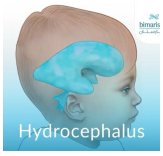
# CNS Pathology – High Yield Review

## Intracranial Pressure

Disorders that cause dangerous increases in intracranial contents volume leading to increased intracranial pressure. Includes cerebral edema, hydrocephalus, and herniation.

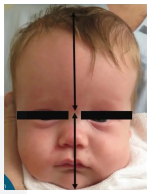
## Cerebral Edema

Accumulation of excess fluid within the brain parenchyma. Vasogenic edema results from blood–brain barrier disruption with extracellular fluid accumulation. Cytotoxic edema results from intracellular fluid accumulation due to neuronal and glial injury. Grossly, the brain is soft with flattened gyri, narrowed sulci, and compressed ventricles.



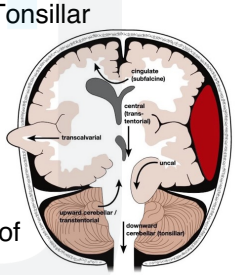

## Hydrocephalus

Increase in CSF volume within the ventricular system. Noncommunicating hydrocephalus is caused by obstruction within the ventricular system. Communicating hydrocephalus is caused by reduced CSF resorption. In infants the head enlarges; after suture closure intracranial pressure increases without head enlargement.



## Herniation

Displacement of brain tissue across rigid dural folds due to increased intracranial pressure, leading to vascular compromise and infarction. Types include subfalcine, transtentorial (uncinate), and tonsillar herniation. Tonsillar herniation is often fatal due to medullary compression.

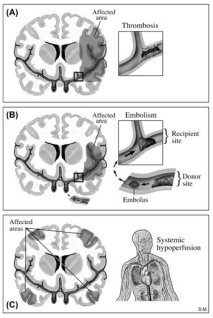



## Cerebrovascular Diseases

Brain disorders caused by pathological processes involving blood vessels and represent a major cause of neurologic morbidity and mortality.

## Hypoxia, Ischemia, and Infarction

The brain requires continuous oxygen and glucose supply. Stroke refers to acute onset of neurologic deficits caused by thrombotic occlusion, embolic occlusion, or vascular rupture.



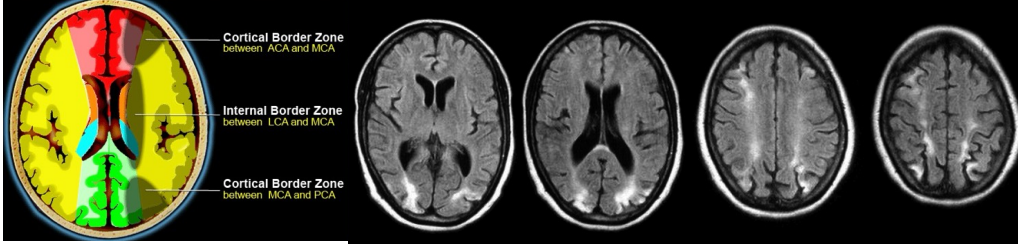

## Global Cerebral Ischemia

Occurs with severe hypotension such as cardiac arrest or shock. Vulnerable neurons include hippocampal pyramidal cells and cerebellar Purkinje cells. Severe cases lead to vegetative state or brain death.

## Morphology of Infarction

Acute changes include red neurons and neutrophil infiltration. Subacute changes include tissue necrosis, macrophage influx, vascular proliferation, and reactive gliosis. Repair after two weeks is characterized by removal

of necrotic tissue and gliosis.

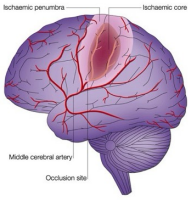



## Watershed Infarcts

Occur at distal arterial territories, most commonly between ACA and MCA, typically after hypotensive episodes, producing wedge-shaped cortical necrosis.

## Focal Cerebral Ischemia

Results from arterial occlusion with infarction in the affected vascular territory. Collateral flow limits cortical damage but deep structures lack collateral supply.




## Embolic Infarctions

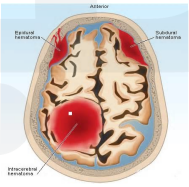
More common than thrombotic infarctions. Often arise from cardiac mural thrombi or atherosclerotic plaques. MCA territory is most frequently affected.

## Thrombotic Occlusions

Occur on atherosclerotic plaques, commonly at carotid bifurcation, MCA origin, or basilar artery. Occlusion of small penetrating arteries causes lacunar infarcts associated with hypertension.

## Hemorrhagic vs Nonhemorrhagic Infarcts

Nonhemorrhagic infarcts result from acute vascular occlusion. Hemorrhagic infarcts result from reperfusion of ischemic tissue and show petechial hemorrhages.



## Intracranial Hemorrhage

Caused by hypertension, vascular malformations, tumors, or trauma. Primary intraparenchymal hemorrhage is most commonly due to chronic hypertension.

## Subarachnoid Hemorrhage and Saccular Aneurysms

Most commonly caused by rupture of a berry aneurysm. Presents with sudden thunderclap headache. Risk of rupture increases with aneurysm size and is associated with vasospasm and hydrocephalus.