CRANIO-CEREBRAL TRAUMA

GENERAL NOTIONS; CLASSIFICATION

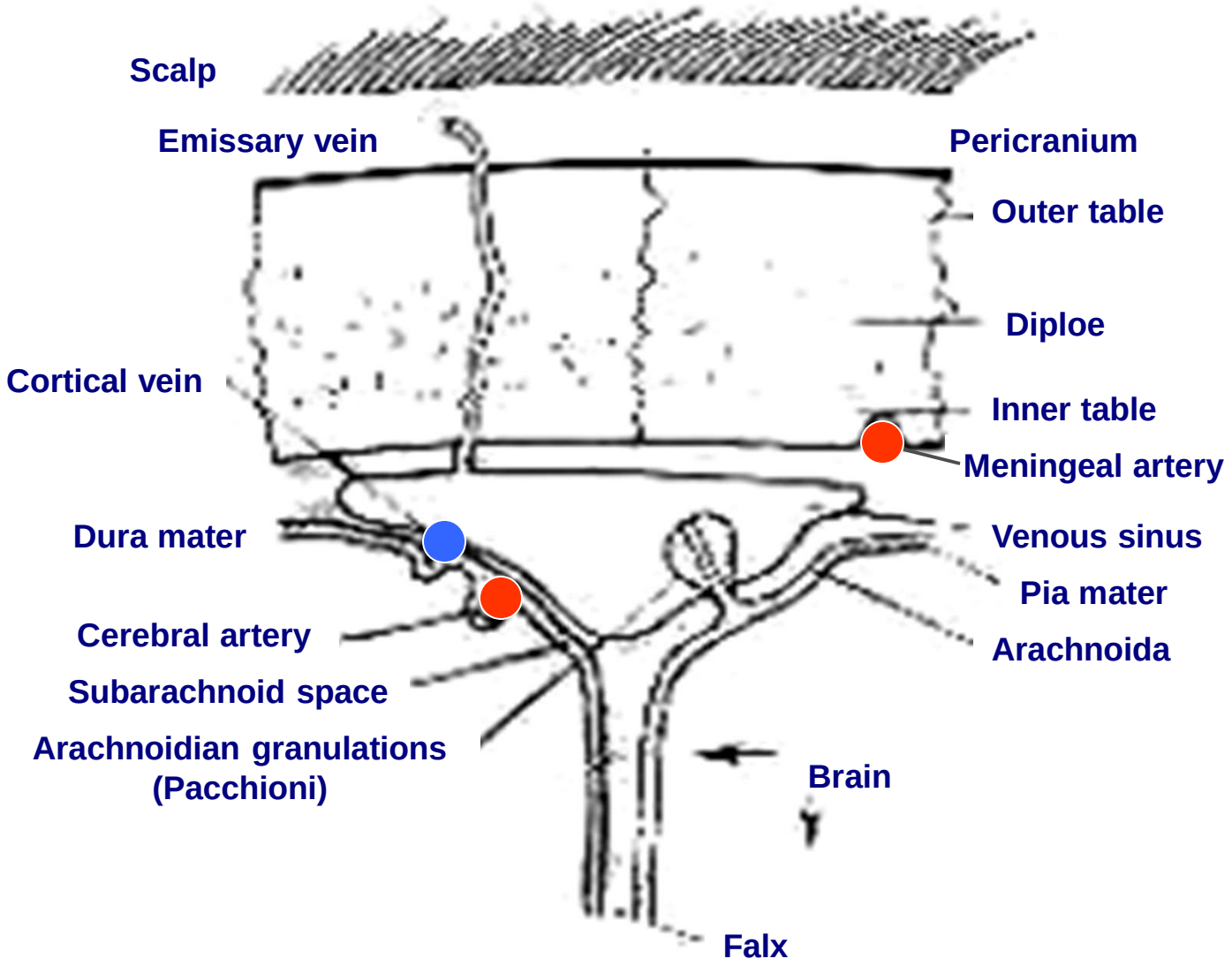
In criminal and accidental injuries, the head is especially vulnerable and an understanding of the mechanism of head injuries is essential in forensic medicine.

The head is the heaviest part of the body relative to its size and is poised on the spine in a rather unstable position, being mainly secured by the tone of the neck muscles.

Though the brain is enclosed within a strong bony cranium, distortion and transmitted forces can use a variety of lesions within the skull, both to blood vessels and neural tissue.

It is most convenient to describe head injuries in sequence from the outside to the interior.

Cerebral traumatic lesions are frequently encountered during forensic autopsies; the interpretation of their involvement in the death generating processes involves a good understanding of their etiologic mechanisms and of the types of structural changes that occur.


Scalp
Emissary vein
Pericranium
Outer table
Diploe
Cortical vein
Inner table
Meningeal artery
Dura mater
Venous sinus
Pia mater
Cerebral artery
Arachnoida
Subarachnoid space
Arachnoidian granulations
(Pacchioni)
Brain
Falx

CLASSIFICATION CRITERIA FOR CRANIO-CEREBRAL TRAUMA (CCT)

a. *Energy transfer mechanism*

- Direct CCT
  - Acceleration
    - linear
    - rotation
  - Deceleration
    - countercoup
  - Compression
    - unilateral
    - bilateral
- Indirect CCT
  - Extracranial impact
  - No impact (whiplash)

b. *Time factor*

CCT
- Acute (recent)
- Subacute
- Chronic (belated)

c. *Severity and Consciousness state (Glasgow Coma Score – GCS)*

CCT

Minor	Moderate	Severe
Consciousness present	Diminished consciousness	Abolished consciousness
Cooperative	Conscious coma	Cortical coma
GCS > 12	9<GCS<12	GCS < 8

d. Topography and aspect of macroscopic lesions

CCT
- Closed - With pericranial lesions
- Without pericranial lesions
- Open - penetrating
- non-penetrating

e. Type and morphology of lesions

CCT with
- Cranial fractures - aligned
- depressed
- Focal lesions - vascular
- cerebral
- Diffuse lesions - vascular
- cerebral

f. Physiopathological consequences

CCT
- Commotion
- Concussion
- Dilaceration

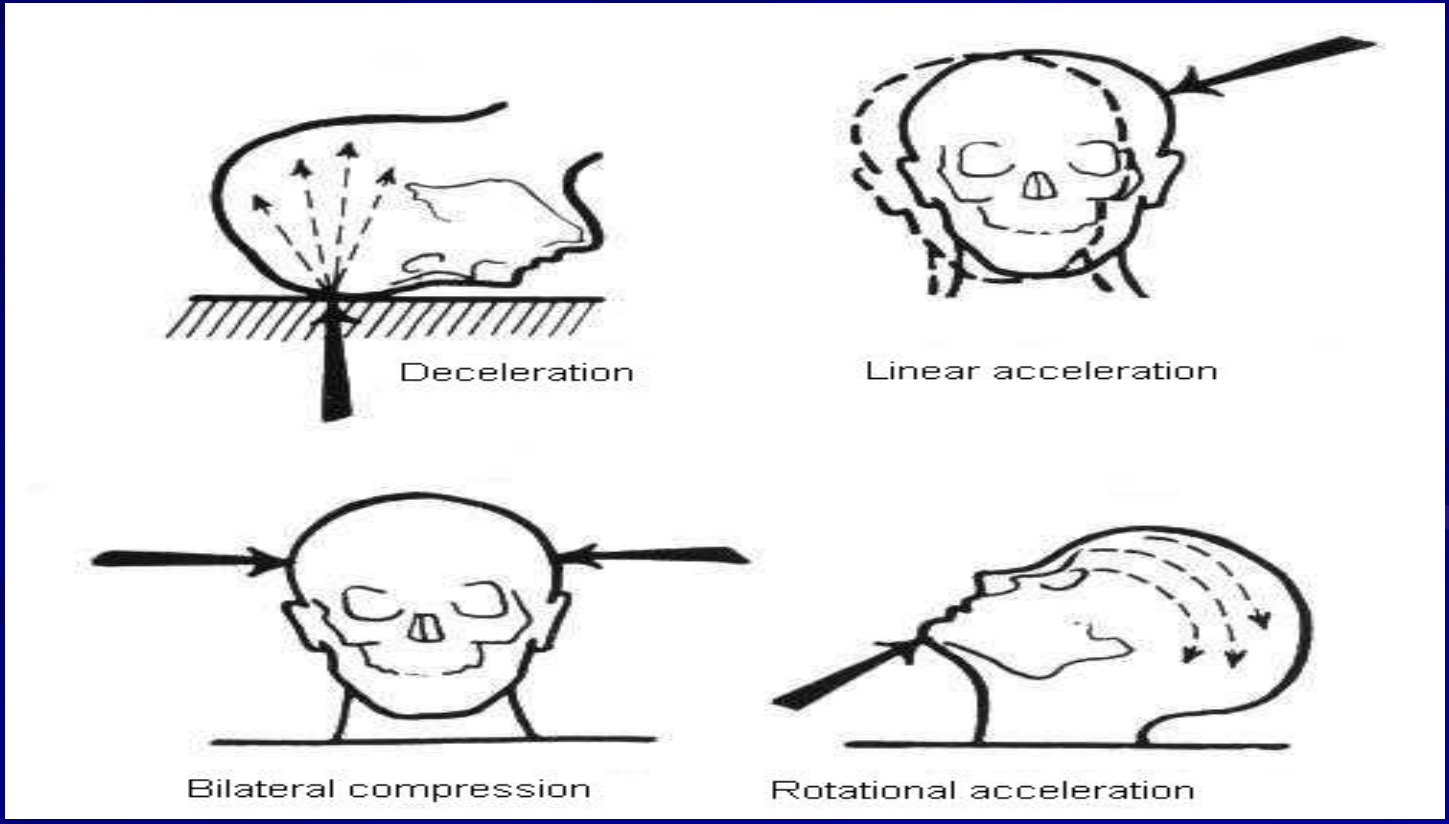
→ Balance disorders
- haemodymanic
- neurodynaimc
- metabolic
- general

GENERAL MECHANISMS

CCT mechanisms are extremely diverse and complex, depending mostly on the type of the generating incident.

These mechanisms can be divided in 4 distinctive categories:

- *linear acceleration*
- *deceleration*
- *rotational acceleration*
- *bilateral compression*



CRANIAL LESIONS

Particularities of the human skull

The specific elements of the human skull are represented by its elasticity, the lack of homogeneity between components, the shape and the position in space.

In the moment of trauma, an inner flexion of the skull takes place at the impact point, accompanied by a compensatory cranial deflexion in the surrounding area.

At the opposite pole a convex cone will appear and underneath it a vacuum area in which the brain is projected by force waves transmitted by the kinetic energy of the impact.

In the centre of the resistance structures situated in the skull base, there is a complex represented by the sphenoid body, the occipital condyles and the resistance of the foramen magnum.

The concept of resistance belt is new; it is used to define the unique structures that incorporate the arches of the skull vault and the rafters of the skull base.

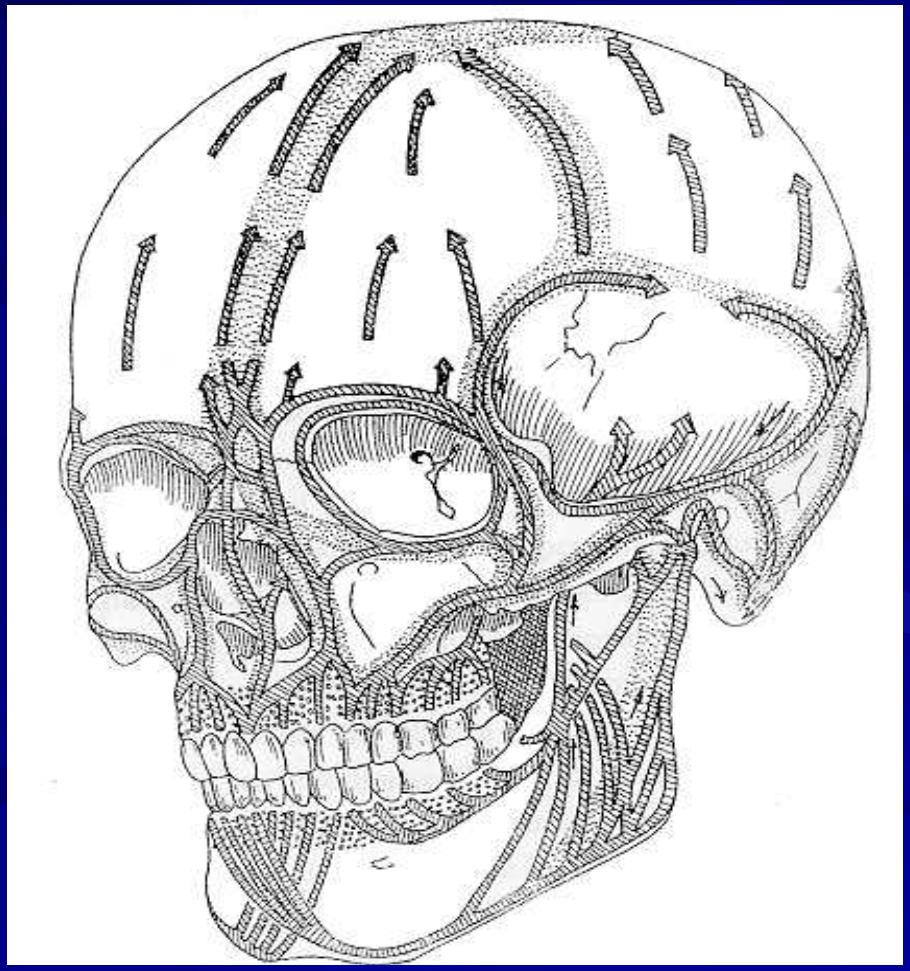
These belts can be grouped in:

- one transversal belt, situated at the limit between the skull base and vault;

- several other belts: sagittal, frontal and oblique (Niculescu).

Felizet described a system of six major resistance areas on the skull base that converge in the basilar apophysis, considered the resistance centre of the skull base.

Cranial traumatisms depend not only on the structural, static characteristics, but also on the dynamic elements, such as intensity, direction and distribution of kinetic energy.



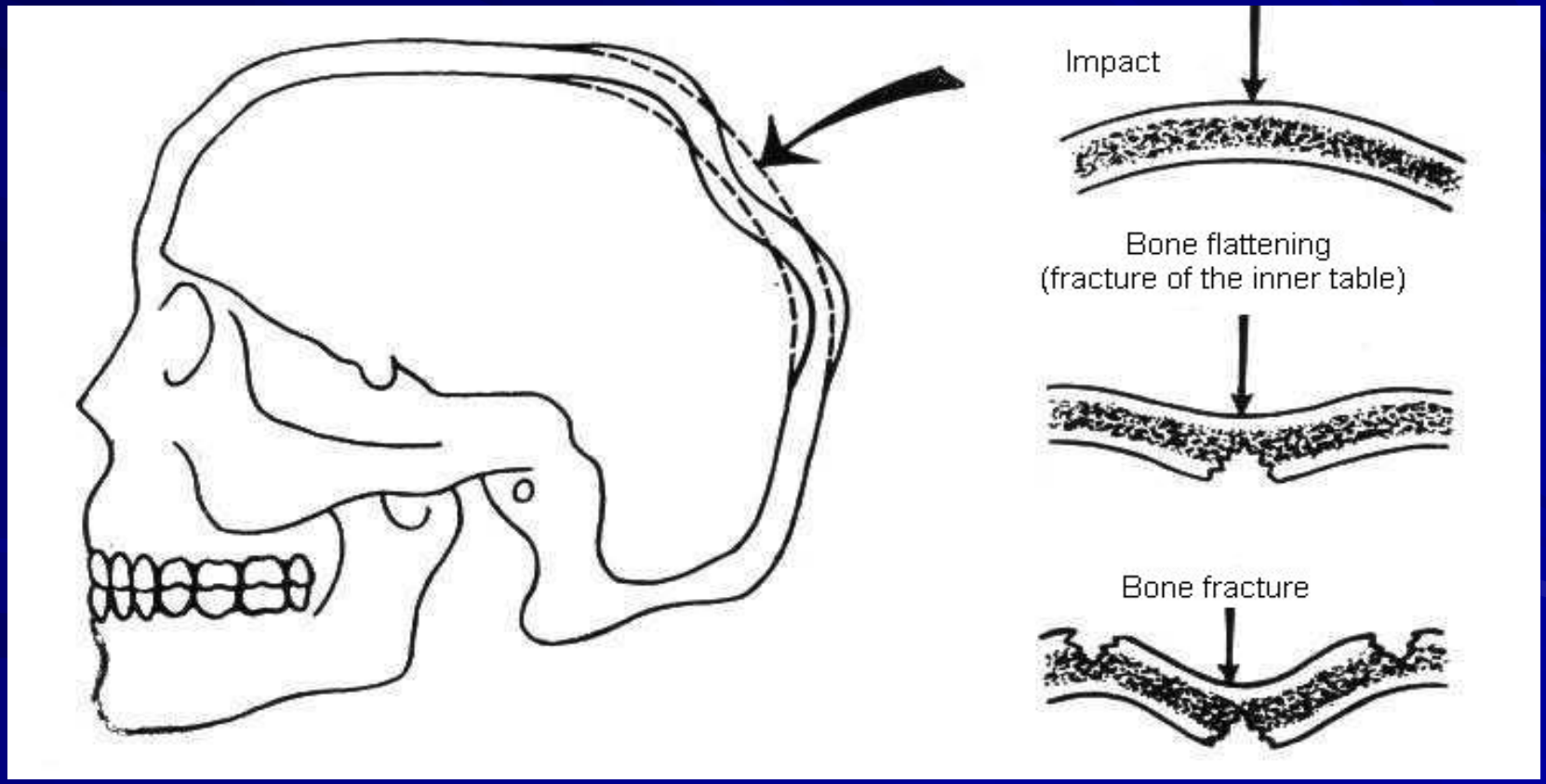
The cranial architecture (Niculescu)

CRANIAL FRACTURES

Cranial fractures are partial or total discontinuities in the skull bones because of direct or indirect traumatisms.

A. DIRECT SKULL FRACTURES

These fractures appear in and are limited to the impact area. Most frequently, the discontinuity appears on the internal table, where the bone convexity is larger so its resistance to impact is lower.



The mechanism of direct cranial fractures

1. Linear fractures

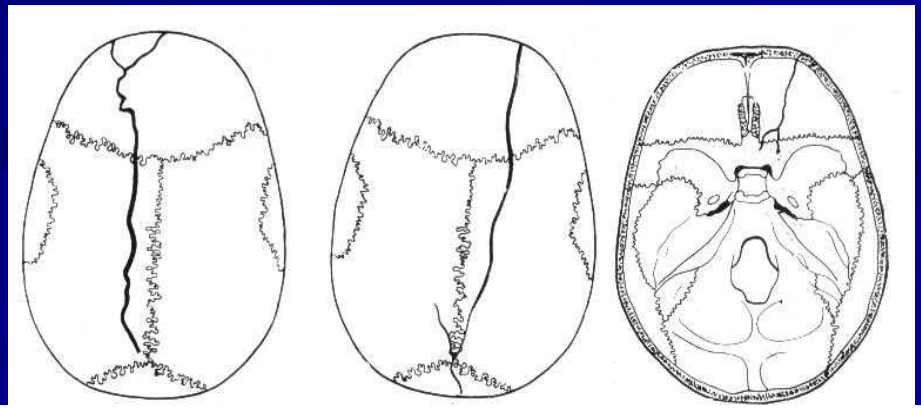
These is the most common and most frequent type of skull fracture.

They are usually unique and have the appearance of a discontinuity that can interest only one of the tables (fissure), or the entire bone structure (fracture line).

The fracture line is generally curved and rarely linear or angular. Along its trajectory, there is escaped blood from the diploic vessels and lakes.

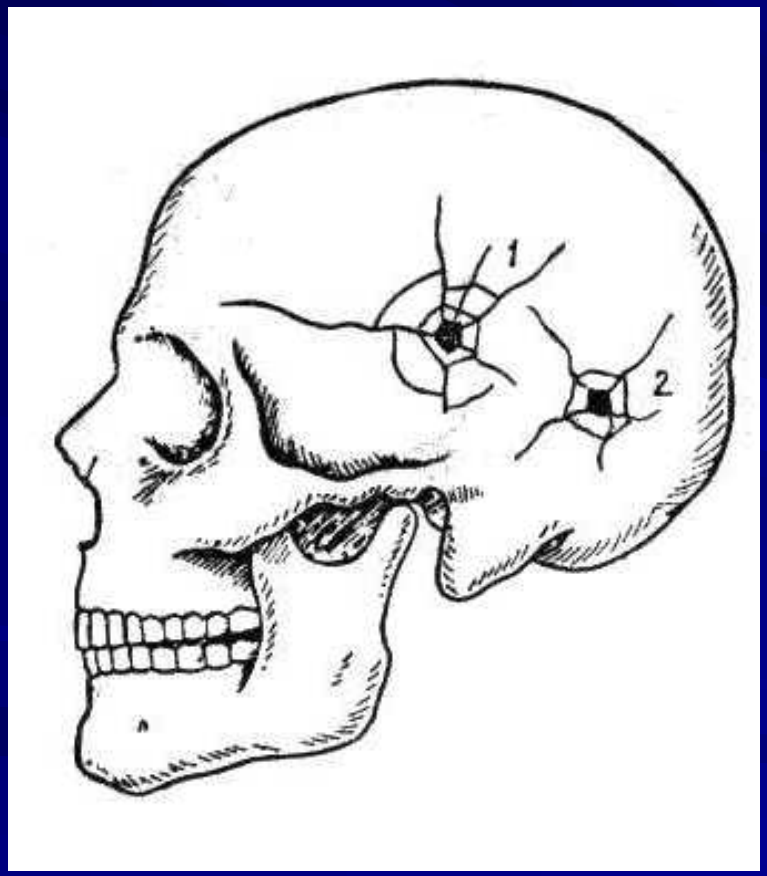
Depending on the characteristics of the traumatic agent and the mechanical features of the skull area, the fracture can ramify.

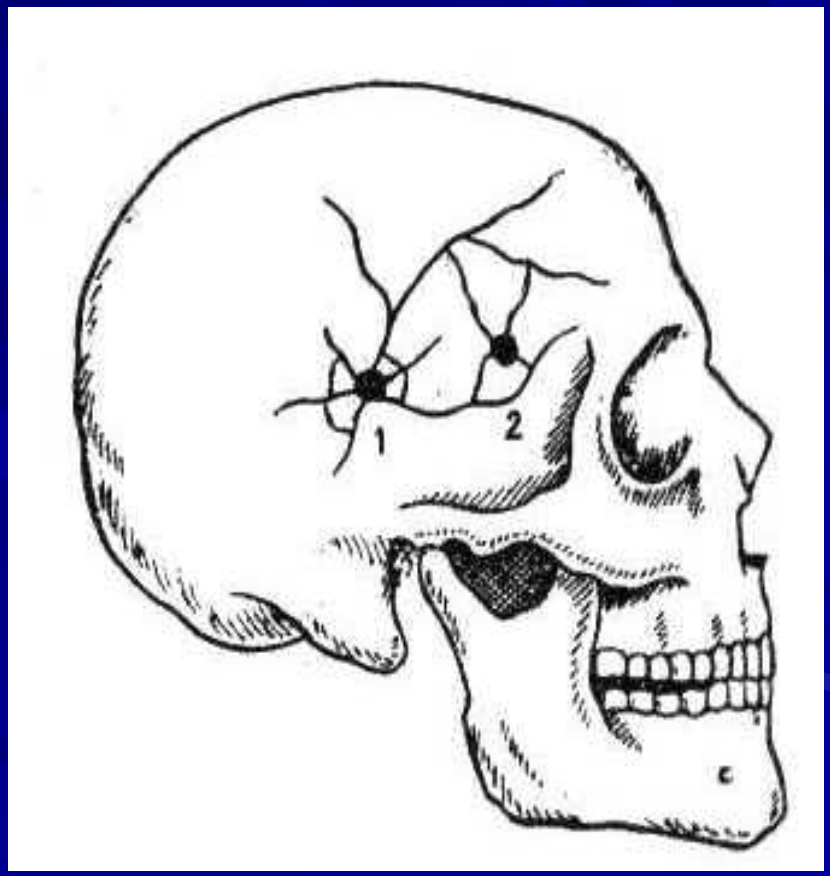
Multiple linear fractures are never crossed. They always end at the contact point, thus establishing the chronology of the impacts - procedure of major importance in legal medicine. A fracture line that ends along the trajectory of another fracture line is ulterior to it. Based on this finding, the succession of the blows to the head can be established.



Circular fractures surround or incompletely circumvent the impact area and always have an equatorial orientation in relation to the impact point.

Linear base fractures are rarely the result of a direct impact. They usually irradiate from a vault fracture, or are mediated (rarely) through other impact structures.





2. Comminuted fractures

Morphologically, the comminuted fractures are defined by the presence of multiple fractures, delimiting bone fragments of various shapes and sizes.

They are more frequent in the anterior half of the skull and are the result of a direct blow with a small surface object, or of a fall on a small convex surface.

Comminuted fractures, like the circular ones, can be uneven, either depression (intrusive) or by extrusion.

Depressed comminuted fractures can have compressive effects on the brain or can determine the brain dilacerations.

Extrusive comminuted fractures are usually the effect of a high-speed penetrating agent, or of skull bilateral compression, with the formation of a cerebral fongus.

Comminuted fractures interest almost the entire skull, (head crush); it appears deformed, irregular; the sound provoked at manual inspection is of "bag of nuts".

Cranial explosions can also occur, due to a hydrodynamic effect.

The bone fragments can dilacerate the scalp and the meninges so the dilacerated cerebral substance can drain in the exterior.

3. Dehiscent fractures

These fractures are characterised by the presence of a free space between the bony fragments.

The prototype is the depressed, "terrace-shaped" fracture; it seems linear but the two fragments are situated at slightly different levels.

Another type of dehiscent fracture is the "gutter" fracture; the two margins of the fracture are oblique towards the discontinuity line.

Dehiscent fractures reproduce relatively well the shape and size of the traumatic agent.

4. Penetrating fractures

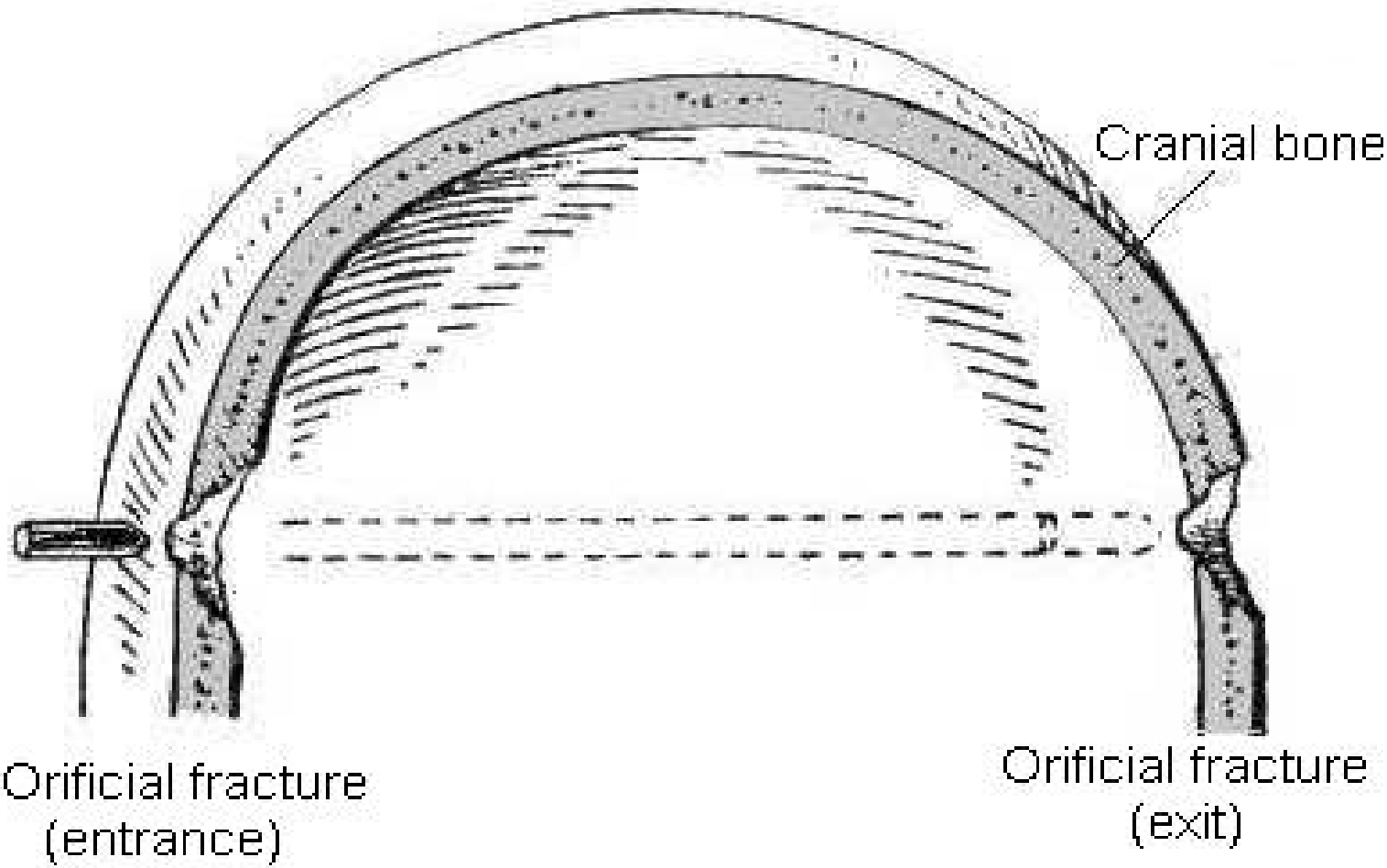
Small agents that penetrate the cranium at high speed (bullets, metal fragments in industry) produce penetrating skull fractures.

The fracture opening can be unique, with the retention of the penetrating object in the skull, or double – one entrance and one exit orifice, each with characteristic features.

Depending on the shape of the agent, the lack of substance can be regular (round, polygonal) or irregular.

A special characteristic is that the margins of the lesion describe a truncated cone, with the base towards the inner table at the entrance orifice and towards the outer table at the exit orifice.

Penetrating fractures are the only type of direct fractures that can interest the base of the skull (direct base fracture).


Cranial bone
Orificial fracture
(entrance)
Orificial fracture
(exit)

5. Particular types of fractures

a. Abrasion fractures appear when a high-speed agent contacts a curved area of the cranial vault. The fracture interests only the outer table and eventually the diploe.

b. The suture disjunction can occur in one or more sutures and can be the result of a traumatic event only until the calcification process is complete. They are more frequent in the coronal and sagittal sutures, rare in the lambdoidal suture and exceptional in other sutures.

c. The extensive-progressive fracture of the child is represented by progressive skull fractures in a closed cranio-cerebral trauma, with cranial vault fractures (without suture dislocation) and meningo-cerebral dilacerations, associated with intracranial hypertension syndrome.

Under these circumstances the initial fracture becomes dehiscent, dura mater is adhesive to the margins of the fracture; the latter is maintained either by the interposition of blood clots or cerebral substance.

d. Mediated fractures

Mediated fractures are rare, intermediated by various extra-cranial structures with the role of transmitting the traumatic force wave to the skull.

- Fractures mediated through the vertebral column occur in falling from heights, with landing on the feet or ischium. The fracture is always situated at the base of the skull and usually is a ring fracture outside the foramen magnum.
- Fractures mediated through the mandible occur when the traumatic energy is transmitted through the mandibular condyles. The fracture is situated around the glenoid cavity, usually comminuted and rarely dehiscent, with condilian depression.

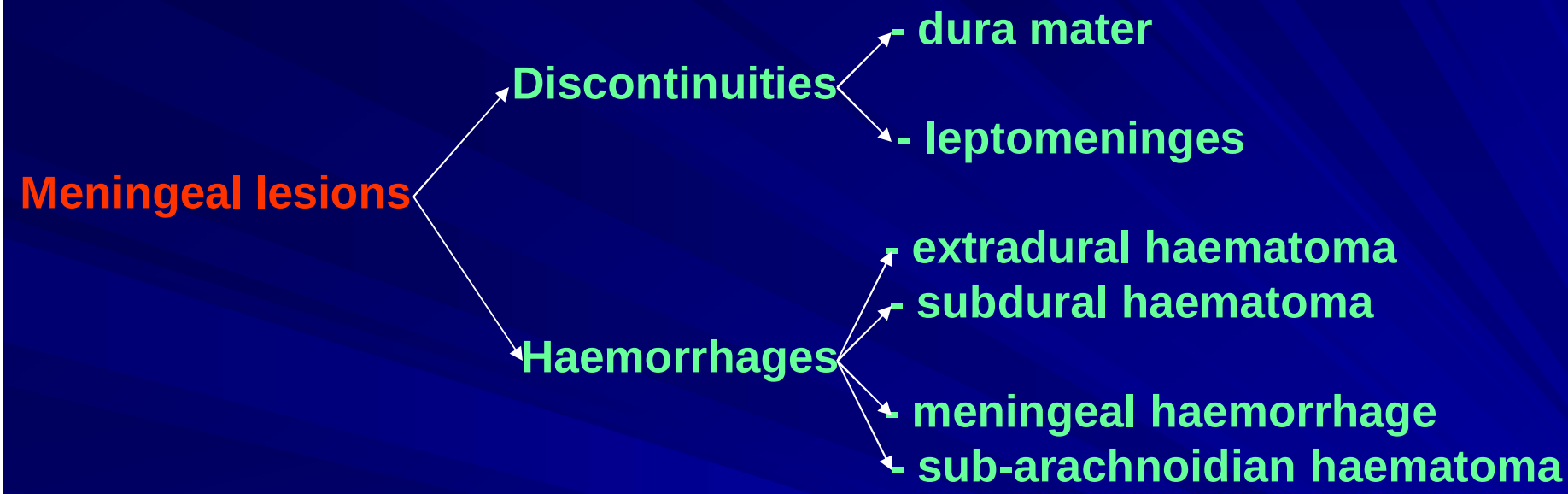
B. INDIRECT FRACTURES

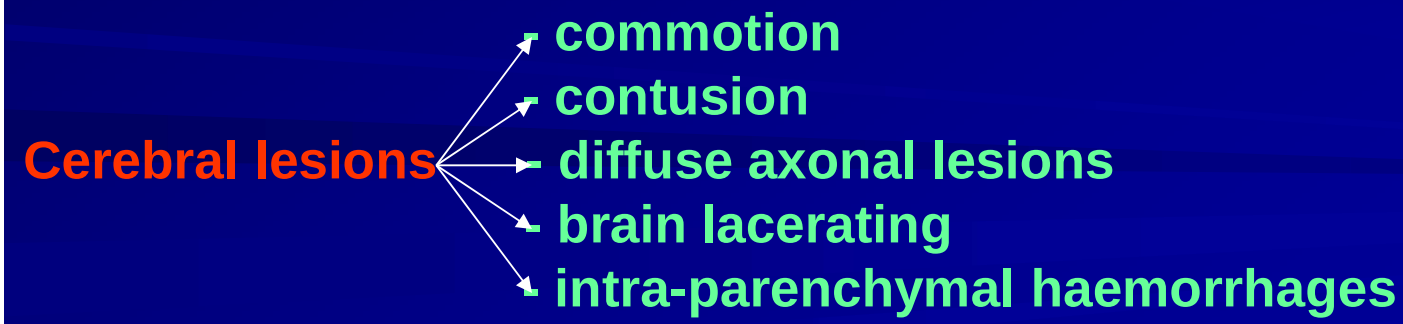
These are all the fractures of the vault or base of the skull that appear at a distance from the impact or deformation area.

They are caused by the general deformations of the skull due to the transmission of impact forces.

MENINGO-CEREBRAL LESIONS

The classification of the meningo-cerebral lesions includes:





1. The extradural haematoma

The extradural haematoma is a delimited blood collection developed between the endocranium and the dura, with compressive effects on the brain and predominantly traumatic aetiology.

The source of bleeding can be either arterial (medial meningeal artery) or venous (superior longitudinal sinus, lateral sinuses, diploic veins – rarely).

The location of the blood collection is either typical, in the temporal fossa, or atypical, affecting any other cranial region, including the posterior fossa.

In rare cases, bilateral, inter-hemispheric or basal extradural haematomas can be encountered.

In its acute form, the extradural haematoma is a mass of blood clots and blood. The clots are brown-reddish or blackish, adhesive to the dura.

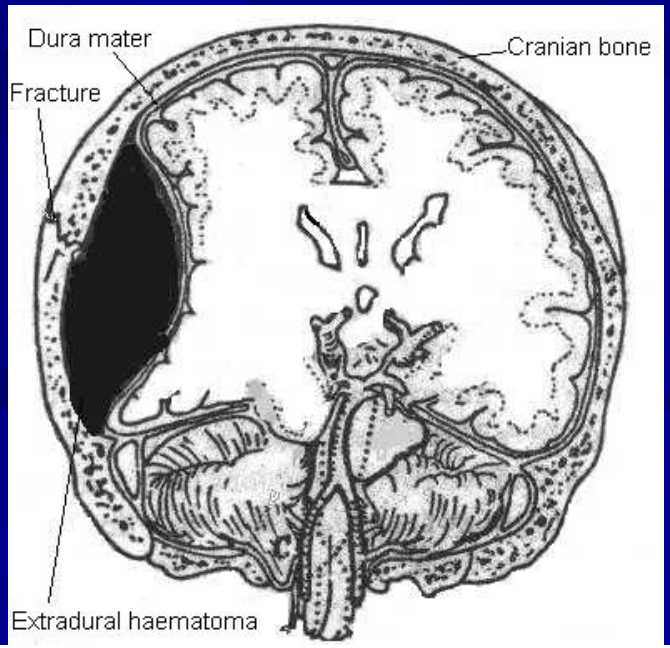
The collection can range from 60-150 ml but in some cases can even reach 300-400 ml.

The free interval can appear in any clinical form; it can last minutes (in supra-acute haematomas), hours (acute haematomas), days (sub-acute haematomas) or weeks (chronic haematoma). The presence of an extradural haematoma usually involves the presence of a skull fracture.

The direct dilaceration or the distortion of the middle meningeal artery can create a strong blood flow that defeats the adhesiveness of the dura mater to the base. The result is the detachment of the dura; also, the usually virtual extradural space becomes real and it is filled with blood; if it reaches a certain volume this blood collection becomes an extra-dural haematoma, with compressive effects on the cerebral tissue.

The main extradural collection area is the temporal fossa because here the adhesiveness of the dura is lower (the Gerard-Marchand detachable area).

The differential diagnosis of the extradural haematoma is made with various extradural pathological haemorrhages that are extremely rare, the pathological intra-dural haematoma and the chronic extra-dural haematoma.



2. The subdural haematoma

The subdural haematoma is a perfectly delimited blood collection developed between the dura mater and the arachnoid mater.

It can be of almost exclusive traumatic nature and it has a compressive effect on the brain.

The source of bleeding is the ruptured median veins that go from the cerebral cortex to the superior longitudinal sinus, crossing the subarachnoidian and subdural spaces.

The blood collection consists of brown-blackish, gelatine-like, friable clots in a mass of liquid blood.

The preferential localisation is parieto-cortico-dural, given the presence of the cortico-dural veins in the area. Delayed subdural haematomas are characterised by encapsulation.

The morphological aspect of the subdural haematoma is of a collection of lacquer blood, perfectly delimited by a fibro-conjunctive capsule (chronic, delayed, encapsulated subdural haematoma). The degree of conjunctive structuring of the capsule can be an indication for the age of the collection.

In penetrating injuries, the most important sources of blood are the vessels in the immediate area, closed traumatisms with extensive contusions (most frequently in the frontal and parietal lobes) and tearing of the veins that cross the subdural space.

In injuries, the most frequent blood source is an injured (crushed or distorted) vein that drains a dural venous sinus (saggital, lateral, spheno-parietal). Two main mechanisms are involved in the genesis of subdural haematomas:

The first mechanism is represented by the direct rupture of injured cortical blood vessels, or of vessels from the dilacerated area (arteries or veins).

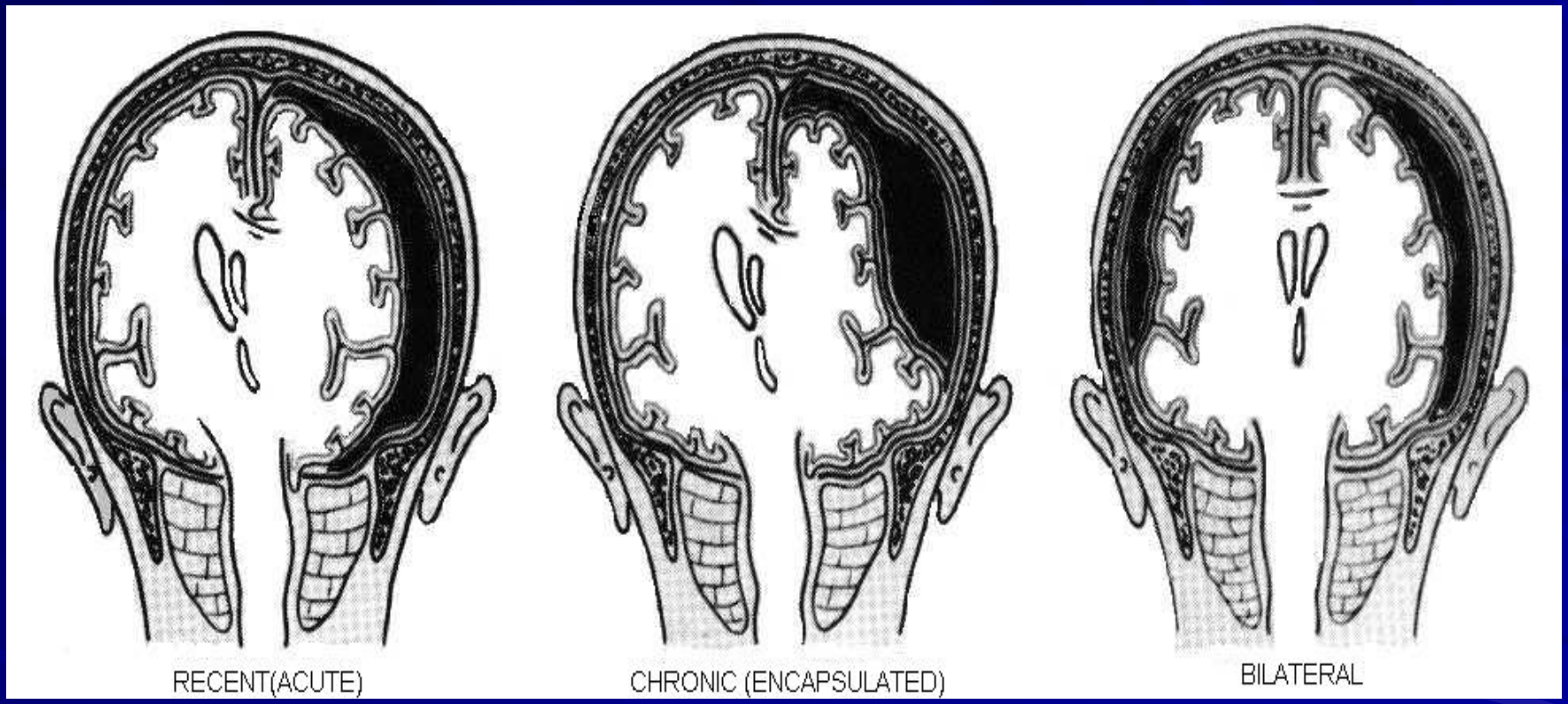
The second mechanism is the secondary development of an acute haematoma after the rupture of an intra-parenchymal post-traumatic haematoma. In this second situation the large collection dilacerates and dissects the cerebral substance and then overflows in the subdural space.

Usually the subdural haematoma appears in the impact area or in the surrounding areas. There are situations in which it appears in the contra-lateral area and it is the result of a countercoup mechanism.

It seems that the head acceleration induced by the impact is more likely the cause of subdural haematomas, rather then the impact in itself.

Most subdural haematomas are of traumatic origin. In rare cases, they can develop due to pathological causes.

The differential diagnosis is made with all the slowly progressive intra-cranial expansive processes, with the ischemic strokes or with non-traumatic subdural haematomas.


RECENT(ACUTE)
CHRONIC (ENCAPSULATED)
BILATERAL

3. Arachnoidal (leptomeningeal) haemorrhage

The subarachnoid haemorrhage is a blood escape constituted between the arachnoid and the pia mater; is can be of traumatic or pathological nature.

Usually the subarachnoidal haemorrhage is diffuse. The delimited forms are mostly situated on the convexities – P, rarely T or F.

The subarachnoidal haemorrhage is often caused by a cranio-cerebral trauma, especially in impacts with large objects. The cranial fracture is not obligatory, but when it is present it is usually linear.

The subarachnoidal haemorrhage can either develop as a direct result of on the trauma, at the impact pole, or at the opposite pole, as an indirect, countercoup injury.

The pathological aetiologies are represented by aneurisms and haemangiomas.

If the blood collection is large, it occupies the space between the arachnoid and the pia mater, it compresses the brain and it is called *subarachnoidal haematoma*.

The subarachnoidal haematoma is always traumatic and it can sometimes be encapsulated. It occurs in severe CCT with primary contusions and dilacerations.

The election area is F-T (the typical area for indirect cerebral contusions and cerebral dilacerations).

The morpho-pathological aspect of the subarachnoidal haematoma is of laquer blood mixed with blood clots.

In recent forms, the collection is not well delimited while in the older ones there is a thin, delimitating membrane.

There are two distinctive categories of mechanisms involved in the constitution of a subarachnoidal haematoma:

- a direct rupture of contusioned cortical vessels or vessels from the dilacerated area;

- secondary to the rupture of a post-traumatic intracerebral haematoma.

CEREBRAL LESIONS

1. The cerebral commotion

The clinical expression of the cerebral commotion is the short-term unconsciousness with no cerebral lesion. The phenomenon is due to functional disorders represented by a sudden depolarisation of the neuronal membranes from the cerebral trunk reticulate area. The effect is temporary, completely reversible, thus with no immediate or belated repercussions for the victim.

2. The cerebral contusion

The cerebral contusion is a primary traumatic lesion, with no cerebral lack of substance or discontinuities. It can appear in any traumatic mechanism. This lesion can be:

- either diffuse - with various intensity contusions in all cerebral layers, limited on larger or smaller areas;
- focal – when the affected area is of only few cm^2, situated beneath a depressed, focal skull fracture.

The cerebral contusion has preferential areas, such as orbito-temporal (because of the specific profiles of the endobase), the hypocampic area and especially the axial and periaxial regions of the cerebral trunk, towards which all traumatic energy lines converge.

Three severity degrees combine the clinical and morphological changes:

- minor cerebral contusion with bloody CSF (cerebrospinal fluid)
- medium cerebral contusion, with short subcomatous or comatous state and objective neurological findings;
- severe cerebral contusion, associated with profound coma and neurological repercussions in survivors, but often with lethal evolution.

The cortico-subcortical contusion appears on well-delimited areas that also include profound cortical layers or even the whole cortex.

The brain examination shows small, well-delimited haemorrhagic areas, situated mostly in the cortical and subcortical areas. In time, these lesions become brown-yellowish, with necrosis that gives the contusive area a homogenous aspect.

Cerebral contusions are specific for some lesional mechanisms, with strong impacts, with acceleration-deceleration mechanisms. It is usually accompanied by cortico-subcortical contusions in the impact or/and the countercoup areas.

Macroscopically, the diffuse contusion appears as small haemorrhagic spots in the cerebral white matter, with emphasis on the basal structures. In some cases, the diffuse contusion areas can coalesce, forming an intra-parenchymal haematoma.

In severe, diffuse cerebral contusions, the entire brain can be congested, with the entire superficial vascular system dilated and wide subpial suffusions, with characteristic violet-bluish aspect.

In severe cerebral contusions, the predominant lesions are either haemorrhagic or necrotic, or both.

The mechanism of cerebral contusion

The presence, extension, severity and topography of the contusions are in direct relation with the intensity, topography and direction of the impact; the morphological particularities of the interested cerebral area also play a role.

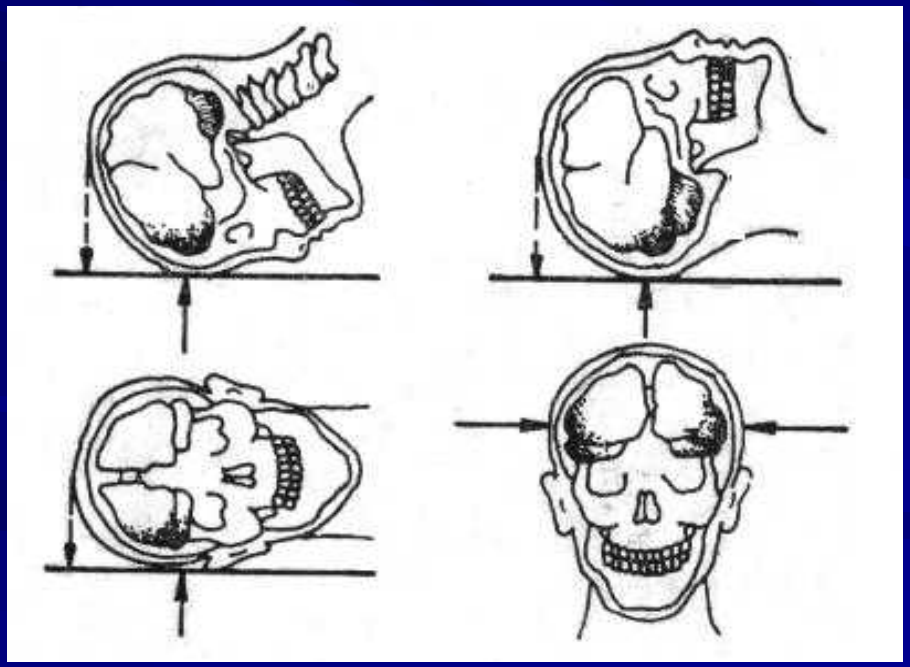
Regardless of the type of impact, the presence of cerebral contusions proves the existence of an external aggression, because the aetiology of the cerebral contusion is exclusively traumatic.

The first phase of the patho-physiological mechanism of cerebral contusions is a paralytic capillary dilation with consequent small, petechial haemorrhages. The cerebral contusion can either be the result of a skull depression in the impact area, or of a countercoup movement of the brain.

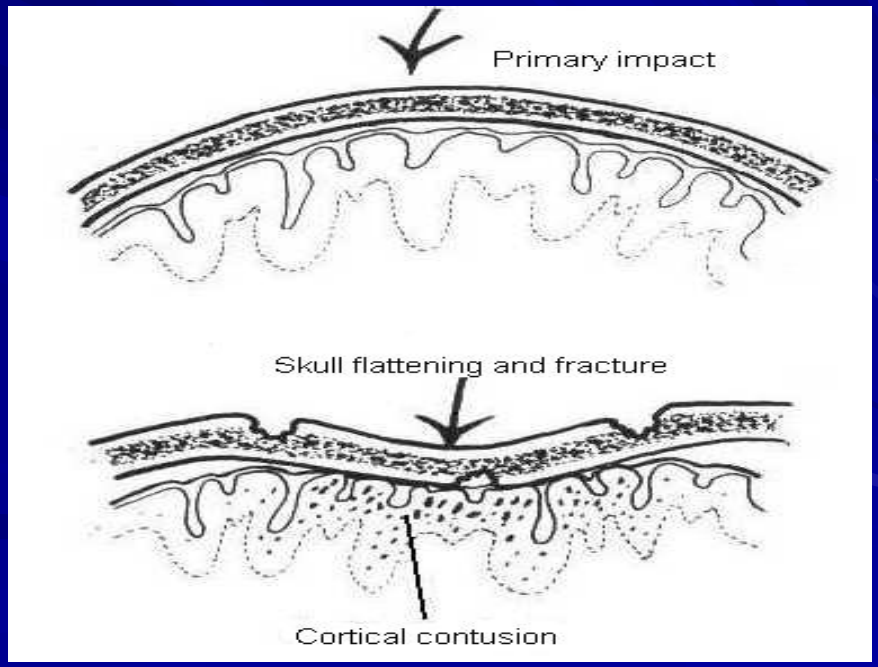
The various and complex physical mechanisms are numerous but can be grouped as follows:

- Cranial inflexion – an intrusion area forms in the impact point;
- The speed gradient between the brain and the skull in acceleration or deceleration is determined by the difference between their specific weights.

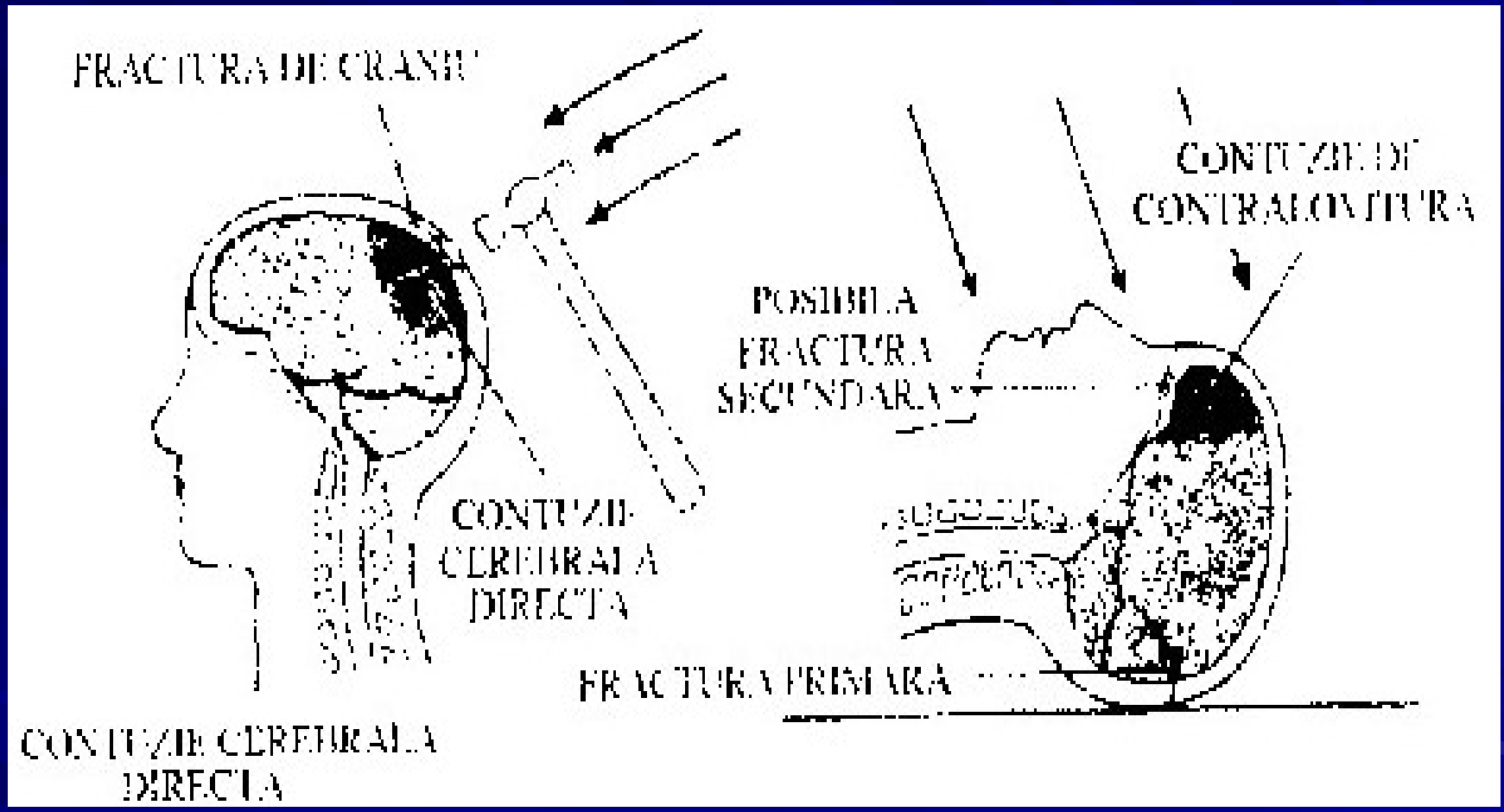
The topography of cerebral contusions is variable, depending on the impact area. Countercoup contusions often appear in the frontal and temporal poles, in the temporo-parietal areas and rarely in the occipital lobes.



The mechanisms of cerebral contusion



The mechanism of cortical contusion


FRACTURA DE CRANIU
CONTUZIE CEREBRALA DIRECTA
CONTUZIE DE CONTRALOVITURA
POSIBILA FRACTURA SECUNDARA
FRACTURA PRIMARA
CONTUZIE CEREBRALA DIRECTA

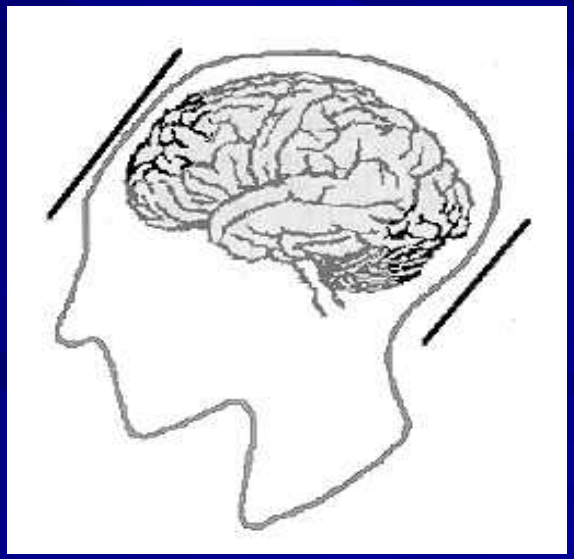
The countercoup lesions

The countercoup lesion is frequent in cranio-cerebral traumatisms.

It represents all meningo-cerebral lesions that appear opposite to the impact area.

It is most common in medium and strong traumatisms and rare in mild and very strong impacts; it is absent in head crushes.

The countercoup lesion can develop during cranial accelerations followed by sudden deceleration; its location varies with the primary impact area, but it is more frequent in the frontal and temporal regions, rare in the occipital area, vertex and the base, and extremely rare along the oblique cranial diameters (temporo-frontal, temporo-occipital).



The countre-coup mechanism

3. Diffuse axonal injuries (DAI)

Head injury can be grouped in 5 categories:

- ***extra-cranial lesions,***
- ***fractures of the skull,***
- ***penetrating injuries,***
- ***focal cerebral lesions and***
- ***diffuse cerebral lesion.***

Many CCT imply generalized cerebral lesions, without the presence of any focal lesion. The name of the DAI refers to their morphological nature; they are considered the most common cause of persistent vegetative state or of severe invalidities after a closed CCT.

The reported incidence of DAI ranges between 13 and 18% of all lethal head traumas. Approximately 12% of the patients with DAI present normal or quasi-normal CT images.

DAI can be considered one of the most severe parenchymal lesions, because its main effect is the prolonged traumatic coma, that raises difficulties of interpretation from the forensic points of view.

DAI can be the result of head acceleration and/or deceleration mechanisms, or of whiplash.

Their effect is the coronal or rotational movements and vibrations of the cerebral mass, with energy dissipation, localised scissoring and often secondary axonotomy.

Microscopy shows the increase of axonal volume

- ***massive, diffuse, even balloon-shaped axonal swelling***
- ***tuberous dilations***
- ***axonal bulbs***
- ***irregularities in the histological aspect of the axons***
- ***retractions or even***
- ***ruptures***

These changes are frequently associated with vacuolisations and glial reaction, with astrocytes and microcytes multiplication.

Based on the microscopical aspects, DAI can be defined as a diffuse axonal lesioning in various cerebral areas (hemispheres, corpus calossum, cerebral trunk) consecutive to a closed CCT, often in the absence of a skull fracture.

The axonal bulbs are the primary post-traumatic element. They can be identified even if the survival time is of only 12-18 hours; they represent the main marker for the positive diagnosis of DAI.

Microscopy can also reveal diffuse astrogliosis or/and glial, peri-neuronal and perivascular, micro-focal crowding.

The reactive astrogliosis syndrome can be considered an indirect marker of DAI but also of an unfavourable, lethal evolution of the case.

4. The cerebral dilaceration

The cerebral dilaceration is a more or less extensive destruction or discontinuity in the cerebral substance, sometimes with lack of cerebral substance, with irregular margins and necrosis.

The cerebral substance appears disintegrated, with unrecognisable components, in fact a blend of devitalised brain and liquid or clotted blood.

In localized dilacerations, contusion areas can surround the destruction mass. In time, they coalesce, undergo changes of colour and add to the dilaceration area.

Cerebral dilaceration is either superficial, after sudden contacts with rough surfaces, or profound, in penetrating mechanisms, when it is channel or cavity-shaped. Vascular necrosis is not constantly associated.

In cerebral dilacerations, the microscopic examination is unable to identify various morphologic elements.

In peripheral areas, some pale and vaguely defined neuronal and glial cells, on a background of necrosis and blood escape can be identified. The necrosis areas include granullar bodies resulted from the microglial phagocyte activity.

Cerebral dilaceration is present both in the impact area, with skull fractures and, rarely, in the countercoup region.

5. The intra-cerebral traumatic (parenchymal) haematoma

Traumatic parenchymal haematomas are well delimited, developed in the cerebral substance, most frequently in contusion centres. They have an expansive behaviour, with progressive, compressive evolution.

The haematoma normally consists of blood clots and laquer blood; their quantity and prevalence vary with the evolutional stage.

The volume of the intra-cerebral haematoma can range between 20 and 100 ml. There is no parallelism between the volume and the clinical effects. Small haematomas can determine a focal signs or intracranial hypertension, while other large haematomas can be oligo-simptomatic.

The preferential topography of the intracerebral haematomas is the F-T region, and rarely, in the posterior areas.

The intracerebral haematoma can also be the result of coalescence between multiple diffuse cerebral contusions.

The mechanism of intracerebral haematoma can be:

- ***a direct, vascular (arterial or venous) lesion (rare, penetrating injuries);***
- ***an indirect vascular changes (most frequently);***
- ***a post-traumatic thrombosis with secondary haemorrhagic necrosis;***

Direct traumatic haematomas are dominantly compressive, both because of their expansive nature and of the accompanying peri-lesional oedema.

THE COMPLICATIONS OF CRANIO-CEREBRAL TRAUMATISMS

CCT, especially the open ones, can develop complications, either immediately after the trauma, or after a variable period.

Complications can be local, distant or general, immediate, semi-belated and belated (sequelae). Belated complications and their effects have a negative influence on the long-term neurological state. The CCT sequelae are permanent lesions.

From the medico-legal stand point, they imply the notion of permanent invalidity or impairment.

The severity of the sequelae is in direct relation with the extent and the topography of the initial lesion, but also with some persistent changes, with the state of neuronal anoxia (prolonged coma, diffuse contusions), or with other complications (meningitis, abscesses).

CCT sequelae include cerebral parenchymal changes consequent to primary, acute or subacute, traumatisms.

The main categories of sequelae are:

- the traumatic atrophy of the white matter
- the meningo-cerebral scarring.

The traumatic atrophy of the white matter, **also known as posttraumatic atrophic sclerosis can be the result of almost all acute and subacute traumas, especially of vascular paralysis, compression and cerebral oedema. It can also appear after delimited lesions, around an area of cerebral infarct, around parenchymal haematomas, or secondary to cerebral contusions.**

In many cases, there is the possibility that a primary traumatic lesion, limited by a cortical area, generates an extensive atrophy of the white matter. Traumatic atrophy can be diffuse or limited to a hemisphere or cerebral lobe.

The leptomeninges appear thickened and the corresponding cerebral substance is discoloured, with higher consistency. Sometimes sectioning reveals higher density areas.

The meningo-cerebral scar**: if it is predominantly meningeal, it mainly involves the dura mater that displays a limited, fibrous-elastic, grey-brownish, area, prominent on the internal side. In most cases, there are fine or dense adherences to the leptomeninges and the cerebral cortex. In the scared area, the leptomeninges are whitish, opalescent, with strong cortical adherences. In recent scaring, the cortex is yellow-greenish or yellow-brownish, friable and porous; in some cases, cyst-like formations also appear.**