

Central Nervous System – Pathology II

1. Hypertensive Cerebrovascular Disease

Hyaline arteriolar sclerosis affects deep penetrating arteries supplying basal ganglia, thalamus, deep white matter, and brain stem → weakened walls → rupture risk.

- Charcot–Bouchard microaneurysms: vessels $<300\ \mu\text{m}$.

Complications:

- 1) Slit hemorrhages: small vessel rupture → resorption → slit-like cavity with brown discoloration.
- 2) Lacunar infarcts: small cavitory infarcts (mm). Locations: basal ganglia, thalamus, internal capsule, pons. Caused by occlusion of single penetrating artery. Can be silent or cause deficits.
- 3) Acute hypertensive encephalopathy: sudden diastolic BP $>130\ \text{mmHg}$ → \uparrow ICP, global cerebral dysfunction. Symptoms: headache, confusion, vomiting, seizures, coma. Pathology: brain edema \pm herniation, petechiae, fibrinoid necrosis.

Exam Pitfall: Lacunes = ischemic (occlusion), not hemorrhagic.

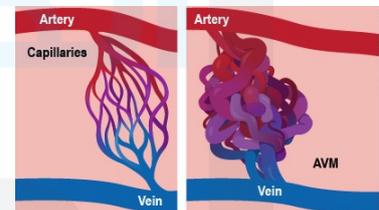
2. Vascular Malformations

Four types: AVMs, cavernous malformations, capillary telangiectasias, venous angiomas.

AVMs: most common & most dangerous. Male predominance. Present age 10–30 years with seizures or hemorrhage (intracerebral or subarachnoid). Newborns may develop high-output heart failure.

Associated with hereditary hemorrhagic telangiectasia (AD, TGF β pathway).

Exam Pitfall: AVMs → highest bleeding risk.



3. CNS Trauma – General Principles

Severity depends on site and extent of injury. External injury does NOT correlate with brain damage. Injuries may affect parenchyma, vessels, or both. Trauma can be penetrating or blunt; open or closed.



4. Traumatic Parenchymal Injuries

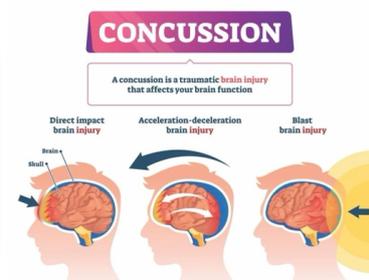
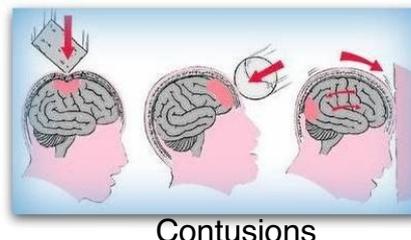
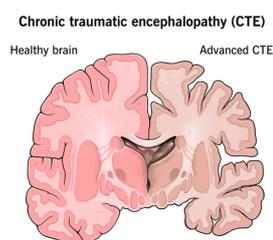
Concussion: reversible altered brain function \pm loss of consciousness. Mechanism: sudden deceleration. Recovery is usual but amnesia persists. Pathogenesis likely transient dysfunction of reticular activating system.

Chronic Traumatic Encephalopathy (CTE): repeated concussions → cognitive decline, parkinsonism. Seen in boxers and contact sports. Pathology: brain atrophy, ventricular enlargement, neurofibrillary tangles.

Contusions: blunt trauma. Common at crests of gyri (orbitofrontal & temporal poles).

- Coup: at impact site.
- Contrecoup: opposite side.

Pia-arachnoid intact. Wedge-shaped hemorrhagic lesions. Old contusions → depressed yellow-brown scars with gliosis.



Diffuse Axonal Injury (DAI): acceleration–deceleration or rotational forces. Affects white matter (corpus callosum, grey–white junction). Major cause of coma after trauma. Morphology: axonal swellings. Outcome: severe disability, vegetative state.

5. Traumatic Vascular Injuries

Epidural hematoma: arterial (middle meningeal artery). Lucid interval then rapid deterioration. Biconvex hematoma → neurosurgical emergency.

Subdural hematoma: venous (bridging veins). Common in elderly & infants. Symptoms are gradual, non–localizing. Morphology evolves from clot → granulation tissue → fibrosis; rebleeding common.

Exam Pitfalls: Epidural = artery + lucid interval. Subdural = veins + gradual symptoms.

