Stroke and increased intracranial pressure/patho1 summary.

Cerebrovascular diseases = CVA= stroke

-CVA is a major cause of death.
-CVA is the most common cause of neurologic morbidity.
-mechanisms:
1. thrombi
2.emboli
3. vascular rupture

-**Stroke:** clinical term applies to all three when symptoms are **acute.**

Definition:
• Stroke: rapidly developing symptoms and signs of loss of focal CNS function lasting for 24 hs or leading to death.

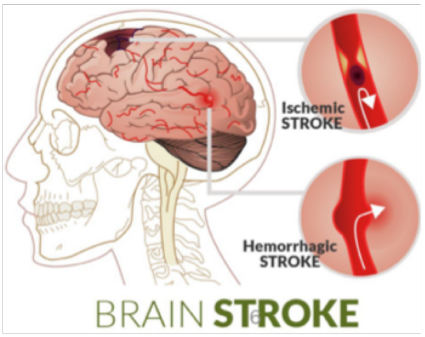
- symptoms develop quickly (within secs or mins) but they persist for at least 24 hs.

• If the symptoms last for less than 24 hours, it is called: transient ischemic attack.

Types of strokes

TWO TYPES OF STROKES:
• 1. **Ischemic** caused by vascular obstruction by a thrombus or an embolus.

• 2. **Hemorrhagic** caused by vessel rupture secondary to several vascular diseases, like **hypertension** or **vasculitis**.



- **Ischemic stroke:** 1. Thrombotic occlusions

• Atherosclerosis of cerebral arteries causing thrombosis.
Common sites:
1. Carotid bifurcation
2. Origin of middle cerebral artery
3. Ends of basilar artery

- **Ischemic stroke:** 2. Embolic infarcts

• More common than thrombotic infarcts
• Source:
1. **cardiac mural thrombi,** arise due to myocardial dysfunction, valvular disease, and atrial fibrillation.

• 2. **arterial atheroma** in carotid arteries or aortic arch

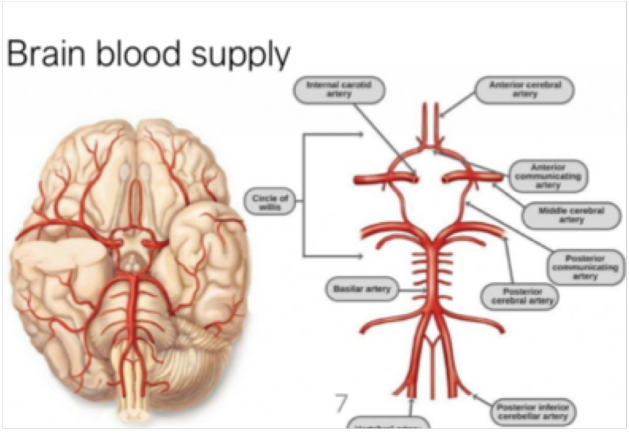
• 3. **venous thrombi** crossing to arterial circulation through cardiac defects = paradoxical embolism... DVT, fat emboli.

Important
• Most common site of embolic occlusion: **middle cerebral artery,** a direct extension of the internal carotid.

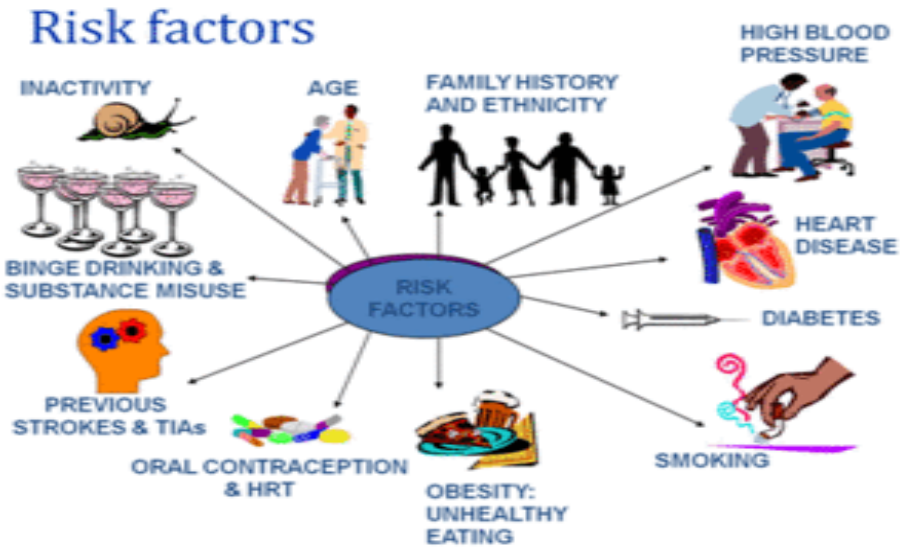
• Emboli lodge where vessels branch or in stenotic areas caused by atherosclerosis.

Hemorrhagic stroke causes hemorrhagic infarcts.

• Can be caused by haemorrhage from a ruptured vessel.
• OR are due to reperfusion through collaterals or after dissolution of emboli.



Stroke risk factors: same risk factors of atherosclerosis



Note:

• Ischemic stroke is treated by anticoagulants, whereas if you use anticoagulants in hemorrhagic stroke you might kill the patient.

Clinical features of stroke

• Signs and symptoms= **FAST**

• The main symptoms of stroke can be remembered with the word FAST: **Face-Arms-Speech-Time.**

• **Face** –dropped on one side, may not be able to smile or mouth or eye may have dropped.

• **Arms** – patient may not be able to lift both arms and keep them there because of arm weakness or numbness in one arm.

• **Speech** –slurred or garbled, or unable to talk at all despite appearing to be awake.

• **Time** – it is time to dial emergency team immediately if you see any of these signs or symptoms.

Transient ischemic attack (TIA)

- Sometimes, stroke is preceded by transient ischemic attacks TIA.
- means that the supply of blood to the brain is temporarily interrupted, causing a "mini-stroke" often lasting between 30 min and several hs.
- It should be treated seriously as they are often a warning sign that there is risk of having a full stroke in the near future.

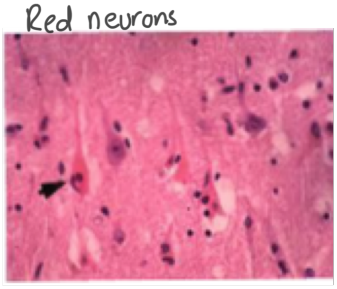
- **Morphology/non-haemorrhagic infarcts, macroscopic appearance:**
- By 48 hours: pale, soft swollen area.

- Day 2-10: gelatinous and friable.

- Day 10 to week 3: liquefaction ending in a fluid filled cavity.

- **Morphology / non-hemorrhagic microscopic appearance:**

- microscopic appearance of brain infarcts is divided into:
- Early changes
- Subacute changes
- repair



- early (first 24 hours): red neurons + edema + neutrophils

- subacute (24 hours to 2 weeks): macrophages, gemistocytic
astrocytes.
- repair (after 2 weeks): gemistocytes regress, cavity persists

Early changes

• Acute neuronal cell damage= red neurons, followed by cytoplasmic eosinophilia then pyknosis and karyorrhexis

- Similar changes later on glial cells
- Then: neutrophilic infiltrate.

Subacute change

24 hours to 2 weeks

- Necrosis
- Macrophages
- Vascular proliferation
- Reactive gliosis

Repair

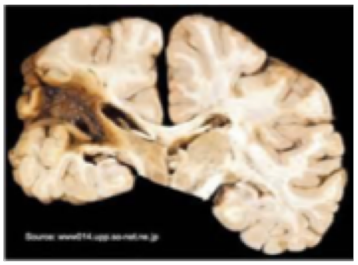
- After 2 weeks
- Removal of necrotic tissue
- Gliosis
- Loss of organized CNS structure
- Astrocytes=main cells responsible for repair and scar formation (gliosis).

• Injury... Causes:

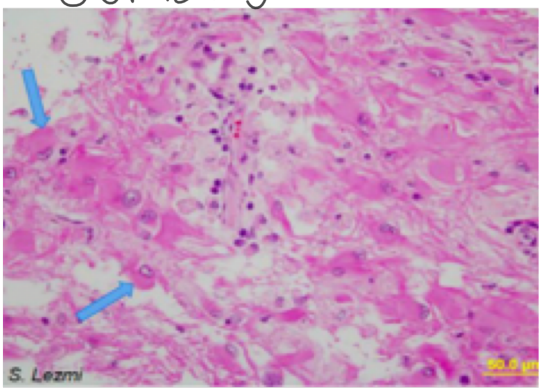
1. hypertrophy and hyperplasia in astrocytes.
2. enlarged nuclei
3. prominent nucleoli.
4. increased pink cytoplasm.
5. increased, ramifying processes

These changes in astrocytes: gemistocytic astrocyte.

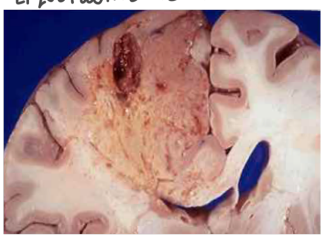
Brain infarct

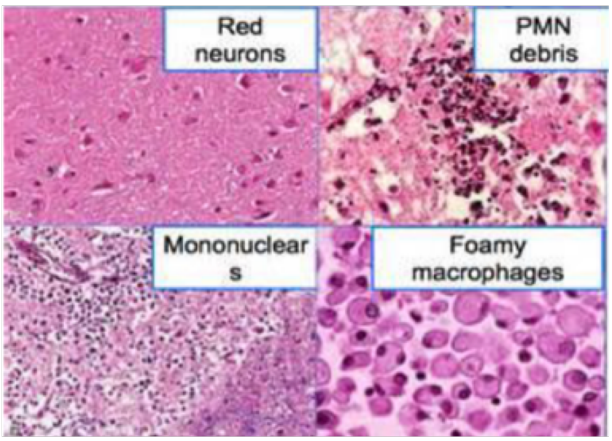


Gemistocytes

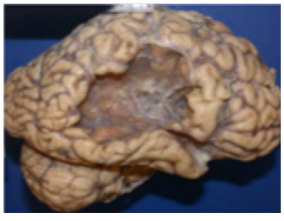


Liquefactive necrosis





Old infarct is resolved = cavity



Intracranial pressure

What's inside the cranium?

• 80% brain tissue (including intracellular and interstitial fluid
which is around 75% of brain weight) • 10% blood
• 10% CSF (cerebrospinal fluid).
If any of these components increases, the intracranial pressure increases.
• It’s the pressure inside the skull and is measured in millimeters of mercury
• at rest, it is normally 7–15 mmHg for a supine adult.
• If pressure in the cranium is higher than this upper limit= increased intracranial pressure (= intracranial hypertension.

MonroKellie hypothesis

Intracranial volume= VCNS +VCSF + VBlood + Vlesion

• This hypothesis indicated that any space occupying lesion in the brain will increase the volume inside the cranium and this will result in increased intracranial pressure.
• Space occupying lesions occur with all major brain diseases (except degenerative diseases). Ex: brain tumors, trauma, stroke, haemorrhage.

Causes of increased intracranial pressure:

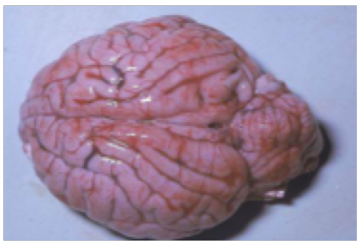
- **Mass effect**: brain tumor, hematoma, or abscess.
- **Generalized brain swelling**: ischemia, hypertension
- **Increase in venous pressure**: heart failure.
- **Obstruction to CSF flow and/or absorption or increased CSF production**: hydrocephalus.
- **Idiopathic or unknown**

Clinical presentation:

- **Early stages:** nonspecific symptoms like headache and vomiting, patients might have Cushing reflex (Cushing response or Cushing triad) which manifests by: increased BP, bradycardia and irregular breathing.

- **More advances cases:** neurological manifestations including disturbed level of consciousness.

- **Later:** complications can occur, mainly herniation and seizures.



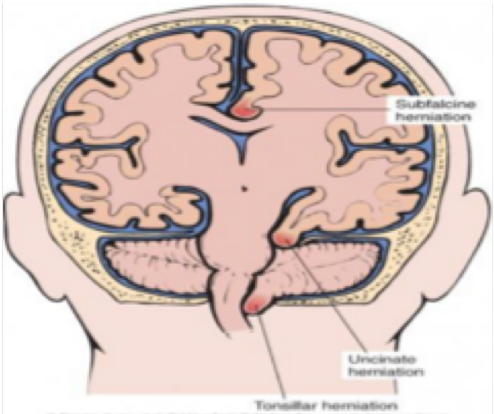
Brain edema= cerebral edema

- accumulation of excess fluid within the brain parenchyma.
- • Two types: vasogenic and cytotoxic edema... Usually coexist.

Herniation: a complication of brain edema

- Increased volume of tissue inside the skull causes Increased intracranial pressure which causes focal expansion of the brain tissue.
- Because the cranial vault is subdivided by rigid Dural folds (falx and tentorium).... The expanded brain tissue is displaced in relation to these folds.
- Expansion= herniation

herniation is a complication of increased intracranial pressure and it occurs in relation to margins of the Dural folds.



herniation: 3 types

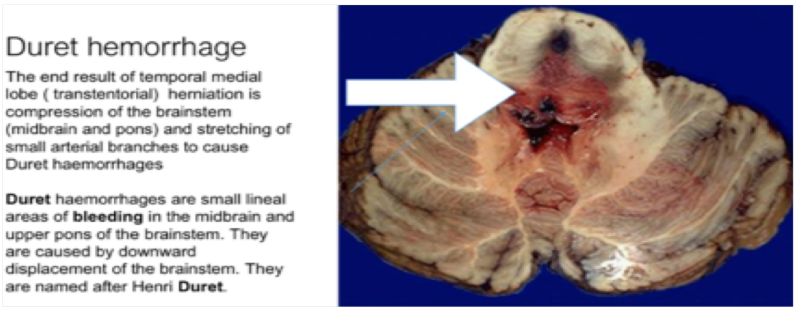
- Subfalcine = cingulate
- Transtentorial = uncinate
- Tonsillar.

1. Cingulate herniation

-Cingulate gyrus displaced under edge of falx
-Can cause compression of **anterior cerebral artery**; so the territory supplied by this artery can suffer ischemic damage and infarction if severe.

2. Transtentorial herniation

- Medial aspect of temporal lobe compressed against the free margin of the tentorium.
- the brain tissue is forced from supra-tentorial towards the infra-tentorial compartment.
- **Third cranial nerve** compressed... Dilated pupil, impaired ocular movement on the side of the lesion (ipsilateral side)
- **Posterior cerebral artery** can be affected... Ischemic injury to tissues supplied by it including visual cortex.
- Transtentorial herniation can cause hemorrhage in the midbrain and pons (Duret haemorrhage) which is usually fatal.



3. Tonsillar herniation

- Displaced cerebellar tonsils through foramen magnum.

- Brain stem compression... respiratory and cardiac centres in
medulla compromised.
- LIFE THREATENING