

# V. CENTRAL NERVOUS SYSTEM

## TRAUMA

# **I. Concussion**

- Is a clinical syndrome of altered consciousness 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  
Instantaneous 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

## *II. 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, tissue injury

- Blood can extend into the subarachnoid space
- The crest of gyri are most susceptible than the depth of sulci
- Are common in regions of the brain overlying rough and irregular inner skull surfaces, such as:

- a. The frontal poles
- b. The orbital surfaces of the frontal lobes
- c. 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



- A person who suffers a blow to the head may develop a contusion at the point of the contact called ***coup contusions***
- Or may suffer a contusion on the brain surface opposite to the site of the contact called ***contrecoup contusion***

- 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 :

- 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)

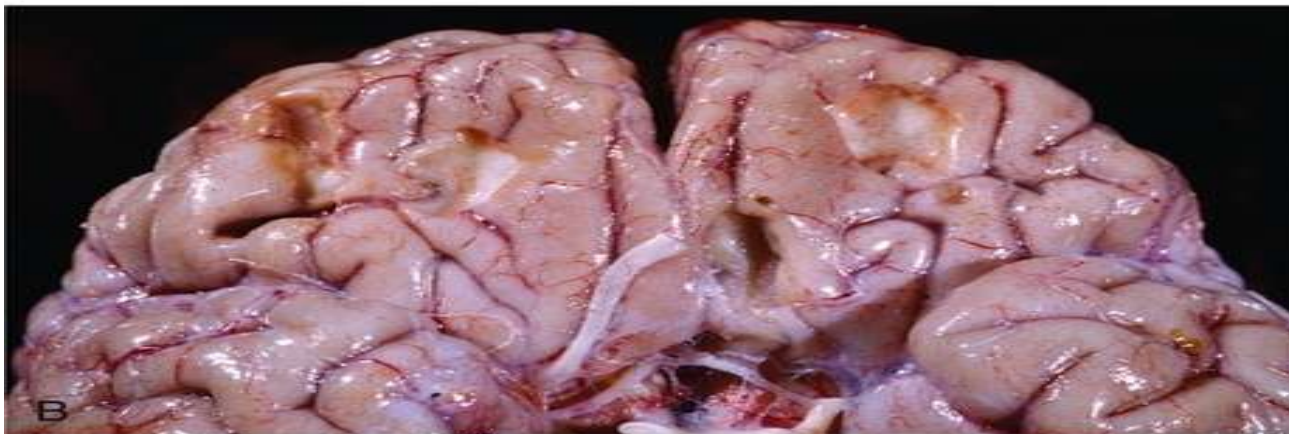
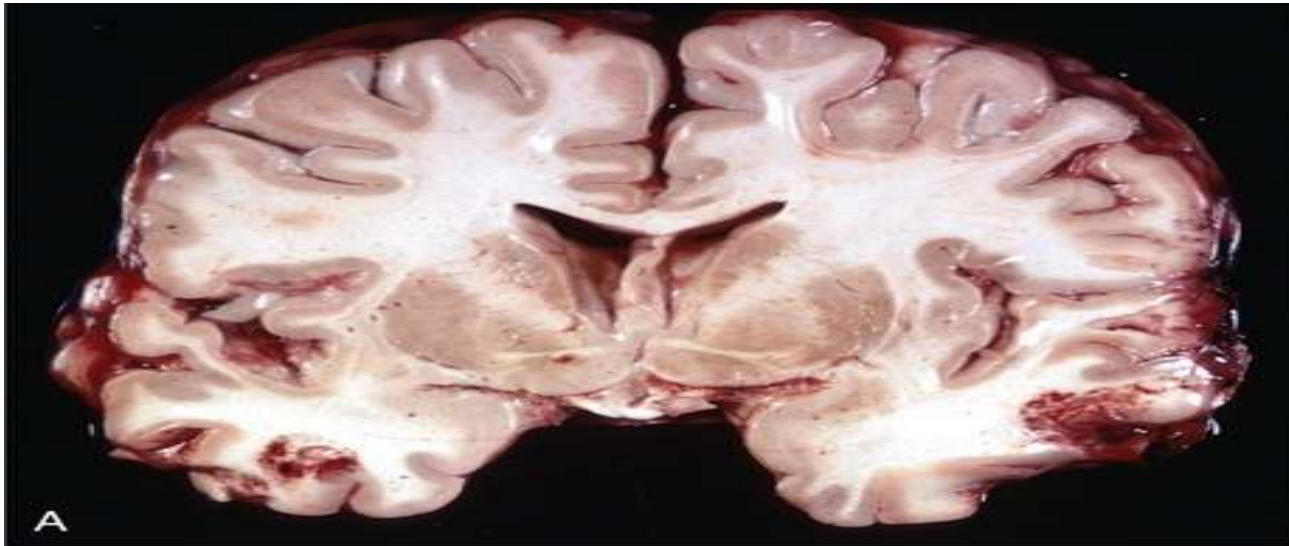
# Early contusions at orbital gyri of frontal lobes



# Old contusions



# Contusions: Recent and old





### III. Diffuse axonal injury

- Trauma can also cause more subtle but widespread injury to axons within the brain with devastating consequences
- Axons are injured by
  - a. The direct action of mechanical forces with subsequent alteration of axoplasmic flow

b. Or by angular acceleration alone, which can cause axonal injury even in the absence of impact

**Note:**

- As many as 50% of patients who develop coma shortly after trauma are believed to have white matter damage and diffuse axonal injury.

-These injuries are widespread, and asymmetric and are most commonly found in

- a. Corpus callosum
- b. Paraventricular area
- c. Cerebral peduncles
- d. Reticular activating formation

# Morphology

- They take the form of axonal swellings that appear within hours of the injury and may persist for much longer
- The swelling can be demonstrated immunostains for axonally transported proteins such as **amyloid precursor protein**

## IV. Traumatic vascular injuries

- It results from direct trauma and disruption of the vessel wall and leads to hemorrhage in different anatomic sites

# 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

## **Note**

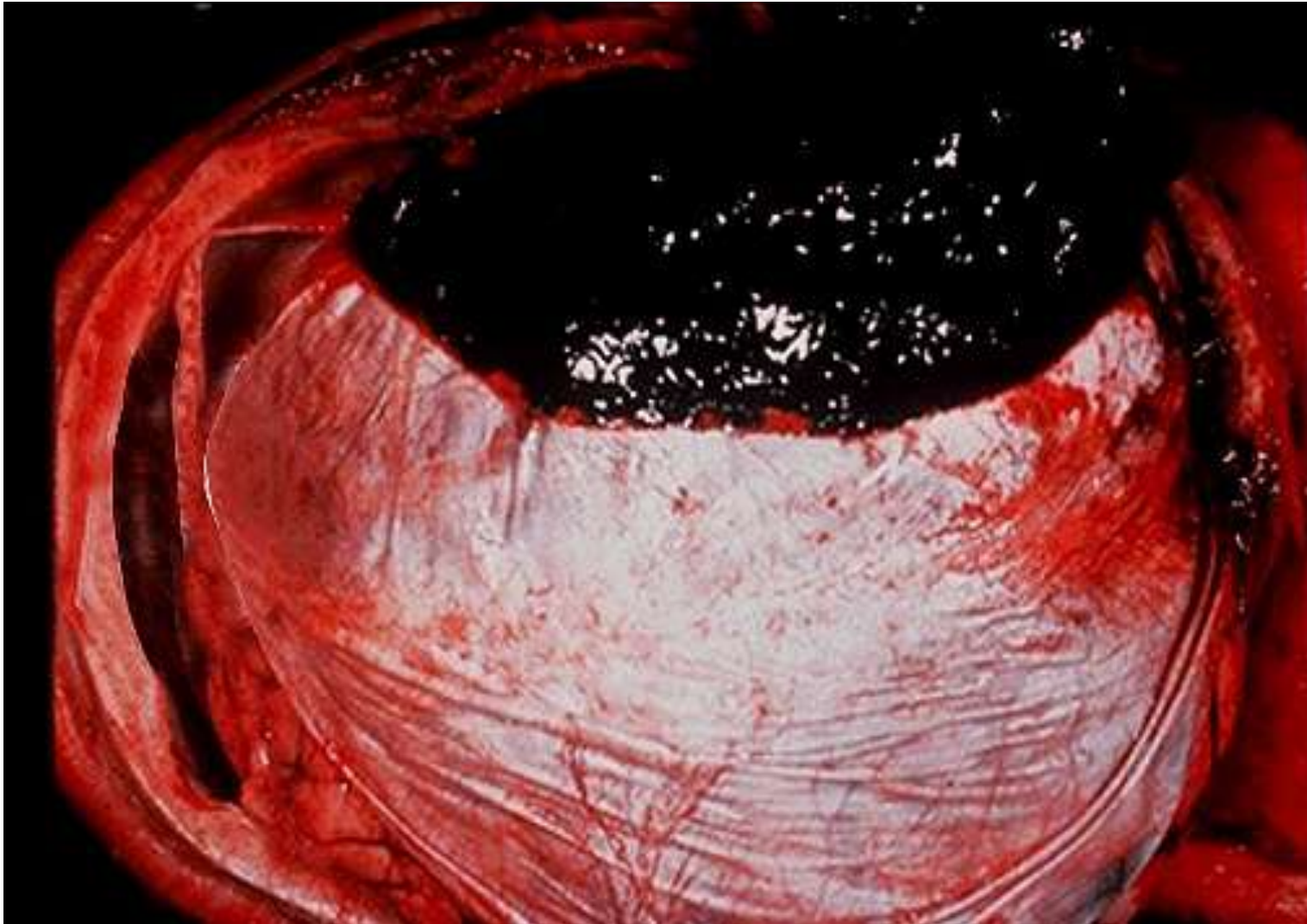
- In children in whom the skull is deformable, a temporary displacement of skull bones leading to lacerations of a vessel can occur in the absence of skull fracture

- Once a vessel is torn, blood accumulating under arterial pressure can dissect the tightly applied dura away from the inner skull surface producing a epidural hematoma that compresses the brain surface.



- *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

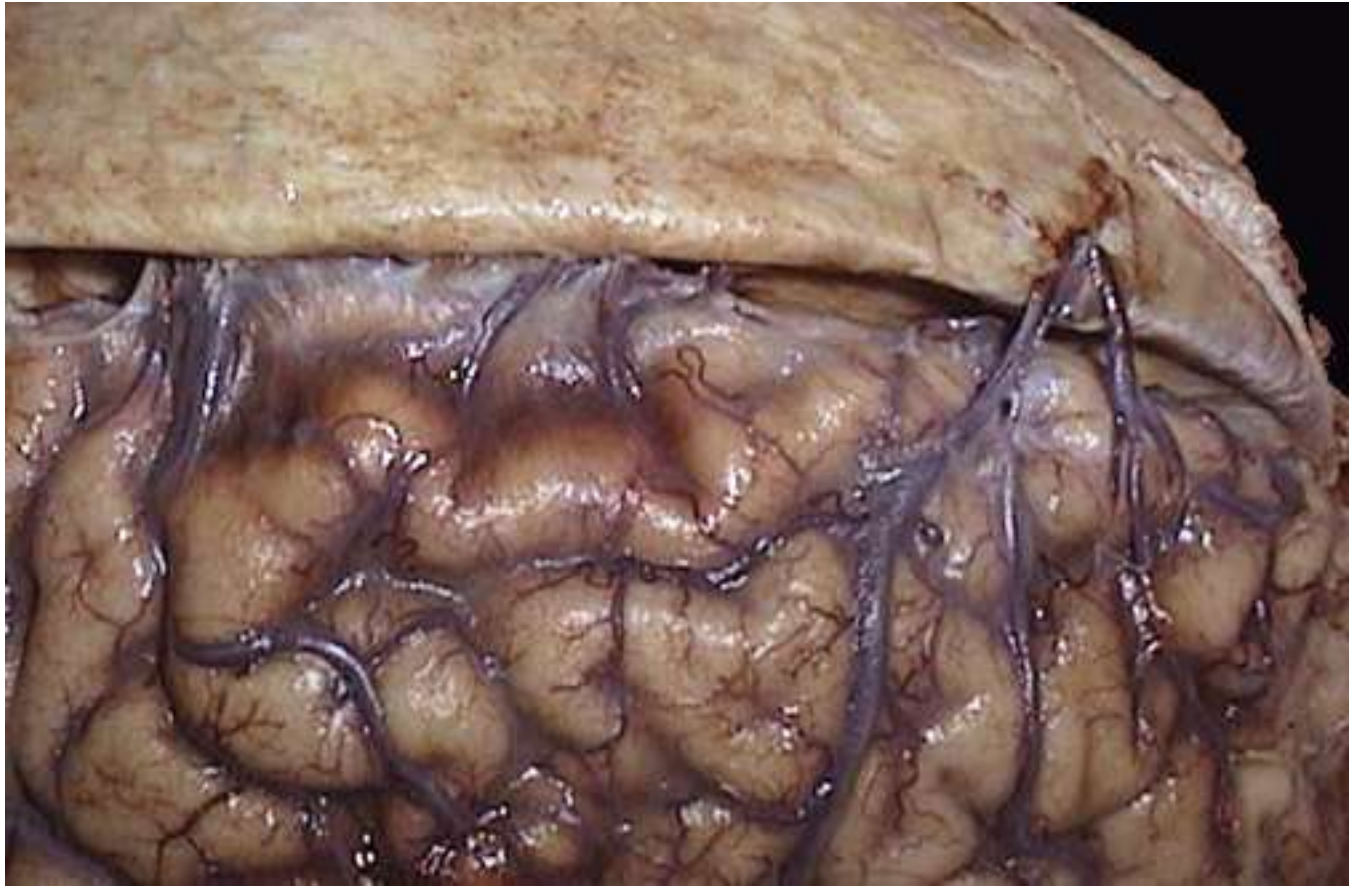


## B. Subdural Hematoma

- The dura is composed of two layer,
  - a. The external collagenous layer
  - b. and inner border cell layer with scant fibroblasts and abundant extracellular space devoid of collagen

## Notes:

- 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



- 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

## 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.

# 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