CNS II

Traumatic vascular disease of the nervous system

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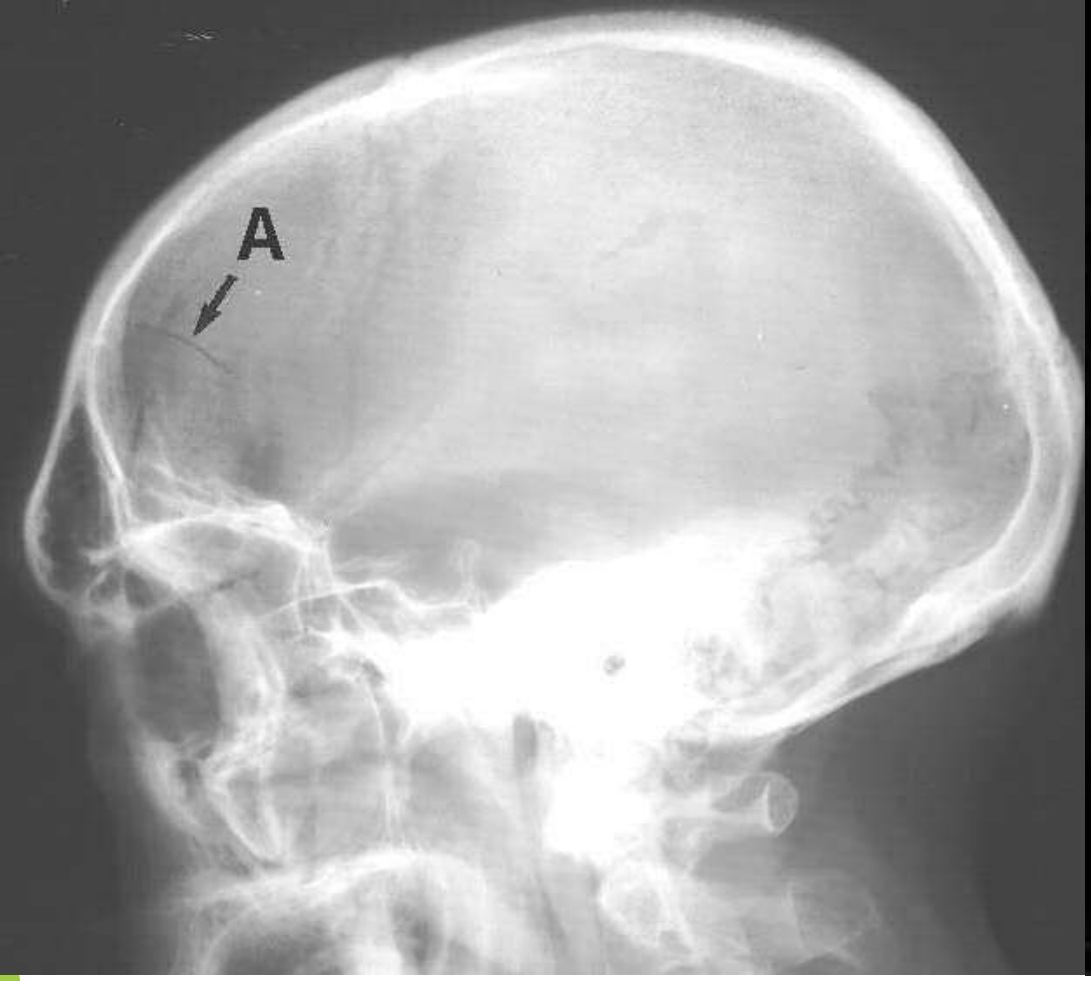
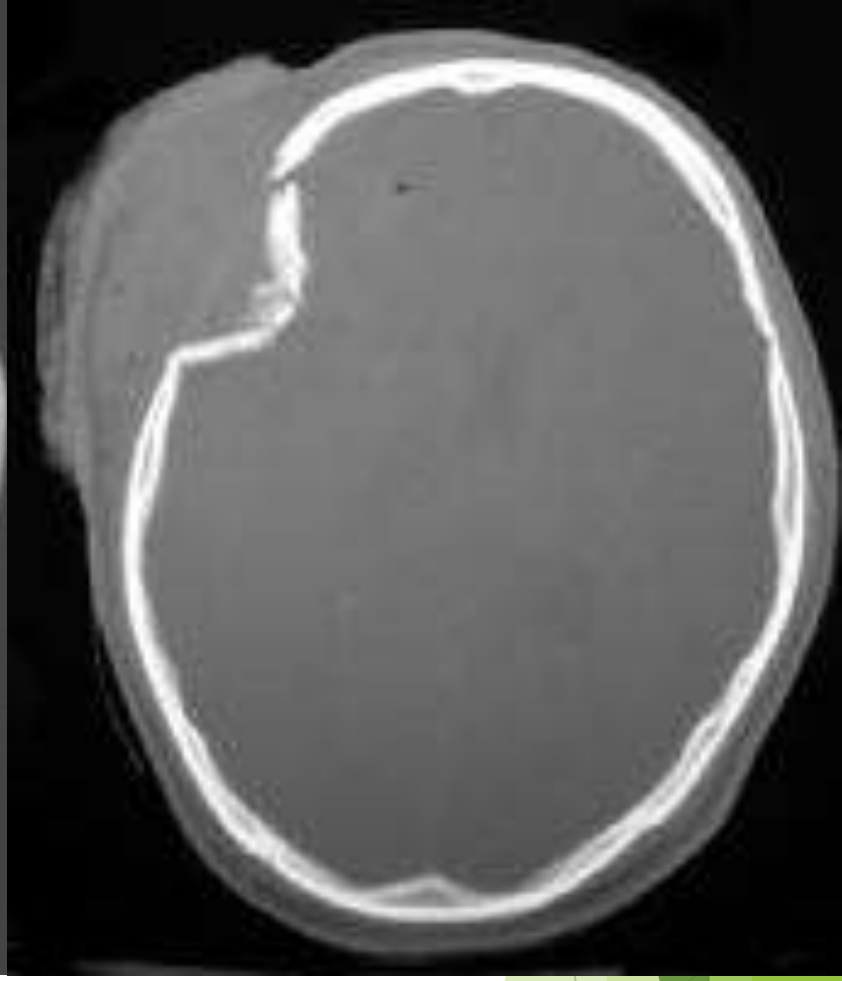
CENTRAL NERVOUS SYSTEM TRAUMA:

Result & morphology depend on:

- Penetrating or Blunt trauma→ Open or Closed
- Mobile or immobile head at time of trauma
- Lesions at bony prominences e.g
  - Frontal, orbital, temporal & occipital poles
  - Spinal cord
- Edema may occur & worsen the condition
- ? skull fracture





“HAIRLINE”

“DEPRESSED” or “DISPLACED”

A- Parenchymal injury:

1- Concussion:

- Immediate and temporary disturbance of brain function with reversible altered consciousness.
- No demonstrable lesion
- Signs: confusion, headache, visual disturbances, nausea, vomiting, dizziness…. Then recovery with Amnesia for the event

I. Concussion

- Is a clinical syndrome of altered consiousness secondary to head injury.
- Brought by a change in the momentum of the head when a moving head suddenly arrested by impact on a rigid surface.

- The characteristic neurologic picture includes onset of transient neurologic dysfunction including:

1. Loss of consciousness,
2. Temporary respiratory arrest
3. Loss of reflexes.

- Although neurologic recovery is complete , amnesia for the event persists.
- Pathogenesis is unknown but may result from temporary deregulation of the reticular activating system in the brainstem.

Complications

1. Post concussive neuropsychiatric syndromes typically associate with repetitive trauma are well recognized
2. Significant cognitive impairment with distinct pathologic findings called chronic traumatic encephalopathy

Direct parenchymal injuries

1. Contusions

- Caused by blunt trauma to the brain

-The pia- arachnoid is not breached

-Mechanism

- A blow to the surface of the brain transmitted through the skull leads to rapid tissue displacement , disruption of vessels , hemorrhage and tissue injury.
- Tissue injury, more on crests of gyri ± The crest of gyri are most susceptible than the depth of sulci

- Intraparenchymal, subarachnoid hemorrhage
- Coup Contusion & Contrecoup contusion

2- Contusions & lacerations of cortex:

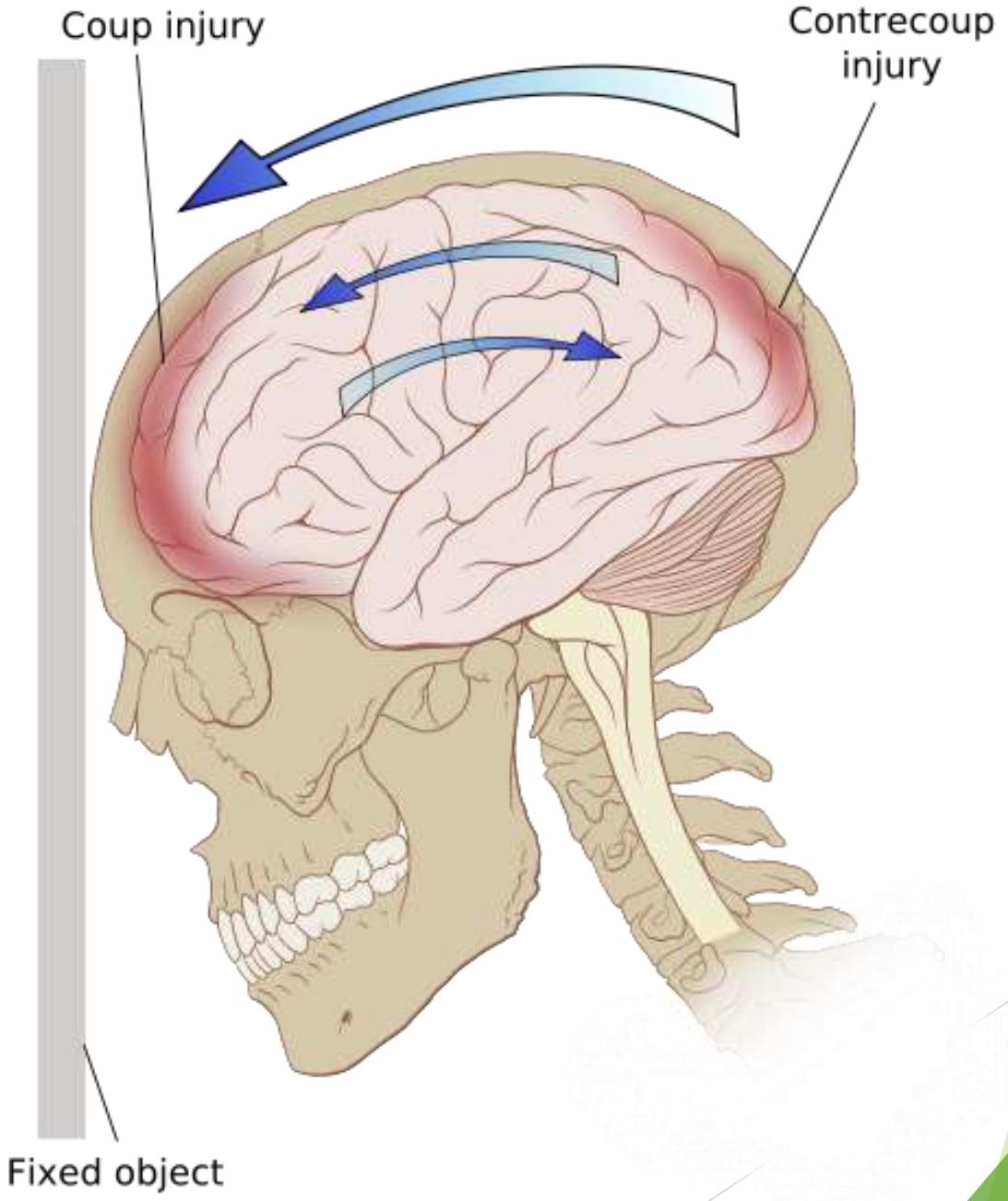
- Coup lesions:

*Contusions immediately beneath and associated with direct trauma
*Stationary head, no fracture, enough energy to damage brain

- Contrecoup lesions:

* Contusions at a distance from & frequently opposite to the point of trauma

- * Often represent rotational & deceleration injury, related to irregularities of the skullopposite point of impact


Coup injury
Contrecoup injury
Fixed object

Contusions are common in regions of the brain overlying rough and irregular inner skull surfaces, such as:

a. The orbital surfaces of the frontal lobes

b. And the temporal lobe tips

Note

- Contusions are less frequent over the occipital lobes, brainstem and cerebellum until these sites are adjacent to a skull fracture.

- Both types of contusions have similar gross and microscopic appearances.
- The distinction is made on identification of the point impact.

a. If the head is immobile at the time of trauma, only a coup injury is found.

- Is caused by contact between the surface of the brain and skull at the site of impact

b. If the head is mobile at the time of the trauma, both coup and contrecoup contusions may be found.

- Is thought to arise when the brain strikes the opposite inner surface of the skull after sudden deceleration

MORPHOLOGY :

- Contusions are wedge-shaped with the broad base lying along the surface at the point of the impact
- Microscopic examination

a. In the earliest stage:

Edema and hemorrhage.

b. During next few hours:

- Extravasation of blood extend throughout the cortex to white matter then to the subarachnoid space.

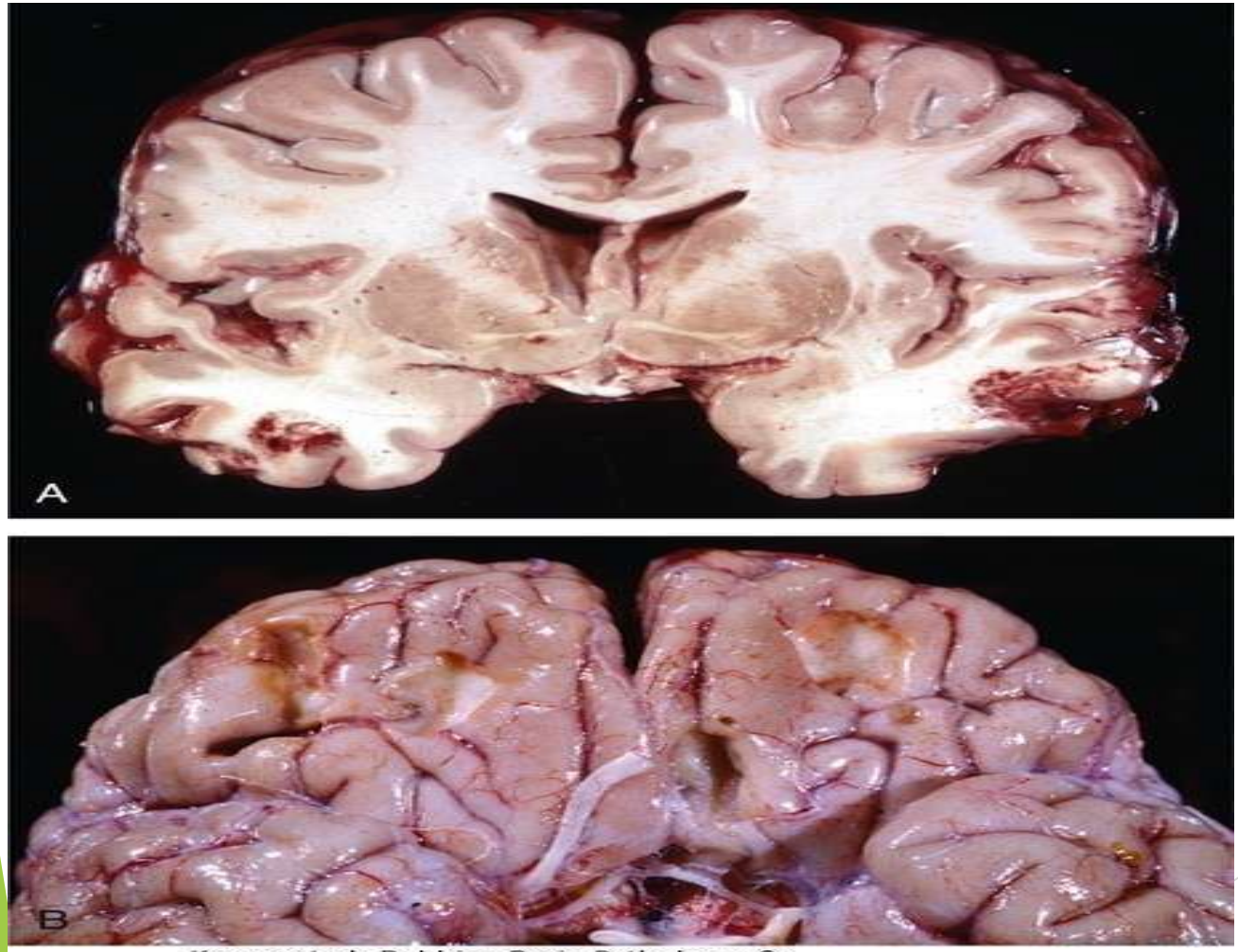
c. Old traumatic lesions:

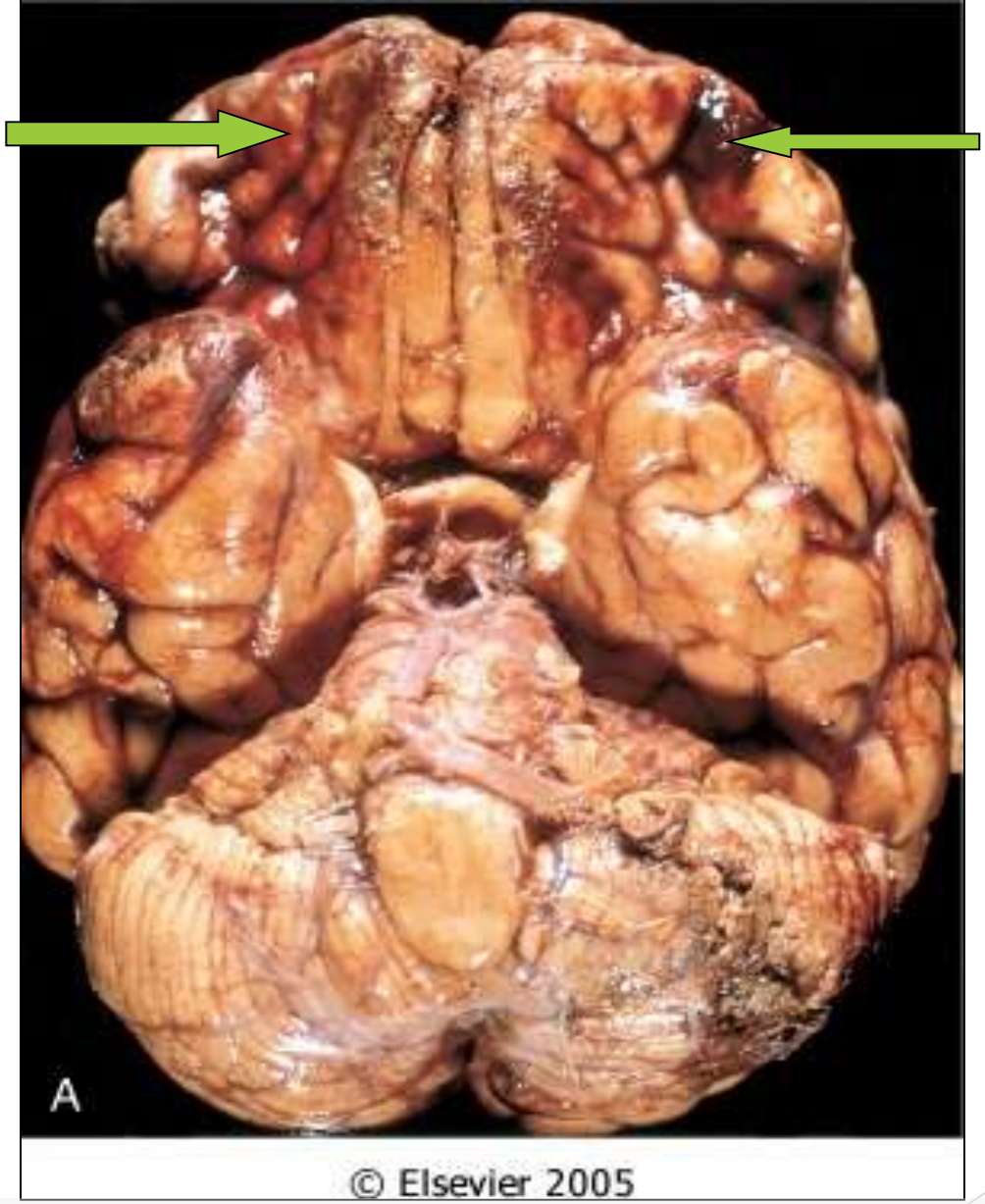
- Are depressed retracted yellow brown patches (called plaque jaune).

Early contusions at orbital gyri of frontal lobes

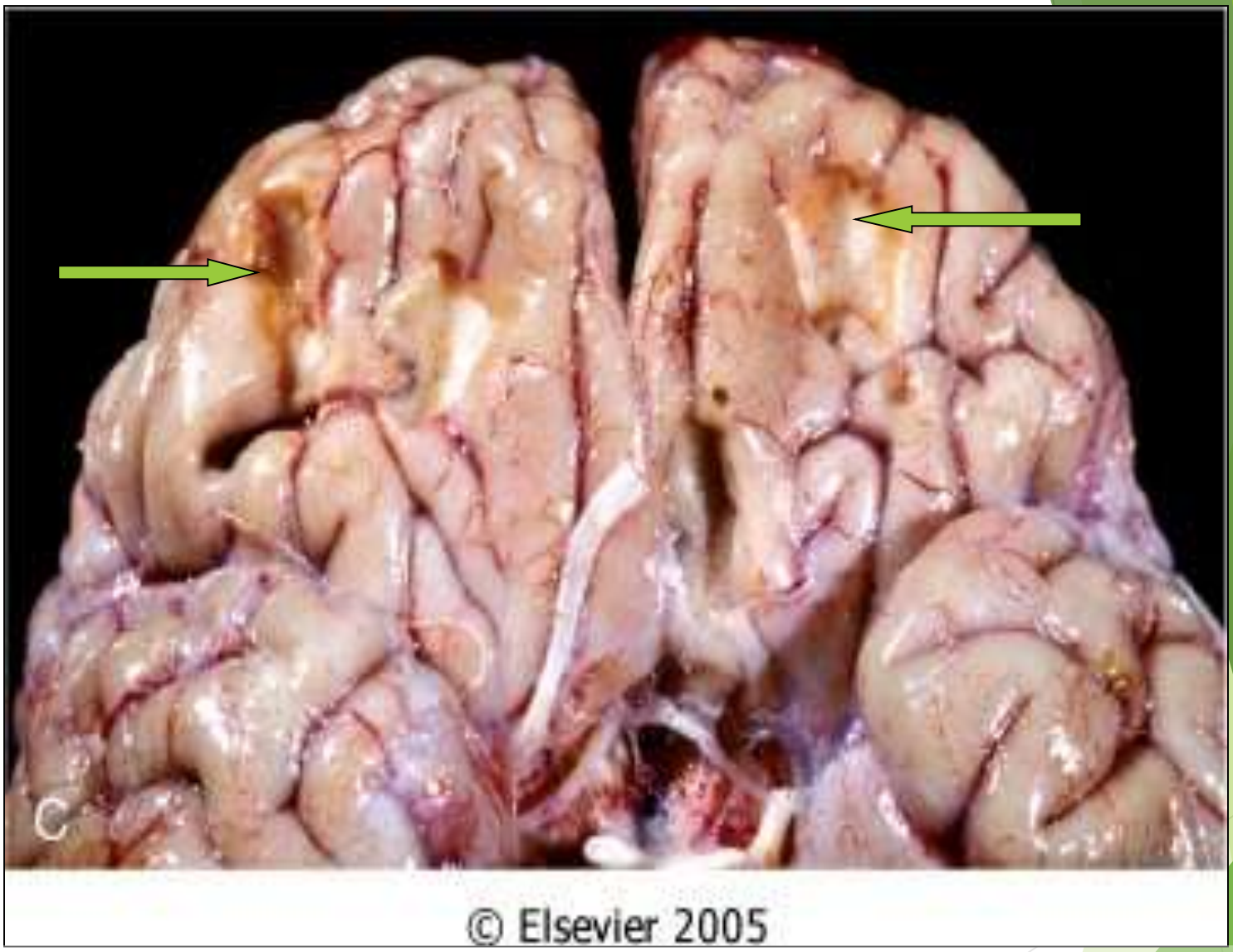
Old contusions

Cerebral trauma. (A) Acute contusions are present in both temporal lobes, with areas of hemorrhage and tissue disruption. (B) Remote contusions, seen as discolored yellow areas, are present on the inferior frontal surface of this brain





Multiple contusions involving the inferior surfaces of frontal lobes, anterior temporal lobes, and cerebellum.



Remote contusions are present on the inferior frontal surface of this brain, with a yellow color

3- Intracerebral hemorrhage:

Cutting of brain vessels, high impact

4- Cerebral Edema: Occurs with and without an obvious structural lesion

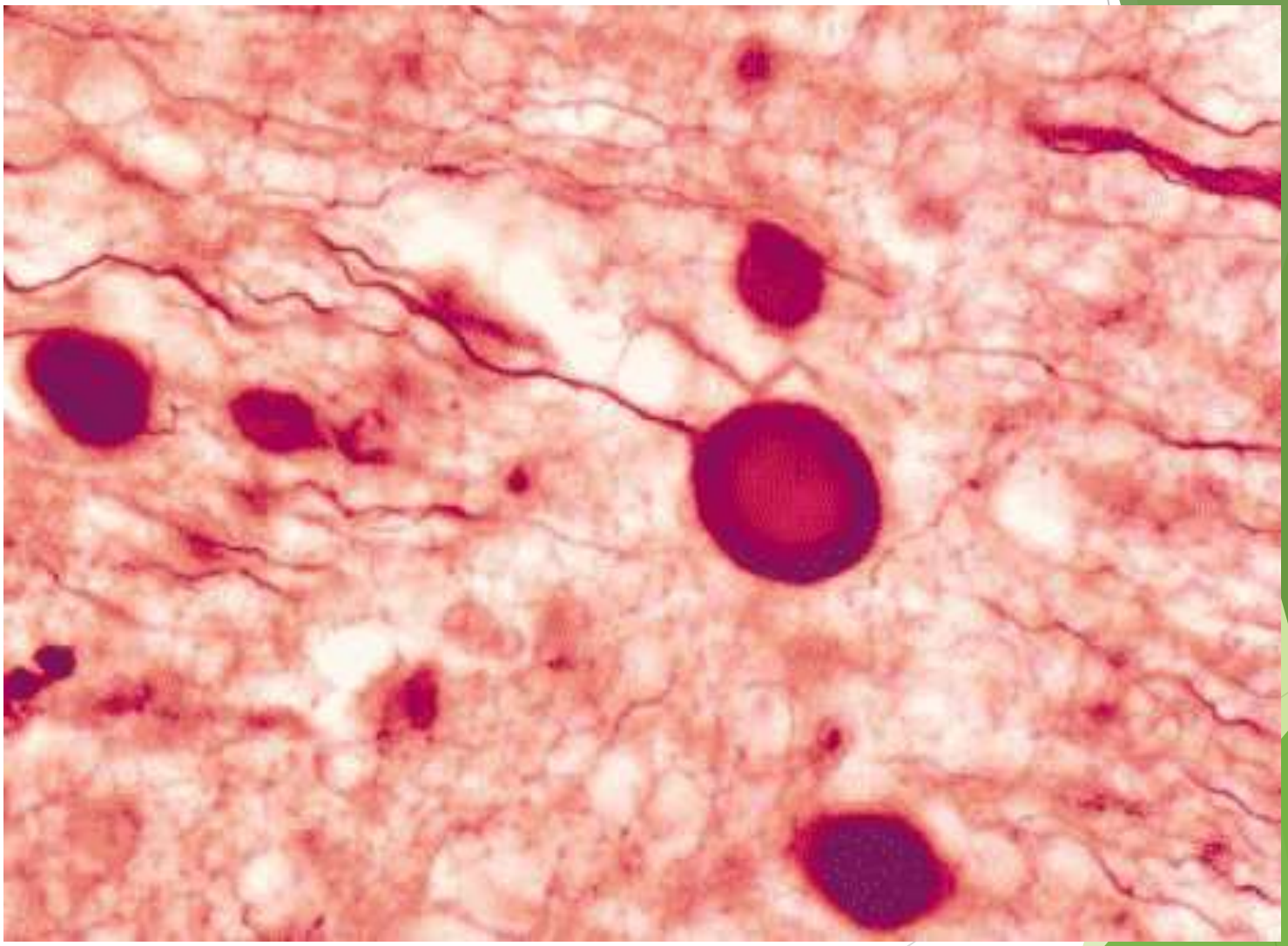
Note: Can occur without evidence of hemorrhage

5- Diffuse Axonal Injury:

Stretching force & cutting of axons

- **Diffuse Axonal Injury:**
  - Acceleration / Deceleration injury
  - 50% of patients with post-traumatic coma
  - Affect white matter (corpus callosum, paraventricles, hippocampus…etc) & at junction of grey & white matter
  - Characteristic asymmetric axonal swelling (Retraction Balls), micro – hemorrhages, ↑microglia, later gliosis
  - Post-traumatic dementia & vegetative state.

Axonal retraction balls



B-Traumatic vascular injuries

1- Epidural Hematoma:

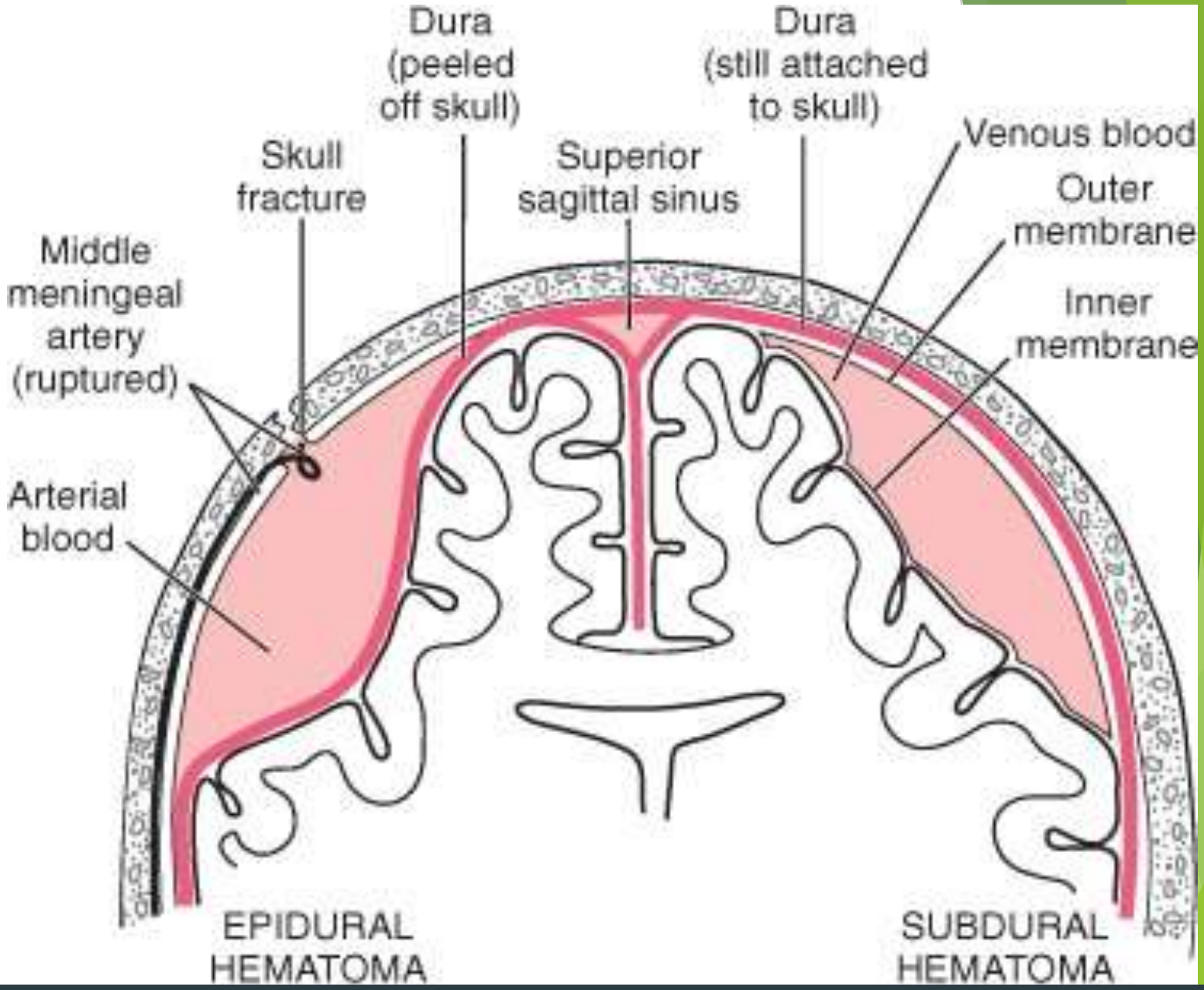
- Normally the dura is fused with the periosteum on the internal surface of the brain.
- - Dural arteries, most importantly, the middle meningeal artery are vulnerable to injury especially with skull fracture in which the fracture cross the course of the vessel.
- Usually acute & accompanied by skull fracture
- Seen in 3% of significant trauma
- Rupture of **middle meningeal artery**
- Rapid collection of blood (30 – 50ml → symptoms)

1- Epidural Hematoma:

- Mass effect ▫ Dura & Brain compression
- Rapid increase in ICP
- Clinically: Patient has LUCID interval for hours followed by rapid loss of consciousness
-
- Fatal within 24 – 48 hrs. if untreated

Note

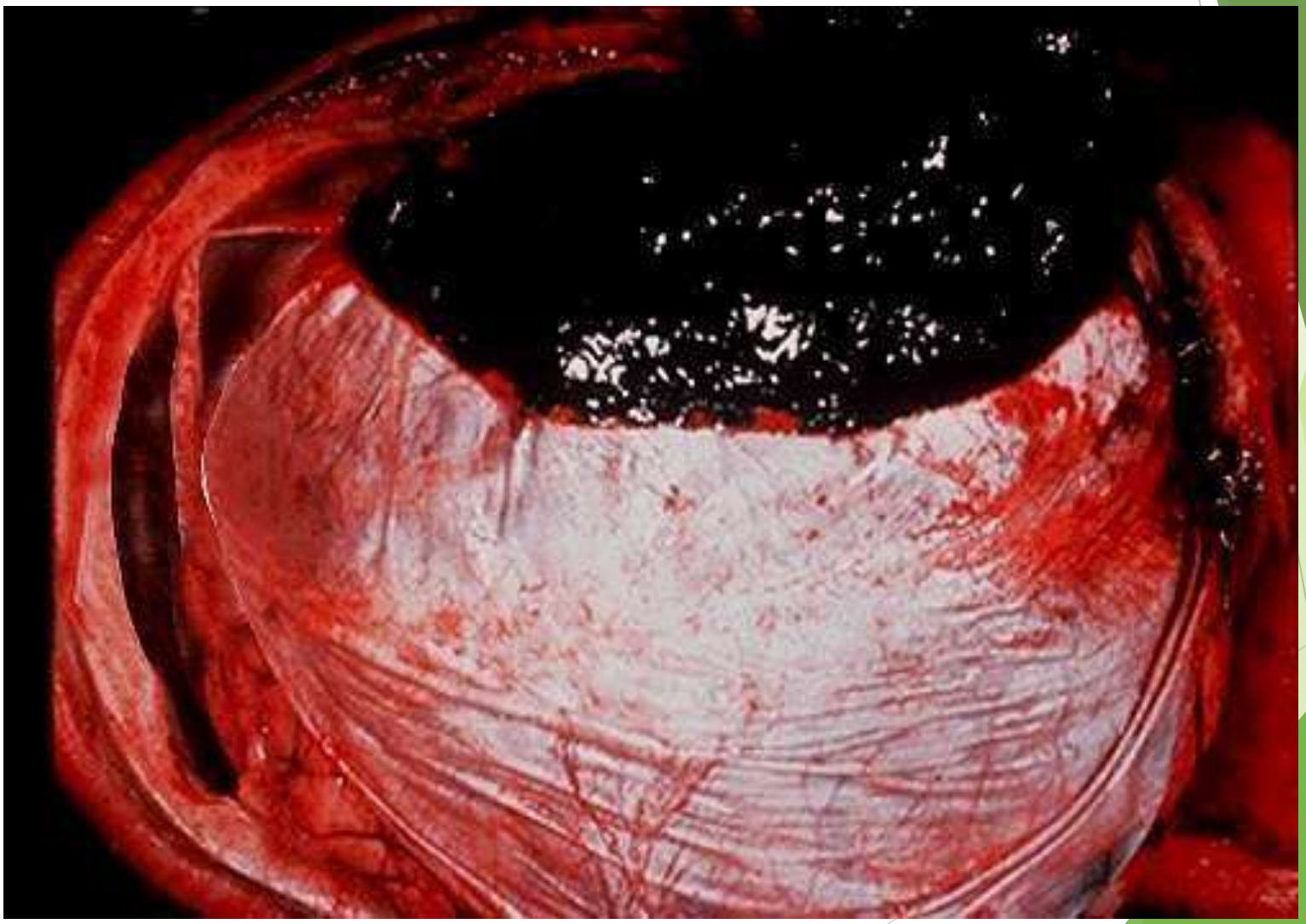
- In children in whom the skull is deformable, a temporary displacement of skull bones leading to lacerations of a vessel can occur in the absence of skull fracture



Comparison of epidural to subdural hematomas

- *When blood accumulates slowly, patients can be lucid for several hours between the moment of trauma and the development of neurologic signs..*
- An epidural hematoma may expand rapidly and constitutes a neurosurgical emergency necessitating prompt drainage and repair to prevent death

Epidural hematoma



Epidural hematoma covering a portion of the dura. Multiple small contusions are seen in the temporal lobe.

2- Subdural hematoma:

- The dura is composed of two layer, The external collagenous layer and inner border cell layer with scant fibroblasts and abundant extracellular space devoid of collagen
- When bleeding occurs, these two layers separate and create the subdural space in which the blood accumulates
- Bridging veins travel from convexities of the cerebral hemispheres through the subarachnoid space and the subdural space to empty into superior sagittal sinus

2- Subdural hematoma:

- These vessels are prone to tearing along their course through the dural layers

- The venous sinuses are fixed relative to the dura, so the displacement of the brain that occurs in trauma can tear the veins at the point where they penetrate the dura

2- Subdural hematoma:

- Most occur with changed head velocity e.g. boxers, battered baby & old age
- More common than epidural
- Disruption of Bridging Veins from brain to dural sinuses, more over convexities
- About 50% of acute are accompanied by fracture.

Susceptible people:

1. Old people with brain atrophy

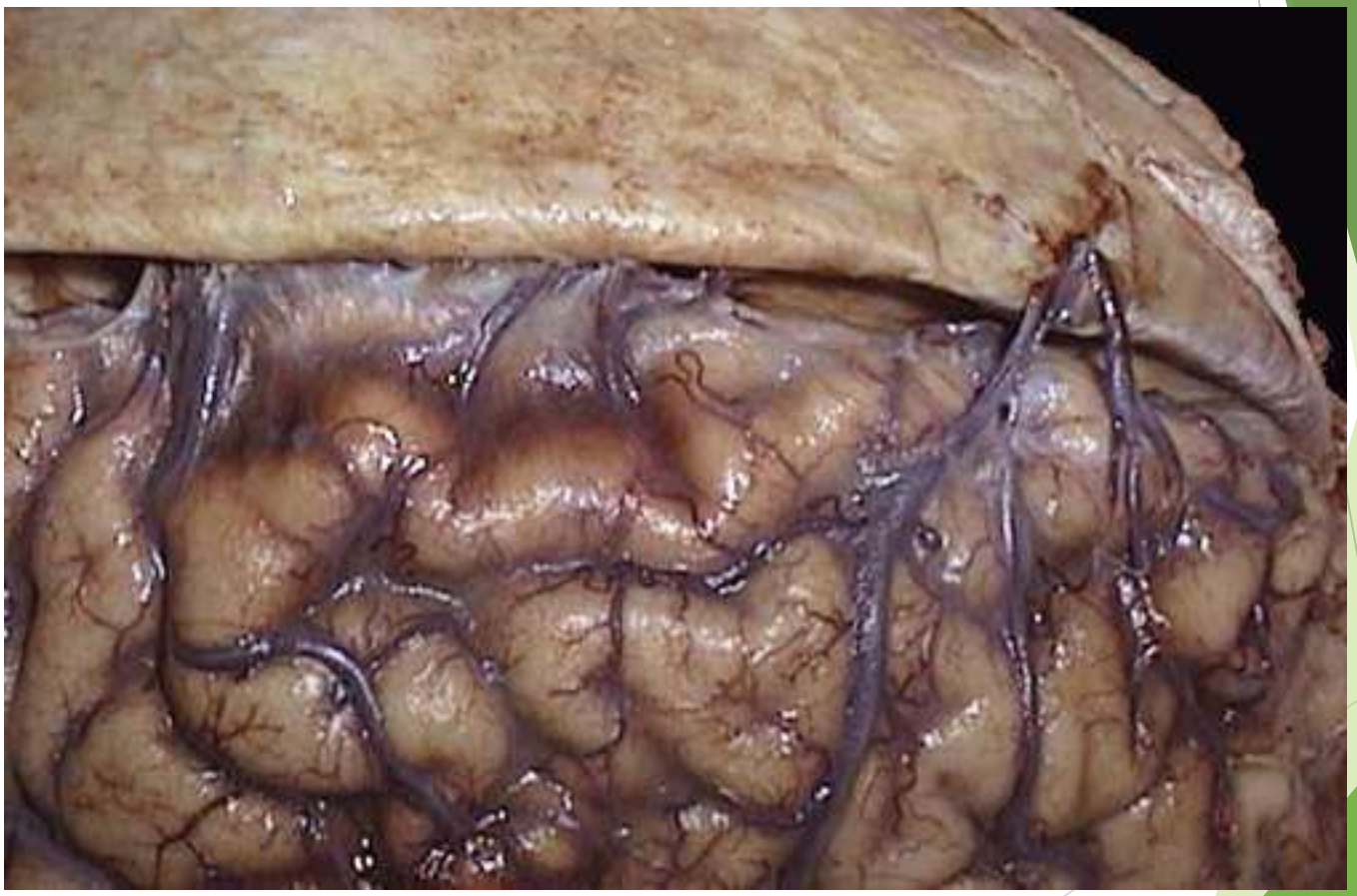
- Patients with brain atrophy, the bridging veins are stretched out, and the brain has additional space within which to move, accounting for the higher rate of subdural hematomas in elderly persons.

2. Infants also are susceptible to subdural hematomas because their bridging veins are thin-walled.

- May be categorized based on the interval between the hematoma and the traumatic event

  - Acute: within 3 days of trauma
  - Subacute: between 3 days and 3 weeks after trauma

  - Chronic: develops after 3 weeks



Morphology

- Grossly,

- Acute subdural hematomas appear as a collection of freshly clotted blood along the brain surface, without extension into the depths of sulci

- Flattened underlying brain and subarachnoid space is often clear.

- Typically, venous bleeding is self-limited; breakdown and organization of the hematoma take place over time

1. Lysis of the blood within one week
2. Growth of granulation tissue from the dural surface into the hematoma (2 weeks)

- Typically, the organized hematoma is firmly attached to the inner surface of the dura and is free of the underlying arachnoid, which does not contribute to healing.
- The lesion can eventually retract as the granulation tissue matures until only a thin layer of reactive connective tissue remains (“subdural membranes”).

- In other cases, however, multiple recurrent episodes of bleeding occur (chronic subdural hematomas), presumably from the thin-walled vessels of the granulation tissue.
- The risk of repeat bleeding is greatest in the first few months after the initial hemorrhage

Clinically

- Neurologic signs are attributable to the pressure exerted on the adjacent brain.

- Symptoms may be localizing but more often are nonlocalizing, taking the form of headache confusion, and slowly progressive neurologic deterioration.

- Subdural hematomas typically become manifest within the first 48 hours after injury.

- They are most common over the lateral aspects of the cerebral hemispheres and may be bilateral.

- Symptomatic subdural hematomas are treated by surgical removal of the blood and associated reactive tissue

- **Acute:**
  - Clear history of trauma
  - Frontoparietal is common
  - Slow collection of clotted blood with surrounding edema→↑ ICP
  - Nonfatal cases → Chronic

Chronic:

- Trauma often not recorded
- More in elderly with brain atrophy or

  ‘ Battered Baby Syndrome’
- Hematoma → Fluid filled cyst enclosed by membrane may be resorbed, or calcified
- Risk of rebleeding in first few months
- Clinically: confusion, dementia….etc

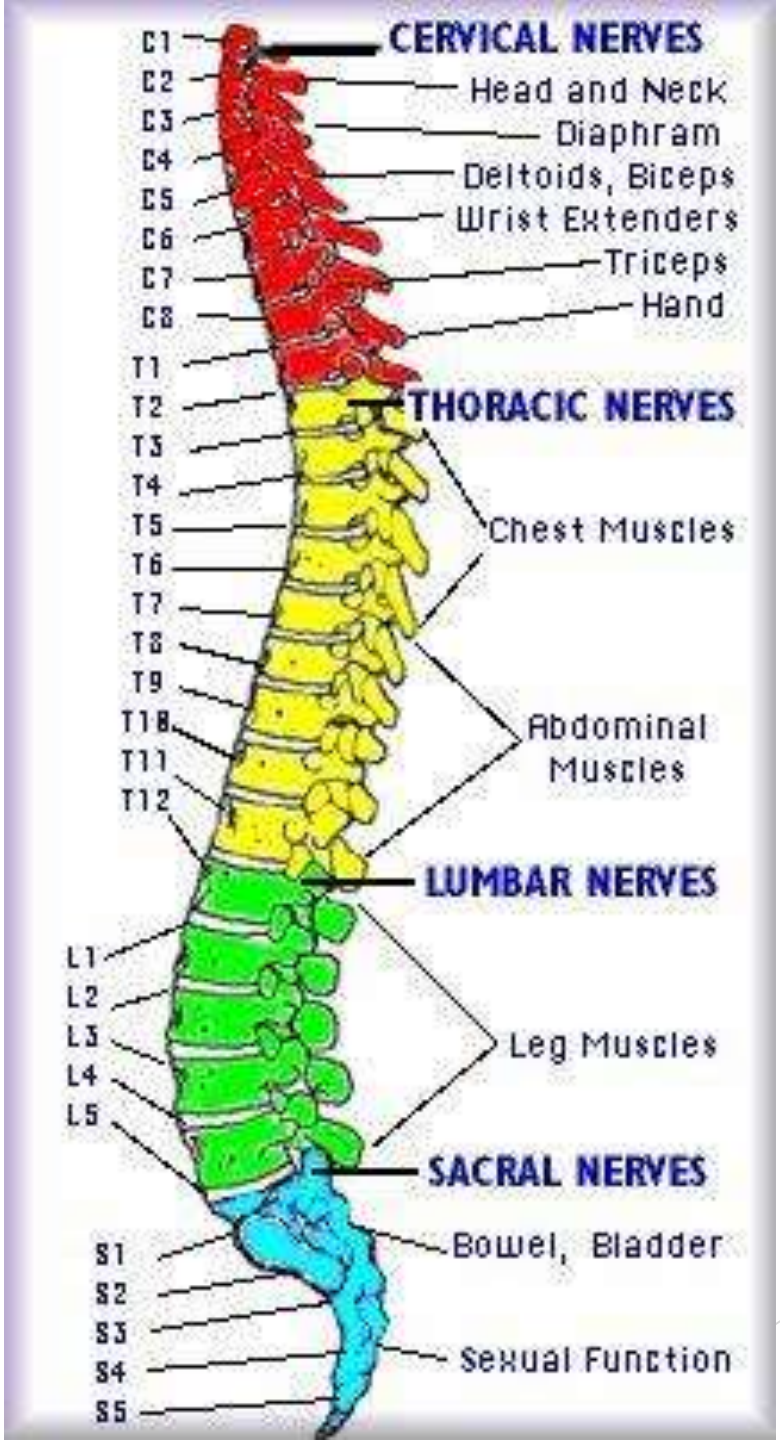
A. Large organizing subdural hematoma attached to the dura.
B. Coronal section of the brain showing compression of the hemisphere underlying the hematoma.

Table 23.1 Patterns of Vascular Injury in the Central Nervous System

Location	Etiology	Additional Features
Epidural space	Trauma	Usually associated with a skull fracture (in adults); rapidly evolving neurologic symptoms requiring intervention
Subdural space	Trauma	Level of trauma may be mild; slowly evolving neurologic symptoms, often with a delay from the time of injury
Subarachnoid space	Vascular abnormalities (arteriovenous malformation or aneurysm)	Sudden onset of severe headache, often with rapid neurologic deterioration; secondary injury may emerge due to vasospasm
	Trauma	Typically associated with underlying contusions
Intraparenchymal	Trauma (contusions)	Selective involvement of the crests of gyri where the brain contacts the skull (frontal and temporal tips, orbitofrontal surface)
	Hemorrhagic conversion of an ischemic infarction	Petechial hemorrhages in an area of previously ischemic brain, usually following the cortical ribbon
	Cerebral amyloid angiopathy	"Lobar" hemorrhage, involving cerebral cortex, often with extension into the subarachnoid space
	Hypertension	Centered in the deep white matter, thalamus, basal ganglia, or brain stem; may extend into the ventricular system
	Tumors (primary or metastatic)	Associated with high-grade gliomas or certain metastases (melanoma, choriocarcinoma, renal cell carcinoma)

Spinal cord trauma:

- Level of injury determines outcome
- Lesions include contusions, fiber transection, hemorrhagic necrosis $\rightarrow$ cystic foci in spinal cord
- Late effects: Ascending & descending degeneration in nerve tracts & systemic effects.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CERVICAL NERVES
Head and Neck
Diaphram
Deltoids, Biceps
Wrist Extenders
Triceps
Hand
THORACIC NERVES
Chest Muscles
Abdominal Muscles
LUMBAR NERVES
Leg Muscles
SACRAL NERVES
Bowel, Bladder
Sexual Function

Effects depend on level:

- Cauda equina ▫ pain, weakness of lower limbs...
- Thoracolumbar → Paraplegia
- Cervical → dysfunction loss of bladder & anal control, bed sores …. Quadriplegia
- Above C4 → Paralysis of diaphragm

- Result: Infections, genitourinary

Perinatal Brain Injury

- May be intrauterine or during birth
- Causes include:
- Hypoxic/Ischemic injury
- Germinal matrix hemorrhage
- Infections & Toxins
- Birth trauma
- May not be noticed at birth
- Important cause of Cerebral Palsy ▫ Neurological motor deficits, e.g. spasticity, dystonia, ataxia….

Secondary Complications of Trauma:

- 1- Diffuse brain edema, secondary hemorrhage & herniations
- 2- Hypoxic brain damage
- 3- Infection
- 4- Fat embolism
- 5- Post-traumatic dementia
- 6- Persistent vegetative state
- 7- Epilepsy.

The End

Good Luck