Central Nervous System pathology-2

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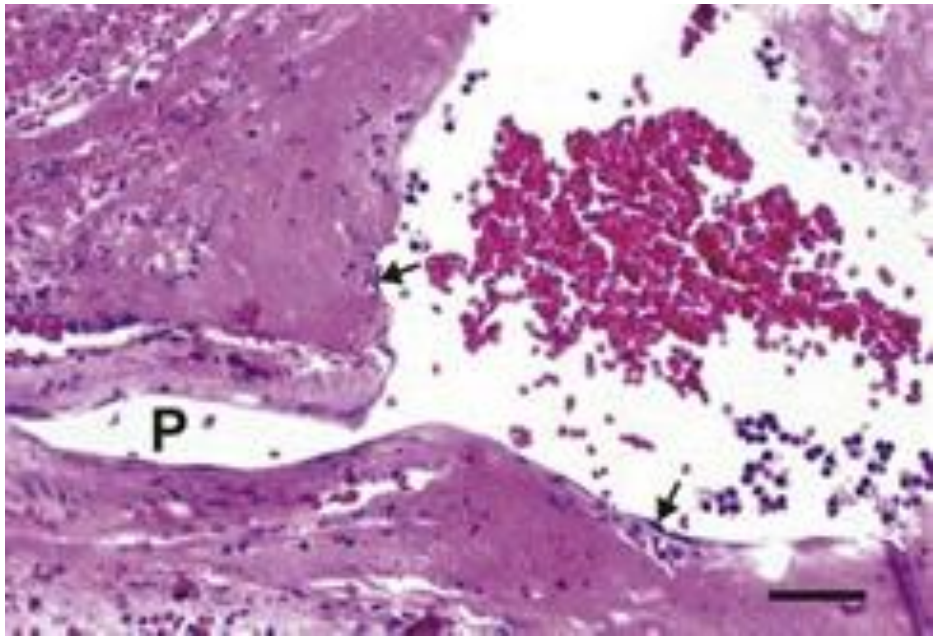
Hypertensive Cerebrovascular Disease

- Hyaline arteriolar sclerosis of the deep penetrating arteries & arterioles that supply the basal ganglia, the hemispheric white matter, & the brain stem.
- Affected arteriolar walls are weakened → vulnerable to rupture.
- In some instances, minute aneurysms (Charcot-Bouchard microaneurysms) form in vessels less than 300 µm in diameter.
- In addition to massive intracerebral hemorrhage, several other pathologic outcomes are related to hypertension.



I. **Slit hemorrhage** Rupture of the small-caliber penetrating vessel→ small hemorrhages.

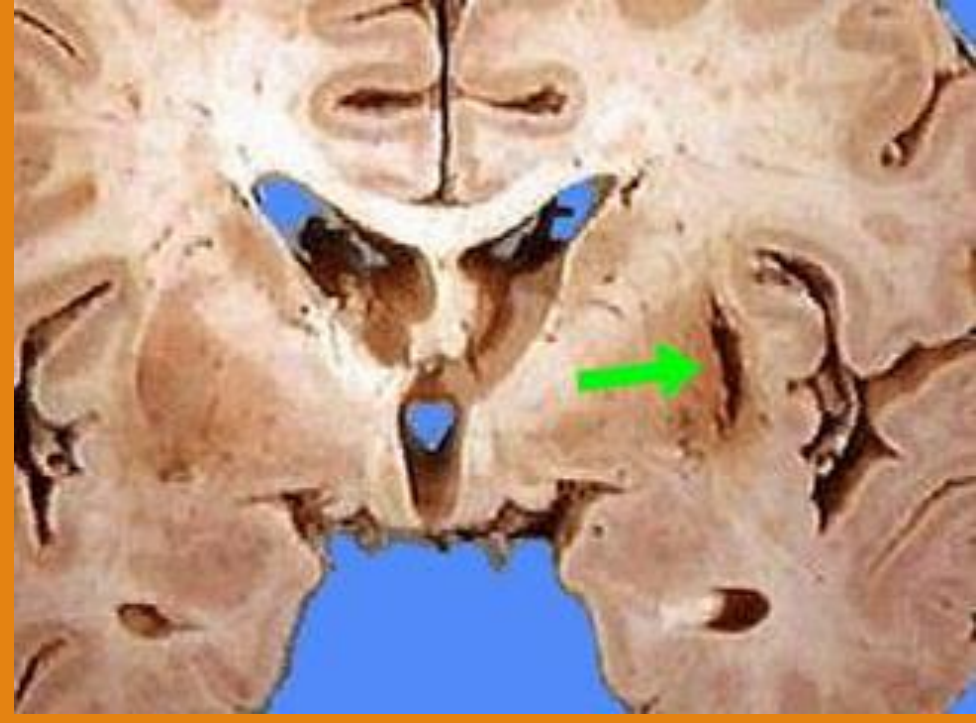
• After resorption → a slitlike cavity (slit hemorrhage) surrounded by brownish discoloration

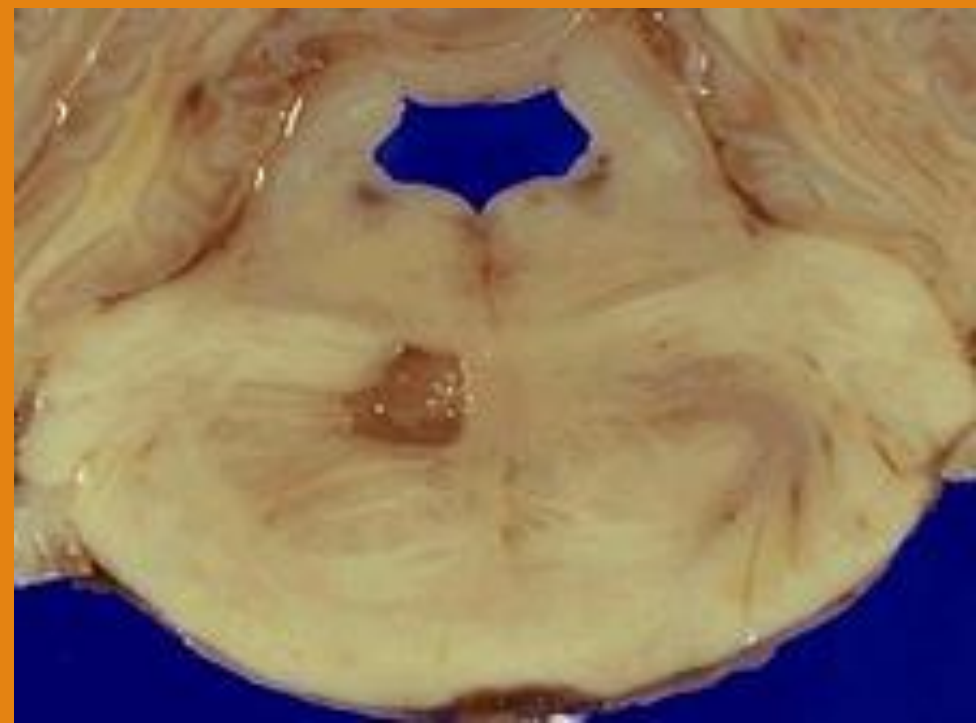
II**. Lacunes or lacunar infarcts:** small cavitary infarcts (few millimeters in size).

• Location: most commonly in deep gray matter (basal ganglia and thalamus), internal capsule, deep white matter, the pons.

• Caused by occlusion of a single penetrating branch of a large cerebral artery.

• Depending on location, lacunes can be silent clinically or cause significant neurologic impairment.





III. Acute hypertensive encephalopathy:

- Most often is associated with sudden sustained increases in diastolic blood pressure to greater than 130 mm Hg.
- It is characterized by increased intracranial pressure & global cerebral dysfunction.
- Symptoms: headaches, confusion, vomiting, convulsions, & sometimes coma. Rapid therapeutic intervention to reduce the BP is essential.
- Postmortem examination may show brain edema, with or without transtentorial or tonsillar herniation.
- Petechiae and fibrinoid necrosis of arterioles in the gray matter and white matter may be seen microscopically.

Vascular Malformations

Vascular malformations of the brain are classified into four principal types based on the nature of the abnormal vessels:

1. Arteriovenous malformations (AVMs)
2. Cavernous malformations
3. Capillary telangiectasias
4. Venous angiomas.

Arteriovenous malformations (AVMs)

- The most common Vascular malformation.
- Males twice as females Most commonly manifest between 10-30 years of age →seizures, intracerebral hemorrhage, or a subarachnoid hemorrhage. In the newborn period, large AVMs may lead to high-output congestive heart failure because of blood shunting from arteries to veins.
- Most dangerous type of vascular malformation → risk for bleeding Multiple AVMs can be seen in the setting of hereditary hemorrhagic telangiectasia; an AD condition often associated with mutations affecting the TGFβ pathway.

CENTRAL NERVOUS SYSTEM TRAUMA

- A significant cause of death and disability.
- The severity & site of injury affect the outcome; injury of several cubic centimeters of brain parenchyma may be clinically silent (e.g.; frontal lobe), severely disabling (e.g.; spinal cord), or fatal (e.g.; brain stem).
- A blow to the head may be penetrating or blunt; and may cause an open or a closed injury.
- The magnitude & distribution the traumatic brain lesions depend on:

(1)shape of object causing the trauma.

(2)force of impact.

(3)whether the head is in motion at the time of injury.

- External signs of head injury does not correlate to how Severe brain damage
- Injuries may involve the parenchyma, the vasculature, or both

Traumatic Parenchymal Injuries

1- Concussion.

2- Contusions.

3-Diffuse axonal injury.

4- Traumatic Vascular Injury.

1-Concussion

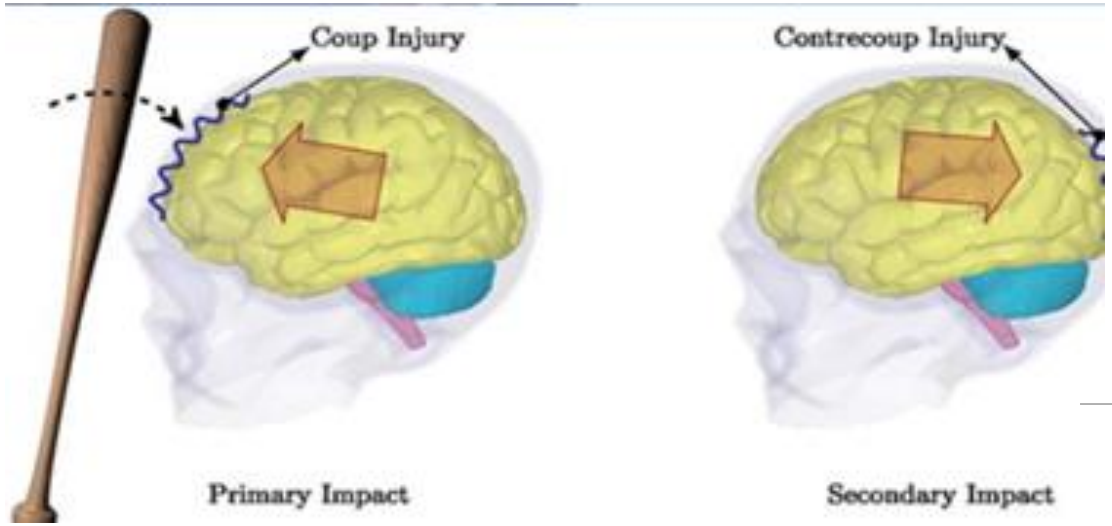
A reversible altered brain function, with or without loss of consciousness.

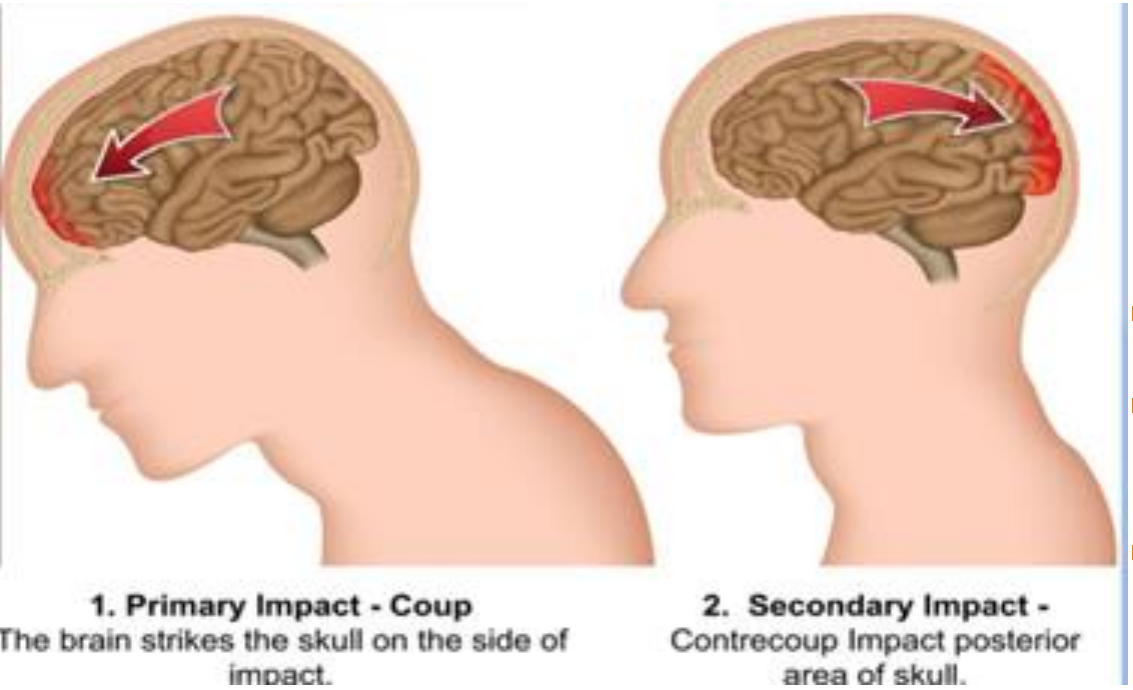
A transient neurologic dysfunction includes loss of consciousness, temporary respiratory arrest, and loss of reflexes.

Mechanism: a change in the momentum of the head when a moving head suddenly arrested by impact on a rigid surface Neurologic recovery is the norm, although amnesia for the event persists. Pathogenesis is unknown, may result from temporary deregulation of the reticular activating system in the brainstem.

Concussion-chronic traumatic encephalopathy

- Repeated episodes of concussion can result in persistent & profound neurologic deficits including cognitive impairment, parkinsonism, and others, and later development of neurodegenerative processes.
- Initially described in boxers (dementia pugilistica) Now recognized to occur in a wider range of settings, such as in athletes participating in contact sports (e.g.; American footballers, and boxers).
- A syndrome that termed chronic traumatic encephalopathy and is characterized by atrophic brain, enlarged ventricles & accumulation of tangles in cerebral cortex and other brain regions.





2-Contusions

- A blunt trauma to the brain.
- Mechanism: A blow to the surface of the brain transmitted through the skull→ rapid tissue displacement, disruption of vessels, hemorrhage, tissue injury, & edema. The crest of gyri are most susceptible than the depth of sulci→ closest to skull common in regions overlying rough & irregular inner skull surfaces: orbitofrontal regions & temporal lobe tips.
- A coup injury: at the site of impact.
- A contrecoup injury: at opposite the site of impact on the other side of the brain.
- The pia-arachnoid is not breached.

Morphology

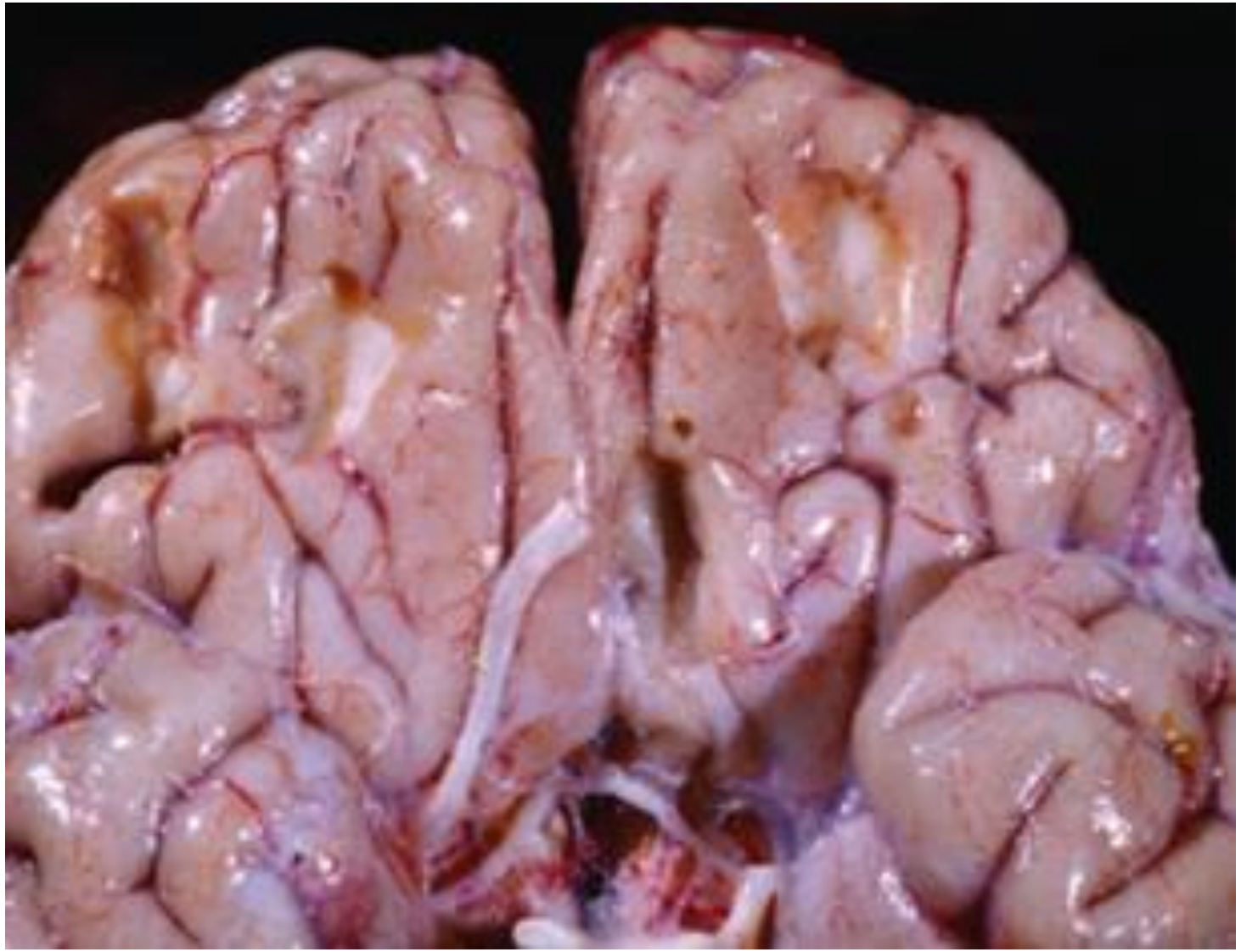
Contusions are wedge-shaped: widest aspect closest to the point of impact.

In Few hours: blood extravasates in the involved tissue, across cerebral cortex, and into the white matter & subarachnoid spaces. Functional effects are seen earlier than morphologic evidence of neuronal injury

Morphologic: > 24 hours: nuclear pyknosis, cytoplasmic eosinophilia, cellular disintegration. Inflammatory response > 48 hours: neutrophils preceding the macrophages.

In contrast with ischemic lesions, in which the superficial layer of cortex may be preserved, trauma affects the superficial layers most severely

A coronal section through the frontal lobes reveals extensive contusions involving the inferior gyri. This was a contracoup injury from a fall in the bathtub by an elderly person.



Morphology: Remote (old) contusions

characteristically appear as depressed, retracted, yellowish brown patches involving the crests of gyri. These lesions show gliosis and residual hemosiderin-laden macrophages.

3-Diffuse axonal injury

- Caused by accelerating and decelerating motion or angular acceleration (rotation of the head), even in the absence of impact
- The movement of one region of brain relative to another is thought to disrupt axonal integrity and function → injury (stretching and tear)
- Affect white matter (corpus callosum, paraventricles, hippocampus…etc) & at junction of grey & white matter
- In the Up to 50% of patients who develop coma shortly after trauma → white matter damage and diffuse axonal injury.
- Morphology: axonal swellings that appear within hours of the injury.
- Can have devastating consequences (Post-traumatic dementia ---- vegetative state)

4-Traumatic Vascular Injury

Leading to hemorrhage, depending on the affected vessel, the hemorrhage may be epidural, subdural, subarachnoid, or intraparenchymal occurring alone or in combination.

Subarachnoid & intraparenchymal hemorrhages most often occur at sites of contusions and lacerations.

Patterns of hemorrhage in CNS

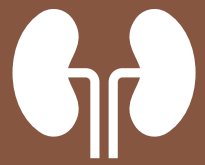
Location	Etiology	Additional Features
Epidural space	Trauma	Usually associated with a skull fracture (in adults); rapidly evolving neurologic symptoms requiring intervention
Subdural space	Trauma	Level of trauma may be mild; slowly evolving neurologic symptoms, often with a delay from the time of injury
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)

Epidural Hematoma

- Normally the dura is fused with the periosteum on the internal surface of skull.
- Dural vessels, most importantly the middle meningeal artery are vulnerable to traumatic injury especially with skull fracture in which the fracture tears the course of the vessel.
- Once a vessel tears, blood accumulates under arterial pressure and dissects the tightly applied dura away from the inner skull surface producing a hematoma that compresses the brain surface Clinically, patients can be lucid for several hours after the traumatic event before neurologic signs appear.
- Acute presentation: An epidural hematoma may expand rapidly & constitutes a neurosurgical emergency necessitating prompt drainage & repair to prevent death.

Traumatic Vascular Injury -Epidural Hematoma

Rapid movement of the brain during trauma can tear the bridging veins that extend from the cerebral hemispheres through the subarachnoid and subdural space to the dural sinuses. Their disruption produces bleeding into the subdural pace. Because the inner cell layer of the dura is quite thin and in very close proximity to the arachnoid layer, the blood appears to be between the dura and arachnoid, but in reality, it is between the two layers of the dura.



1. older adults: with brain atrophy, the bridging veins are stretched out & the brain has additional space within which to move, accounting for the higher rate of subdural hematomas in.

2. Infants: because their bridging veins are thin-walled

Subdural Hematoma

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

They are most common over the lateral aspects of the cerebral hemispheres May be bilateral. Neurologic signs: attributable to the pressure exerted on the adjacent brain.

Symptoms are most often nonlocalizing, taking the form of headache, confusion, & slowly progressive neurologic deterioration.

Morphology

Appears as a collection of freshly clotted blood apposed to the contour of the brain surface without extension into the depths of sulci Underlying brain is flattened, subarachnoid space is often clear.

Week 1: organized by lysis of the clot.

Week 2: Growth of granulation tissue from the dural surface into the hematoma.

1–3 months: fibrosis Subdural hematomas commonly rebleed→ from the thin-walled vessels of the granulation tissue→ microscopic findings with hemorrhages of varying ages.

Thank you