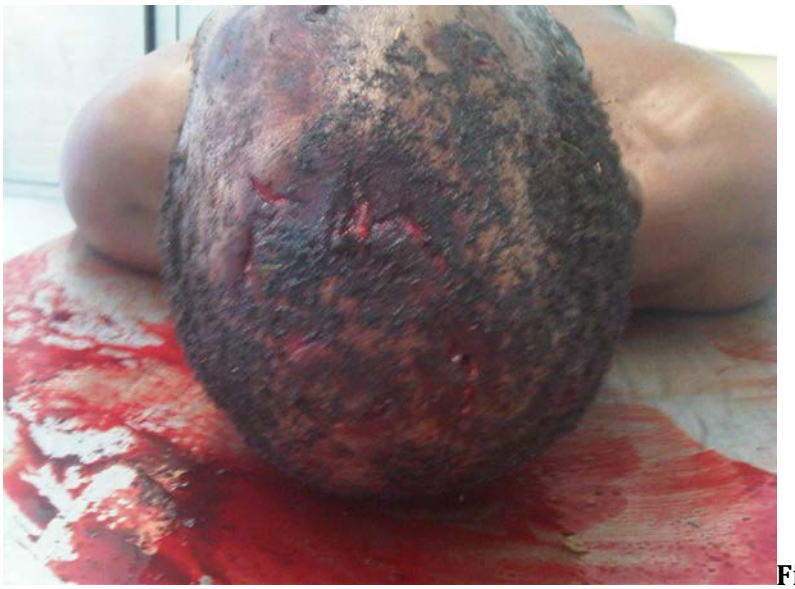


Fig.5.4. *Scalp lacerations*

Incised wounds

- The description is similar to other regions except careful examination to differentiate from scalp lacerations.

Burns

- The description is similar to other regions.
- Depending on the severity, could be associated with heat fracture or heat haematoma especially with severely charred bodies.

NB: Injuries to the vertex should always raise the suspicion of assault, as it is unusual to fall upon the top of the head, even from a considerable height.

B. SKULL

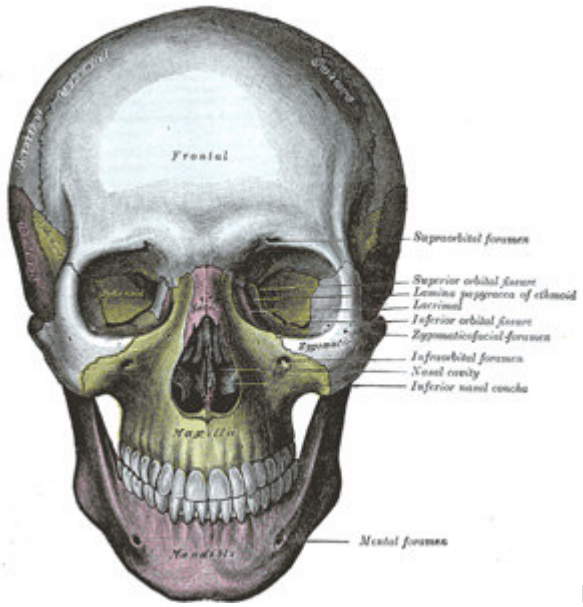


Fig.5.5

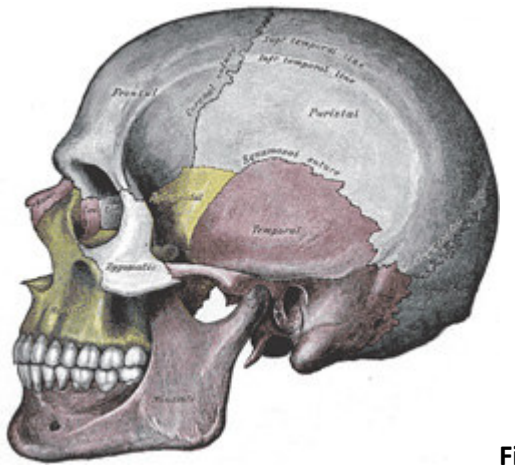


Fig.5.6

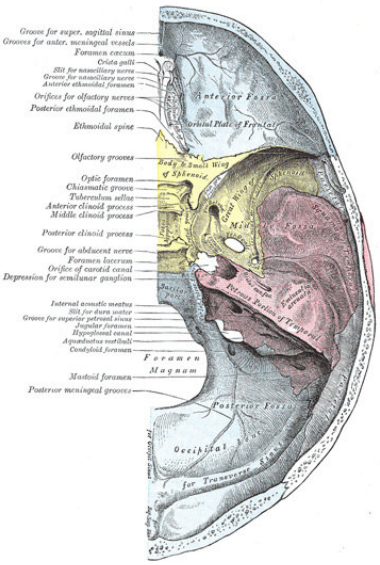


Fig.5.7

- The anterior fontanelle closes functionally between 9 and 26 weeks after birth, though is not sealed until about 18 months.
- The adult cranium consists of two parallel tables of compact bone separated by a central zone of cancellous bone (Diplöe).
- Outer table approximately twice as thick as the inner table.
- Remember intracranial damage can occur with an intact skull.
- The presence of a skull fracture is, however an indication of the severity of the force applied to the head.
- For **biomechanics** of skull fracture see (**BK** p182-183)

TYPE OF SKULL FRACTURES

- The vault and the base of the skull can be fractured.

VAULT SKULL FRACTURES

Fig.5.8

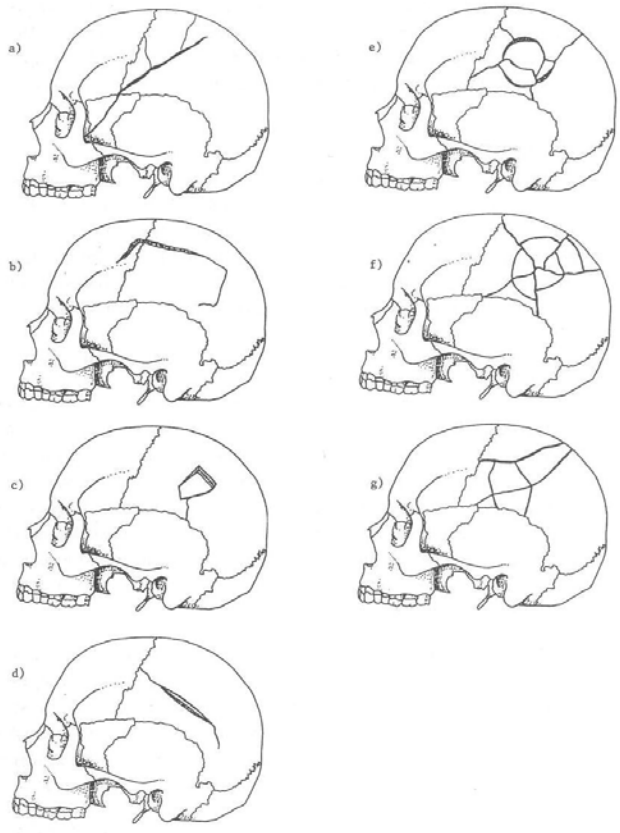


Figure 4 – Different types of blunt force trauma: (a) linear fracture with radiating fissure fractures, (b) depressed fracture, (c) depressed fracture á la signature, (d) depressed fracture produced by a sharp-edged weapon, (e) depressed pond fracture (also comminuted), (f) depressed stellate fracture, (g) comminuted fracture (reproduced by permission of Sarah King[30]).

Linear skull vault fractures

- Can occur anywhere, may involve outer table of the skull bone or transverse both however most common over unsupported plates like the temporal, frontal, parietal or occipital plates.
- Single or multiple and may extend into the suture lines resulting in "Diastasis" of the suture lines.
- Maybe extend into the base of the skull (e.g. Transverse 'hinge' fracture)
- Arise under or at a distance from the impact area or radiate from a depressed zone (radiating fractures).

Pond fractures

- Descriptive term for a shallow depressed fracture forming a concave 'pond'.
- Especially in pliable bones of infants there might be depression without fracturing.

Spider's-web fractures

- Comminuted depressed fractures with fissures radiating away from the fracture.

Depressed fractures

- Outer table with or without inner table depression from focal impacts.

'Contre-coup' fractures

- Usually from a hard surface striking the occipital region of a mobile head (causing a coup damage), transmitted force maybe sufficient to fracture the thin bone on the floor of the anterior fossa.

<u>BASE OF SKULL FRACTURES</u>

Hinge fractures

- Sometime called 'motorcyclist's fracture'
- Fracture line crosses transversely across base of the skull.

Longitudinal fractures

- Fracture line longitudinally at base of skull.

Ring fractures

- Occur in the posterior fossa around foramen

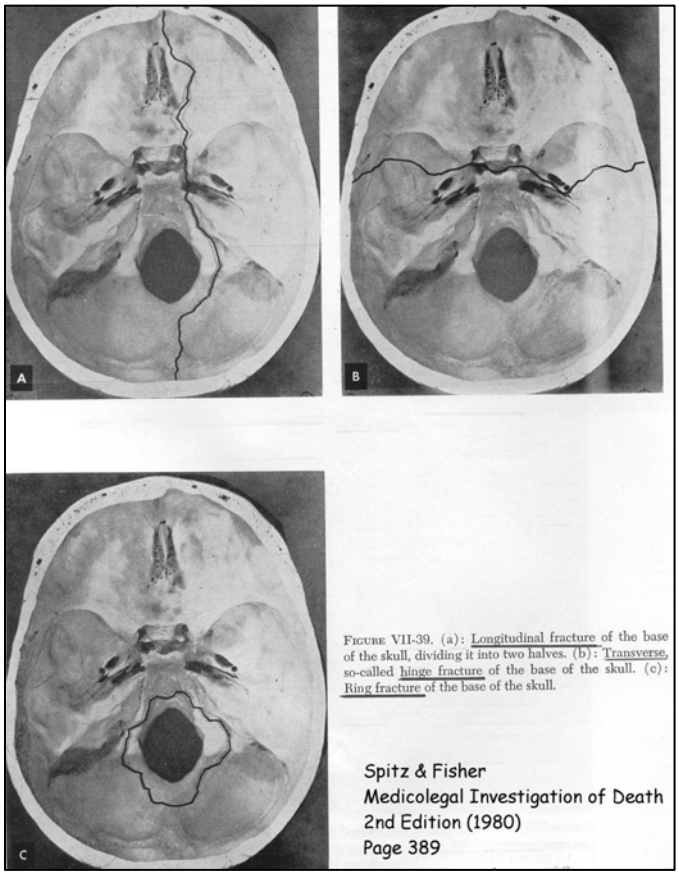


FIGURE VII-39. (a): Longitudinal fracture of the base of the skull, dividing it into two halves. (b): Transverse, so-called hinge fracture of the base of the skull. (c): Ring fracture of the base of the skull.

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magnum and mostly from falls from a height.

OPEN SKULL INJURIES

- May occur anywhere however poorly reinforced regions like temporal or orbital roof are easily penetrated.
- 'Slot' fractures / 'keyhole' defects are caused by the instrument penetrating the skull and often the defect produced leaves an exact impression of the instrument.
- The cause of death is usually due to massive intracranial haemorrhage.

6. INTRACRANIAL (OPEN/ CLOSE)

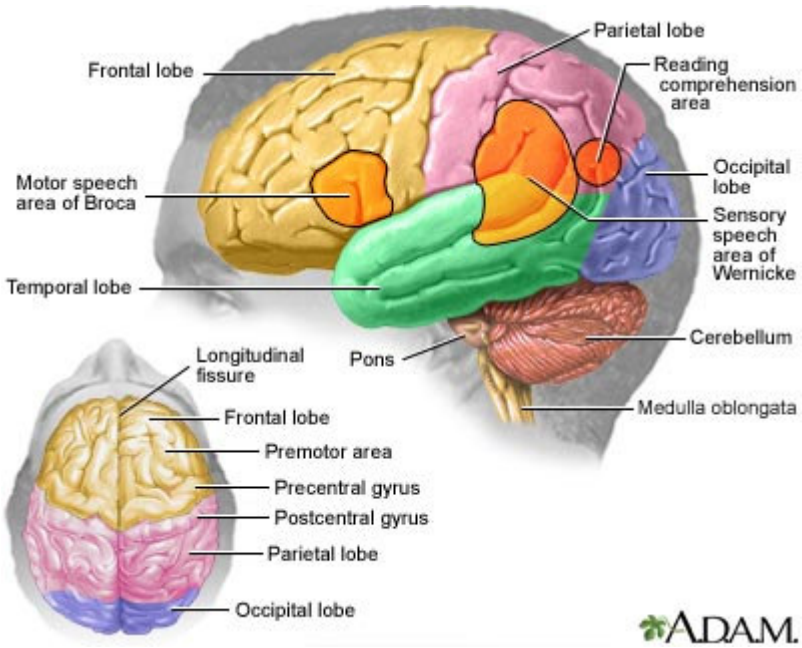


Fig.6.1

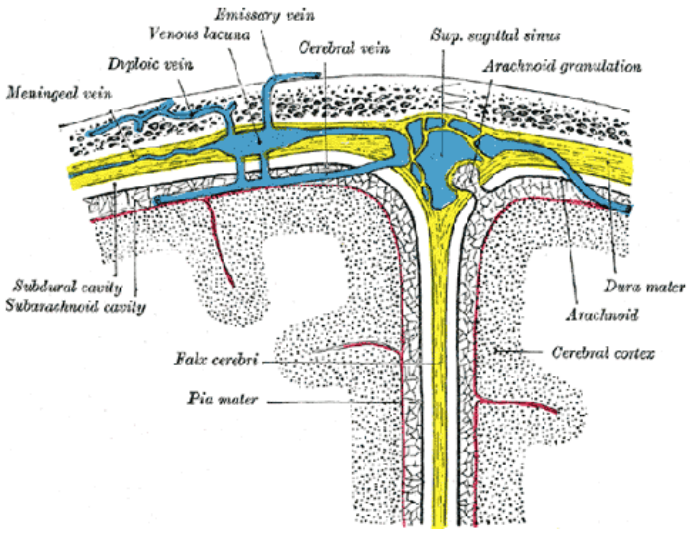


Fig.6.2

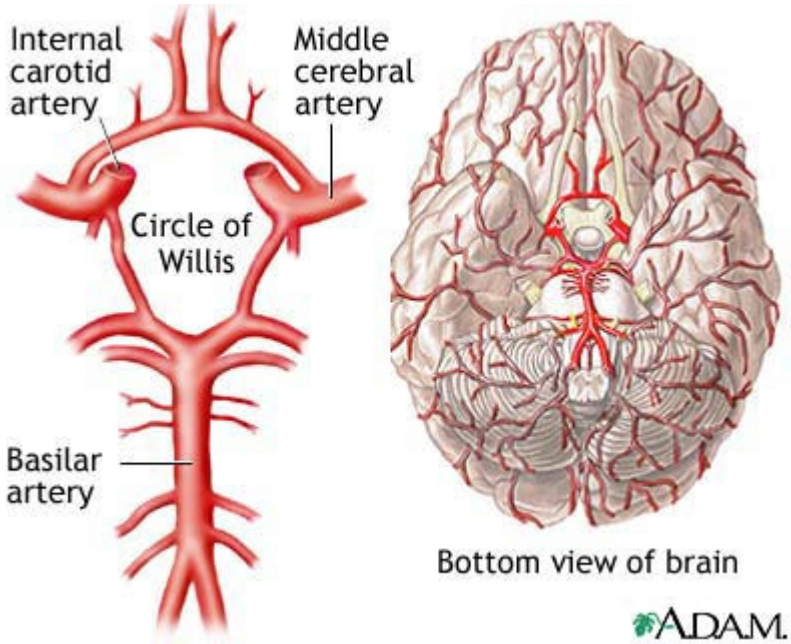


Fig.6.3

NB: Review of anatomy of the brain

EXTRADURAL HAEMORRHAGE/ HAEMATOMA (EDH)

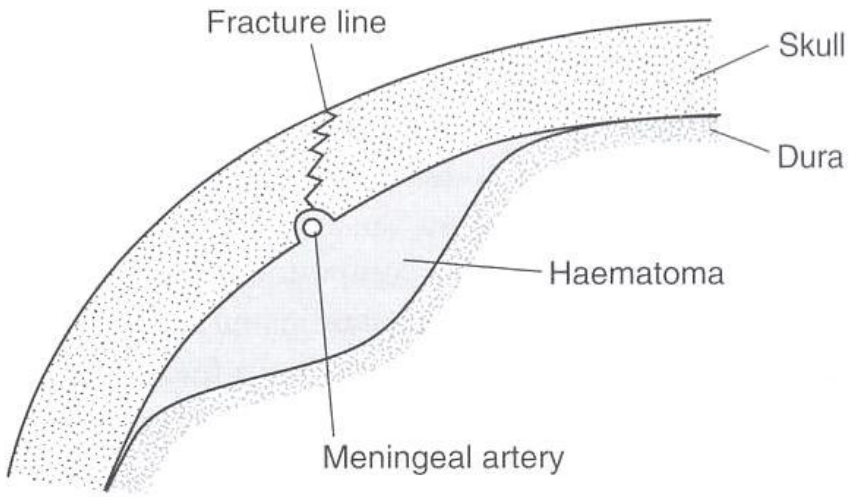


FIGURE 5.27 *Formation of an extradural haemorrhage.*

- An extra- (epi-) dural haemorrhage is situated between the skull and the dura mater and classically is caused by a traumatic rupture of the middle meningeal artery (posterior branch commonly) over the parietotemporal area often associated with a skull fracture over the same area.
- Most cases are of ***arterial*** bleeds than venous.

- Unilateral or bilateral.
- Especially in children there can be an extradural haematoma without a skull fracture.
- It is suggested than a minimum volume of 35ml-100ml of haemorrhage is associated with fatalities.
- Is the least common of the three types of brain membrane haemorrhage.
- An EDH is always the result of an injury and is found in persons involved in MVA's, assaults and in babies with a haemorrhagic tendency after birth.
- The “classical” picture of brief loss of consciousness at time of injury followed by a “lucid” interval is so frequently absent that no diagnostic reliance can be placed upon it.
- Insult---concussion ± epidural bleed---recovery---significant accumulation of epidural---coma/death
- **BURN EXTRADURAL HAEMATOMA:** Has a chocolate-brown, honey-comb appearance and is friable in consistency. Usually diffuse over a wide area, especially over the crown and both cerebral hemispheres. The carboxyhaemoglobin in the burn haematoma is little or absent if a victim suffered a head injury before the fire started.

SUBDURAL HAEMORRHAGE/ HAEMATOMA (SDH)

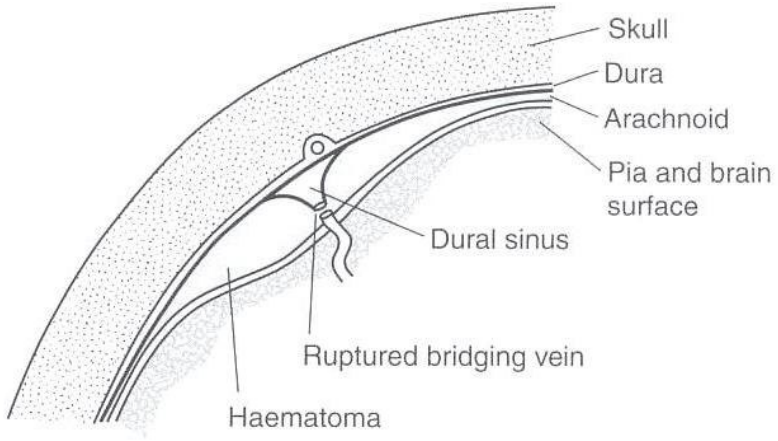


FIGURE 5.32 *Formation of a subdural haematoma.*

- A SHD is situated between the dura and the arachnoids and is always associated with trauma, also more common than EDH.
- The mechanical cause is a change of velocity of the head, either acceleration or deceleration, almost always with a rotational component.

- Subdural bleeding arises from shear stresses in the upper layers of the cerebrum, which moves the communicating veins laterally sufficiently to rupture their junctions at either the cortical ***veins*** or the sinus surfaces.

- In contrast to an extradural haemorrhage the gyri and sulci patterns on the side of the subdural haemorrhage is well preserved whilst there is marked flattening of the convolutions in the contra-lateral hemisphere

- Unlike an EDH a fracture of the skull plays no part in the pathogenesis of an SDH although a skull fracture is present in approximately 50% of cases of SDH.

- It is almost certain that minor subdural bleeds, insufficient to give rise to any neurological or clinical symptoms or signs other than a transient headache, occur with trivial knocks of everyday life.

- Only when the bleeding is extensive enough to become either a cortical irritant or a space occupying lesion (probably between 35ml and 100ml) does it become clinically apparent.

- The position of a subdural can never be interpreted as a 'contrecoup' lesion and is thus of no use in differentiating a blow from a fall.

- In states of vascular fragility, some minimal trauma precipitates the bleed alas too often forgotten or too trivial to be recorded in the history.

Acute SDH

- Common sequel to any substantial head injury.

- The lesion is often 'pure' being associated with a closed head injury where the only other signs maybe scalp bruising BUT it might also be part of a complex that includes subarachnoid bleeding, cerebral laceration and contusions, especially in open head injuries or comminuted skull fractures.

Chronic SDH

- This occurs more often in the elderly and may present weeks to months after a trivial injury.

- A chronic SDH arises from the acute lesion which becomes encapsulated by fibrous tissue after a period of time.

- It may be asymptomatic (haemorrhage resorbed) or it may cause symptoms due to enlargement of the haematoma.

- The mechanism of the enlargement is controversial. One common explanation, which seems the most reasonable, is that it occurs from repeated further bleeding, perhaps from new blood vessels that penetrate the mass as part of the healing process**.**

The dating of a subdural haemorrhage

- The age of the SDH can be estimated by macroscopically but histological analysis is more accurate.
- In the early stages,the blood will be liquid.
- After a few days, a clot forms and the EDH will become orgainsed and adhere to the dura.
- Haemolysis of the EDH then occurs which results in reddish-brown discolouration of the dura.
- After approximately three weeks,a macroscopically recognisable fibrovascular membrane envelops the haemorrhage.

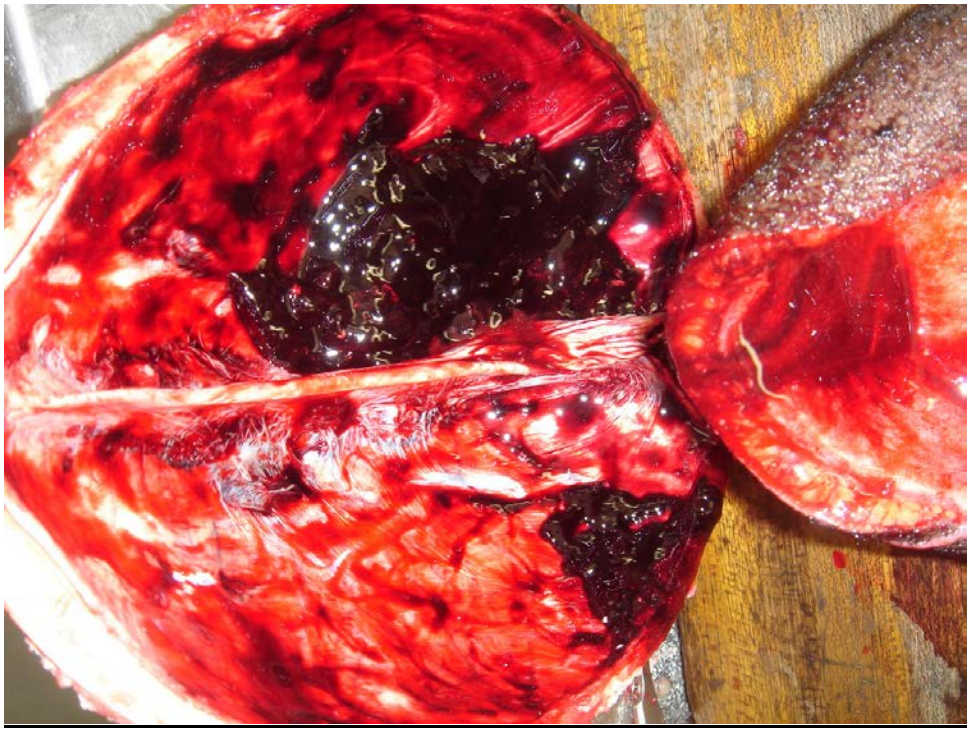
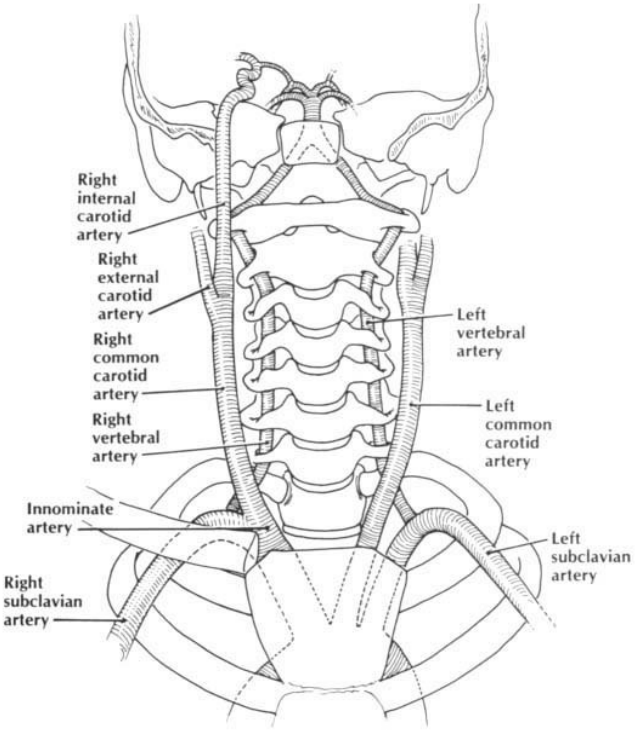


Fig.6.4. Acute SDH

SUBARACHNOID HAEMORRHAGE (SAH)



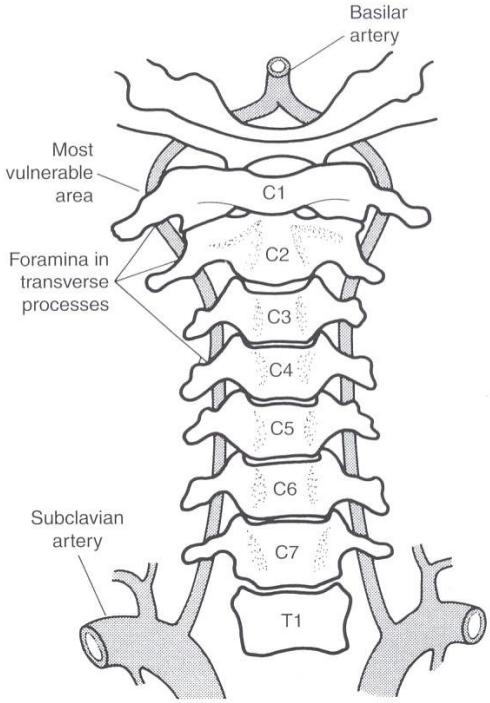


FIGURE 5.38 *The origin and course of the vertebral arteries. They arise from the subclavian arteries at the level of the sternoclavicular joints and ascent via the foramina in the transverse process.*

- SAH lie between the arachnoid and the pia mater and traumatic SAH are more common than the SDH or EDH. The most common cause of a SAH is a ruptured berry aneurysm.
- Whereas extradural and subdural haemorrhages rarely result from anything but trauma, subarachnoid haemorrhage frequently occurs spontaneously.

Traumatic SAH

- It is commonly associated with other head injuries.
- In order to justify a diagnosis of traumatic subarachnoid haemorrhage, there should be objective evidence of mechanical violence other than that afforded by the presence of blood in the subarachnoid space.

Cause of traumatic SAH:

- Direct blunt impact injury associated with cortical contusions
- Lacerations
- Shear stress and rotational movement of the brain tearing the bridging veins leave the cortex and penetrating the arachnoid.
- Blows to the side of the neck resulting in tearing of the vertebral arteries near the foramen magnum may lead to a fatal SAH at base of brain.
- From intracerebral haemorrhages rupturing into the subarachnoid space.

'Atraumatic'/ Spontaneous SAH

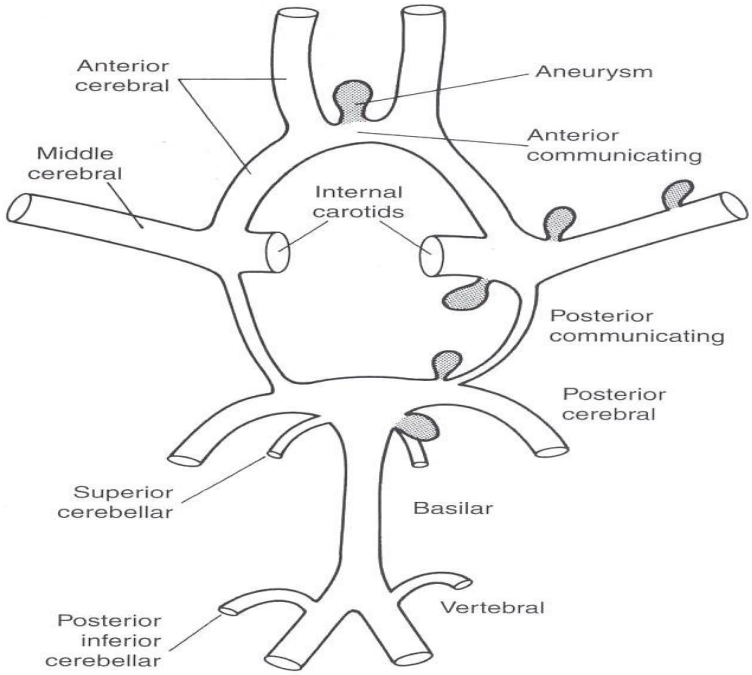


FIGURE 5.37 *Cerebral arteries ('circle of Willis') and common sites for aneurysms.*

Pathology of 'spontaneous' SAH

- Often an extensive basal SAH with/ without clots, extending into the basal cisterns and may, depending on the severity, cover the whole surface of the brain.
- The source of the haemorrhage is often difficult to locate.

Causes of 'spontaneous' SAH

- Rupture of vascular malformations.
- 'Congenital Berry" aneurysm (see figure above)
- Rupture of atherosclerotic aneurysms
- Rupture of syphilitic aneurysms
- Rupture of mycotic aneurysms
- Haemangiomas

Mechanism of death in SAH

- The immediacy of death is sometimes surprising, especially in association with the trauma population, unlike a victim of a spontaneous rupture of a berry aneurysm which may resolve or which may progress over hours or days to coma and death.
- The sudden deaths from SAH may be due to:
    - Sudden irrigation of the brainstem by blood (brainstem irritant) leading to a rapid cardiorespiratory failure ***or***
    - Widespread vasospasm which may affect the vital centres

ALCOHOL AND SAH

- Substantial subarachnoidal haemorrhages can be found after head injuries sustained by people under the influence of alcohol.
- Alcohol may contribute or cause a fall or some traumatic event which may lead to a rupture of an aneurysm.

MEDICO-LEGAL IMPLICATIONS OF BRAIN MEMBRANE HAEMORRHAGES

- A "Lucid interval" can occur in EDH and SDH. This is has important implications in medical negligence cases
- The dating of SDH's is not specific and can only give a broad idea of time: days/weeks/months
- Localisation of the impact site may not be accurately ascertained using the location of the heamorrhage alone
- SAH's can be spontanoeus or traumatic
- Clinically, the symptoms of head injuries may resemble acute alcoholic intoxication and often the two go together.

INTRACEREBRAL HAEMORRHAGE/ HAEMATOMA

- Common in severe head injuries but can occur in natural disease.
- Primary (at time of impact/ soon afterwards) **or** secondary (caused by changes in ICP or bleeding into infarcts caused by vascular damage).
- Can be due to coup or contrecoup mechanisms and can be found anywhere within the hemispheres.
- May rupture through the cortex into the meningeal spaces forming a '**burst lobe**'
- It might be difficult to distinguish between 'natural' vs. 'traumatic' intracerebral bleed, especially in older subjects with co-morbidities like HPT and/or cerebral atherosclerosis.

- It is very important to conduct extensive histological examinations of the brain surrounding a 'natural' intracerebral haemorrhage so that an underlying pathology can either be confirmed or excluded.

TRAUMATIC VS NATURAL INTRACEREBRAL HAEMATOMAS

Intra-cerbral hemorrhage	Cause Natural	Cause Traumatic
Patient's age	Elderly	Any age
Number	Single	More than one
Associated pathology	Athersclerosis Tumour Infarction	Absent unless secondary
Site	Deep-seated and in certain areas e.g. basal ganglia	Near the surface of the brain

PRIMARY BRAINSTEM HAEMORRHAGES: That occur at the time of injury. HPT haemorrhages can occur without trauma in the midbrain, especially on the Pons. These tend to be large, usually with ragged rim of white matter around the periphery.

Traumatic haemorrhages in the brainstem are often well-circumscribed lesions, sometimes rounded, which lie laterally in the tegmetum, the shape of the midbrain being undistorted. The typical site is between the aqueduct and the outer end of the substantia nigra. They are usually associated with occipital impacts and the victim is often unconscious from the time of the injury.

CEREBRAL CONTUSIONS

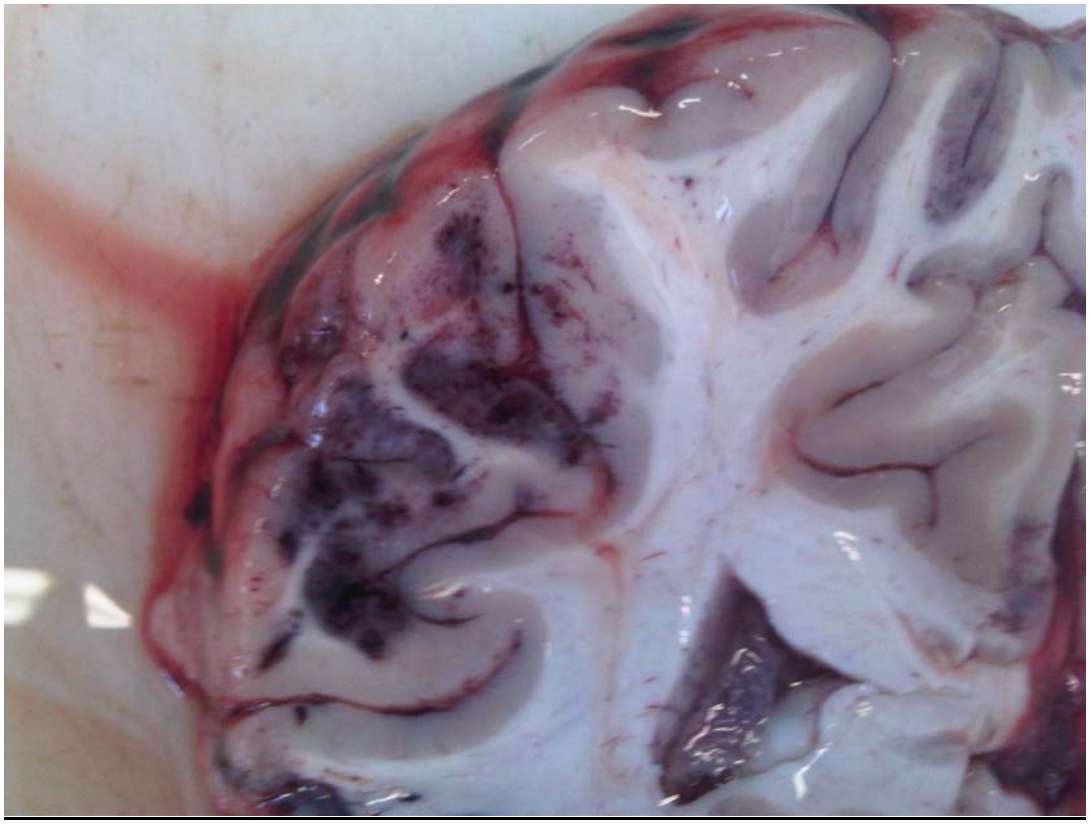


Fig.6.5. Cortical contusions

Types of contusions

- **Cortical contusions**
- **Coup and/or Contrecoup contusions**
- **Fracture contusions**
- **Herniation contusions**
- **Parasaggital contusions (gliding contusions)**

Medico-Legal significance of contusions

- A contusion is a direct consequence of trauma.
- It is necessary to differentiate between a cortical contusion and a cortical infarction or necrosis
    - cortical infarctions/necrosis are not limited to the upper part of a gyrus (the crown); the cortical band is affected and changes are present in the depth of a sulcus.

CEREBRAL LACERATIONS

- A cerebral laceration breaches the pia mater-Arachnoid layers (the leptomeninges) and the underlying brain is torn.
- As lacerations seldom if ever occur alone but invariably together with contusions, the terms "contusion" and "laceration" are generally not separated and hence the term "contusion/laceration" is preferred.
- The results of a laceration are precisely the same as those of a contusion and all considerations in respect of the latter are valid in the case of a laceration.

PONTOMEDULLARY RENT

- This is a tear in the brainstem between the pons and medulla and in severe cases complete avulsion. Possibly the only type of primary injury to the brain stem that may occur in isolation.
- **Mechanism:** Severe hyperextension of the head during high acceleration.
- **Associated injuries:** Ring fracture; Dislocation and/or fracture of C1 and C2.

MULTIPLE PETECHIAL HAEMORRHAGES

- These are seen in patients who die very soon after a head injury; death is usually within a few hours
- The lesions can be seen macroscopically but are usually better visualised microscopically where they take the form of perivascular haemorrhages
- They can occur in any part of the brain but particularly in the white matter of the anterior parts of the frontal and temporal lobes, the brain stem and adjacent to the thalamus.
- The pathogenesis of this type of brain damage has not been conclusively established but it is thought that acceleration/deceleration forces selectively damage arterioles, capillaries and venules in the brain at the time of injury.

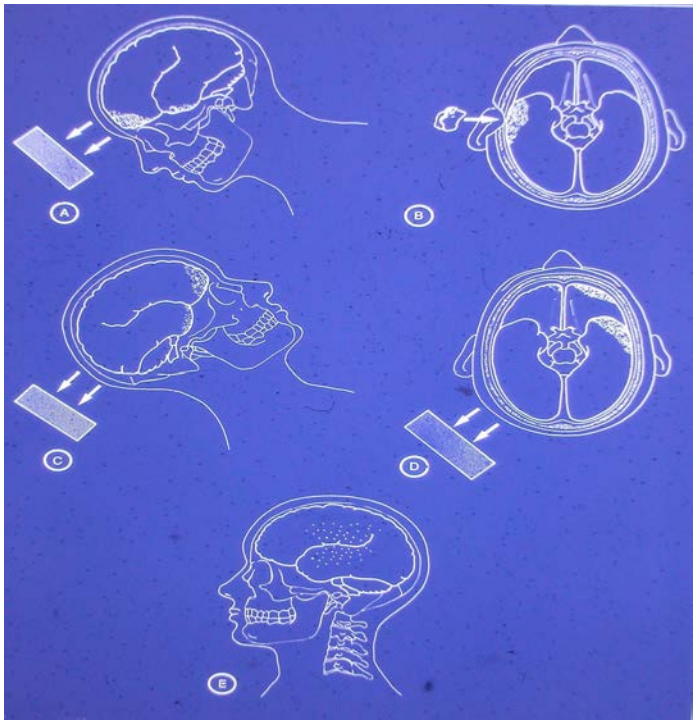


Fig.6.6

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, the so called '***Coup'*** lesion. **Fig.6.6 (a)(b)**
- 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 head, the so called '***Contrecoup***' lesion. **Fig.6.6 (c)(d)**
- **The mechanism of the lesions has been contervential since 1799 at the famous Paris meeting.** (see **BK p207-p209**)
- **In summary:**
    - 'Contrecoup' contusions occur where the brain glides over the irregular, jagged contours of the skull interior and are usually more severe than the corresponding coup-type contusions.
    - Negative suction pressures which develop opposite the point of impact are also involved in their causation.
    - Great contrecoup force may also lacerate the brain surface.

PRIMARY DIFFUSE BRAIN INJURY

- Diffuse brain damage exists in 4 principal forms: ***Diffuse vascular injury, Diffuse axonal injury, Hypoxic brain damage and Diffuse brain swelling.***
- ***Diffuse vascular injury*** see multiple petechial haemorrhages above.
- ***Diffuse axonal injury:*** Damage to axons of any aetiology which traumatic axonal injury (TAI) is but one of the examples. It is primarily a non-impact rotational acceleration-deceleration phenomenon, deformation by stretching probably being the most significant factor.
- TAI is at the severe end of a spectrum of primary diffuse brain injuries that ranges from mild concussion. The types of injury have the same pathophysiological basis but the degree of injury sustained varies.

Cerebral concussion

- Clinical entity
- 'a transient paralytic state due to head injury which is of instantaneous onset, does not show any evidence of structural cerebral injury and is always followed by amnesia from the actual moment of the accident' ***Trotter (1914)***
- Rate of change of velocity of the head is important in producing a concussion, the threshold being atleast ≥ 8,5m/s.
- With or without structural brain damage like contusions.
- True concussion may last for seconds or minutes.
- Occasionally what appears to be simple concussion proves to be fatal, causing respiratory paralysis, though at autopsy no significant lesions are found.

Traumatic axonal injury (TAI)

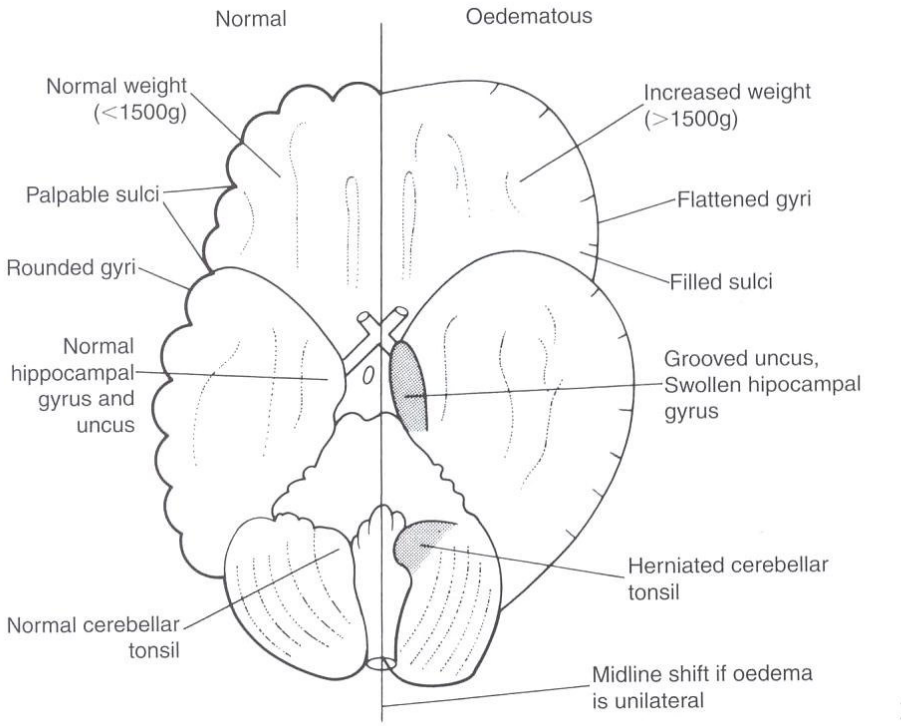
- **'**damage to axons caused by trauma, which may vary from small foci of axons to more widespread brain damage, diffuse TAI is the most severe form of traumatic axonal damage' **BK**
- **Mechanisms of TAI:**
    - The damage to the axons can be attributed mainly to shear and tensile strains at the moment of injury.
    - It is associated with angular/rotational acceleration or deceleration.

- There may be nothing to see on the surface of the brain on slicing at autopsy.
- Diagnosis usually requires expert neuropathological examination using special stains (**βAPP**) to demonstrate subtle microscopic damage to nerve fibres (**axonal bulbs/ retraction balls or globes**).

- There are 3 distinct structural anormalities in the pathology of **TAI**: ***Corpus callosum lesions, Brainstem lesions and Diffuse axonal damage.***

7. SEQUEL OF BRAIN INJURY

fig.6.7



A. CEREBRAL SWELLING

- May be a local phenomenon around almost any lesion, be it contusion, laceration, tumour or infarction.
- Can also be diffusely unilateral or bilateral.
- This swelling is extremely common after substantial head injury, especially in children and it can be the sole pathological entity leading to death in these young victims.
- Macroscopic features at autopsy include narrowing of the sulci, flattening of the gyri and a reduction in the size of the ventricles.
- The swelling may become severe enough to raise the intracranial pressure and thus lead to the sequele of raised intracranial pressure

- **Causes are not always clear** but are thought to be due to an increase in the ***cerebral blood volume*** (Congestive brain swelling) and/ or an increase in the ***water content of brain tissue*** (Cerebral oedema)

- Congestive brain swelling: Dilatation of the cerebral vessels as a result of ***hypoxaemia***, ***hypercapnia*** and/or marked increase in the ***intraluminal arterial pressure***.

- **Cerebral oedema:**

    - It can be defined as an abnormal accumulation of fluid in cerebral parenchyma which causes volumetric enlargement of the brain, with resulting raised intracranial pressure and the secondary changes which this causes.

    - **Cause of cerebral oedema** include ***trauma, infection, vascular occlusion*** and ***hypoxia***.

    - 3 principal varieties of brain oedema: ***Vasogenic, Cytotoxic*** and ***Hydrostatic oedema.***

B. <u>RAISED INTRACRANIAL PRESSURE</u>

- **Causes:** Brain swelling; Space occupying lesions.

- **Mechanisms of development**

    - Time plays an important role in the consequences of raised intracranial pressure

    - If the displacement is slow - weeks to years- eg slowly growing neoplasma, chronic abscesses, chronic sub-dural haematomas, there is a certain degree of adaptation (rember ***Munroe-Kelly Doctrine***)

    - If the displacement is more rapid- days to weeks eg rapidly increasing neoplasms, acute abscesses, acute intracranial haematomata,there are severe effects due to herniation of brain through the narrow spaces and early arterial occlusion.

    - In the case of 'abrupt' displacement – seconds to minutes- eg basal subarachnoid haemorrhage from ruptured cerebral aneurysm, the sudden stretching of the arteries may result in vasospasm and occlusion which causes ischaemia in the areas of the brain supplied by these arteries.

- **PM findings**

    - Cerebral swelling causes a diffuse flattening

    - Flattening of the convolutions and narrowing of the sulci which can be: ***Localised/ Diffuse*** or ***Ipsilateral/ Contralateral***.

    - In an acute subdural haemorrhage the convolution of the opposite hemisphere will be flattened.Those on the same side as the haemorrhage will remain unaltered.

- In the event of an EDH and an encapsulated chronic SDH, the flattening will occur on both sides - the haemorrhage pushes the brain against the skull of the opposite side and also pushes the tough firm dura against the gyri of the same side indenting it.

- **Consequences of raised intracranial pressure**
  - ***Brain herniations***
  - ***Cranial nerve injury***
  - ***Secondary brainstem haemorrhages (Duret Haemorrhages)***
  - ***Secondary infarctions of the brain***
  - ***CSF Obstruction***
  - ***Brain death***

C. <u>HYPOXIC/ ISCHAEMIC BRAIN DAMAGE</u>

- After sustaining a head injury it is accepted that intracranial arterial spasm occurs
- Precisely where the hypoxic/ischaemic damage occurs is not certain but it is safe to say that it occurs fairly soon after injury but not at the moment of injury
- Other causes of hypoxic/ ischaemic brain damage include:
  - profound systemic hypotension
  - cardiac arrest when resuscitation has not been sufficiently expeditious
  - status epilepticus
- This is mainly a histological diagnosis
- Most hypoxic conditions in forensic pathology cause death too early for histological detection - acute deaths from e.g. strangulation, choking, suffocation etc.
- When longer periods of survival follow the acute incident (probably a minimum of two to four hours' survival), it may be possible to detect these hypoxic or ischaemic histological changes.
  - The whole brain is fixed in forminaldehyde for histology and/or immunohistochemistry.
  - The true extent and severity of this type of brain damage can be identified and its distribution determined histologically on representative bilateral sections of the brain.
  - Certain parts of the brain are selectively vulnerable to hypoxia/ischaemia and are sampled for histological analysis.
  - The vulnerable areas include:
    - Hippocampus (Sommer Sector of Ammon's Horn);
    - Basal ganglia
    - Cerebellum (Purkinje cells)
    - The 'watershed areas' of the cerebral cortex
      - These are areas of the brain that are supplied with blood from the smallest peripheral branches of the main cerebral arteries hence receive lower levels of oxygen.
      - These areas are located between the anterior and middle cerebral arterial territories
      - the lateral part of the occipital lobe in the common boundary

zone
    - Between the middle and posterior cerebral arterial territories.
  - In severe cases small wedge-shaped infarcts may develop. This process can be widespread (diffuse) but is not completely uniform, e.g. clusters of damaged cells may be found next to unaffected cells.

- **Pressure necrosis:** Occurs in situations where hypoxic/ ischaemic injury follows brain injury. The lesion is mainly in the grey matter but more often, it looks like a focal infarction involving the white matter as well. These lesions occur in a patchy districution and are not confined to arterial territories.

D. <u>INFECTIONS</u>

- **Meningitis**
- **Brain abscess**

Differenciate between traumatic source vs infection of natural origin. Microbiological examination in all cases.

E. <u>NEUROLOGICAL SEQUELAE</u>

- Are varied and include: ***posttraumatic epilepsy, persistent vegitative state, paralysis, paresis, psychoneurological symptoms*** and ***various cognitive deficits.***

F. <u>ENLARGEMENT OF THE PRIMARY HAEMORRHAGE</u>

- May occur leading to a space occupying lesion and complications thereof.

G. <u>TRAUMATIC ANEURYSM</u>

8. SPINAL INJURIES

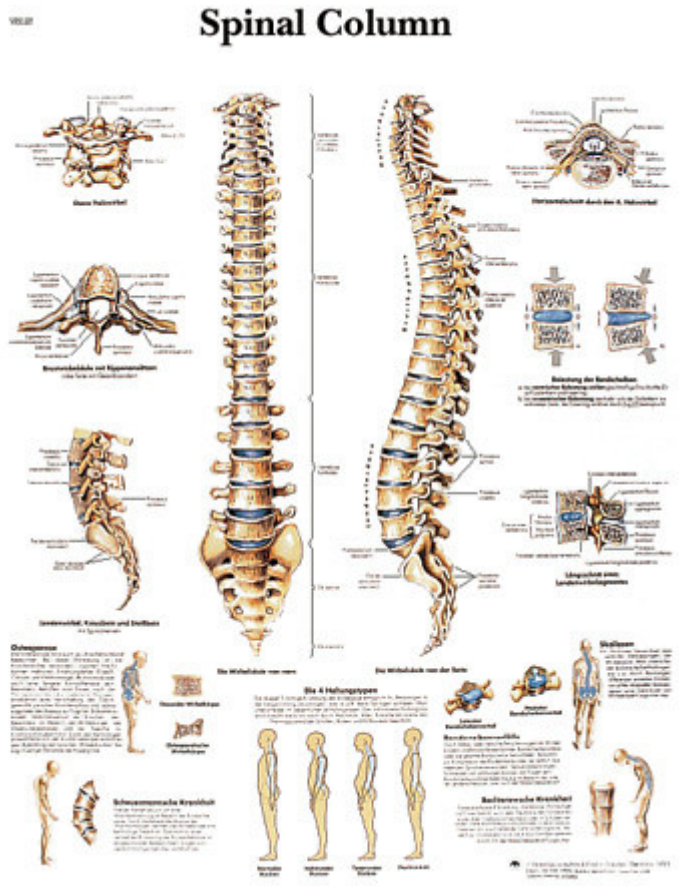


Fig. 8.1

- The head and spine should be imagined, indeed be considered as interelated parts of the same biomechanical system in relation to trauma.
- The mechanisms of trauma involving the spinal column could result from: Direct trauma, compressive damage, hyperflexion and hyperextension

Compression injury/ damage

- Occurs when a victim fall from a height either onto his feet or his head.
- When an individual falls on his feet there is an almost axial transmission of kinetic energy from the ponit of contact (feet) which can go as high as the skull. Fractures can result at one or more points along the axis of the spine and can cause a base of skull fracture in a form of a 'ring' fracture.
- Fall onto the head – may cause a 'burst atlas' where the impact of the occipital condyles in an axial direction forces the superior atlantal articulating facets apart splitting the ring of the vertebra.
- Compression fractures most commonly occur in the lower dorsal and upper lumbar zone – T12 and L1.

Hyperflexion and hyperextension injury

- Hyperextension is more dangerous in causing spinal damage. Why? Possibly because flexion is protected by contraction of the stronger posterior neck muscles.
- A great majority of these injuries commonly occur in frontal deceleration or rear acceleration impact motor vehicle accidents.
- Whatever the cause a myriad range of lesions can occur involving: Cervical, Thoracic and Lumbar segments of the spinal column. Commonly the cervical segment, with the upper two cervical vertebrae being the most vulnerable.
- The commonest injury is dislocation of the atlanto-occipital joint.
- Other situations in which hyperflexion and hyperextension injuries can occur:
    - Fall onto the back of the head – anterior dislocation
    - Blows to the jaw or face that jolt the head backwards – posterior dislocation eg fall on the face from a height(like stairs)
    - Violent movements of the head on the neck – odontoid peg fractures.
    - High speed motor vehicle or railway accidents – the cord may be transected secondary to fractures of the spinal column.

Spinal cord injury

- In most cases, damage to the spinal cord results intrision of some part of the spinal column onto the spinal cord.
- Infarction of the spinal cord may occur secondary to damage of the anterior spinal artery.

9. SPECIAL CASES

- **Firearm injury**
- **Stabwound**
- **The newborn child**
- **Lumbar puncture**
- **Injuries by foreing objects**

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