STROKE

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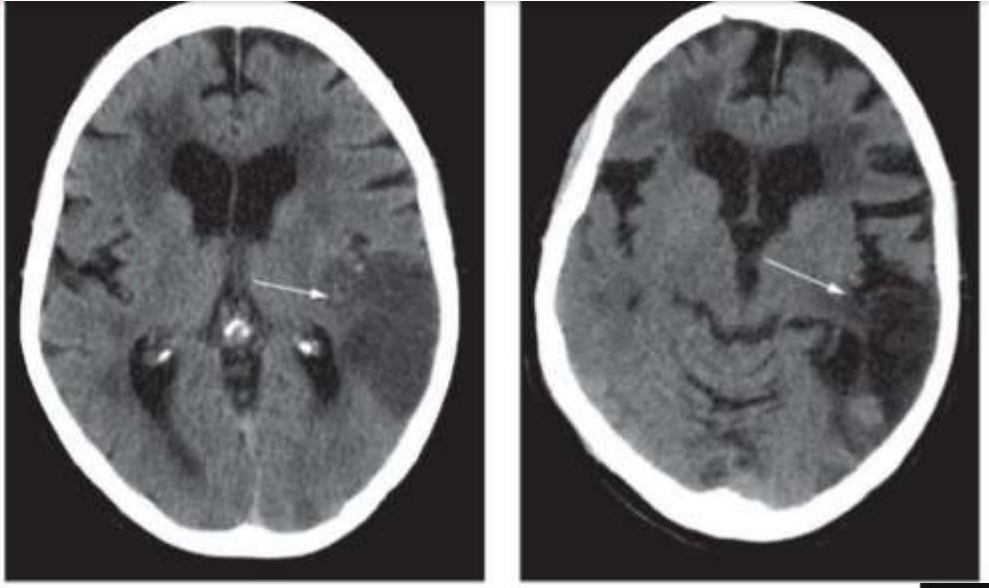
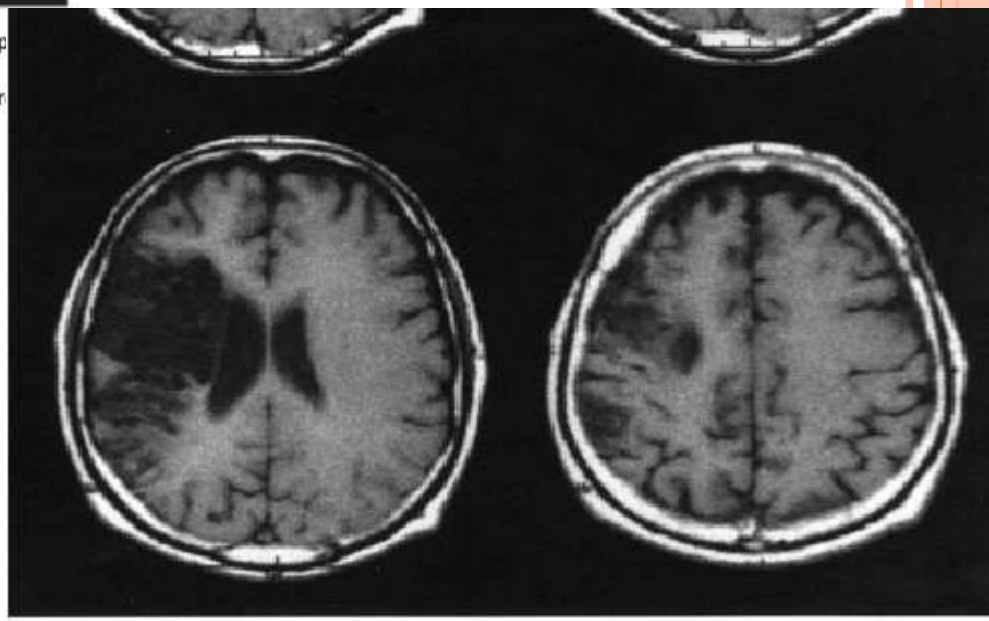
INTRODUCTION

- Acute loss of focal brain function with symptoms lasting more than 24 hours or leading to death.
- It is a rapidly developing clinical signs of focal disturbances of cerebral functions or pre-assumed vascular origin and of more than 24 hours of origin.
- Primary causes include inadequate blood supply to a part of brain or spontaneous hemorrhage into a part of brain or over surface of brain.
- "It is defined as an episode of focal neurologic dysfunction in which the autopsy, CT scan, or MRI Scan shows features consistent with focal brain infections or hemorrhage."- American Stroke Association.
- Also known as CVA or Cerebro-Vascular Accidents.



(Right) Initial CT scan a few hours after the onset of symptoms showing low attenuation in the p
with swelling causing effacement of the local sulci (arrow).
(left)CT scan a month later showing low attenuation gliotic change in the same territory and atr
of the lateral ventricle and widening of the sulci (arrow).

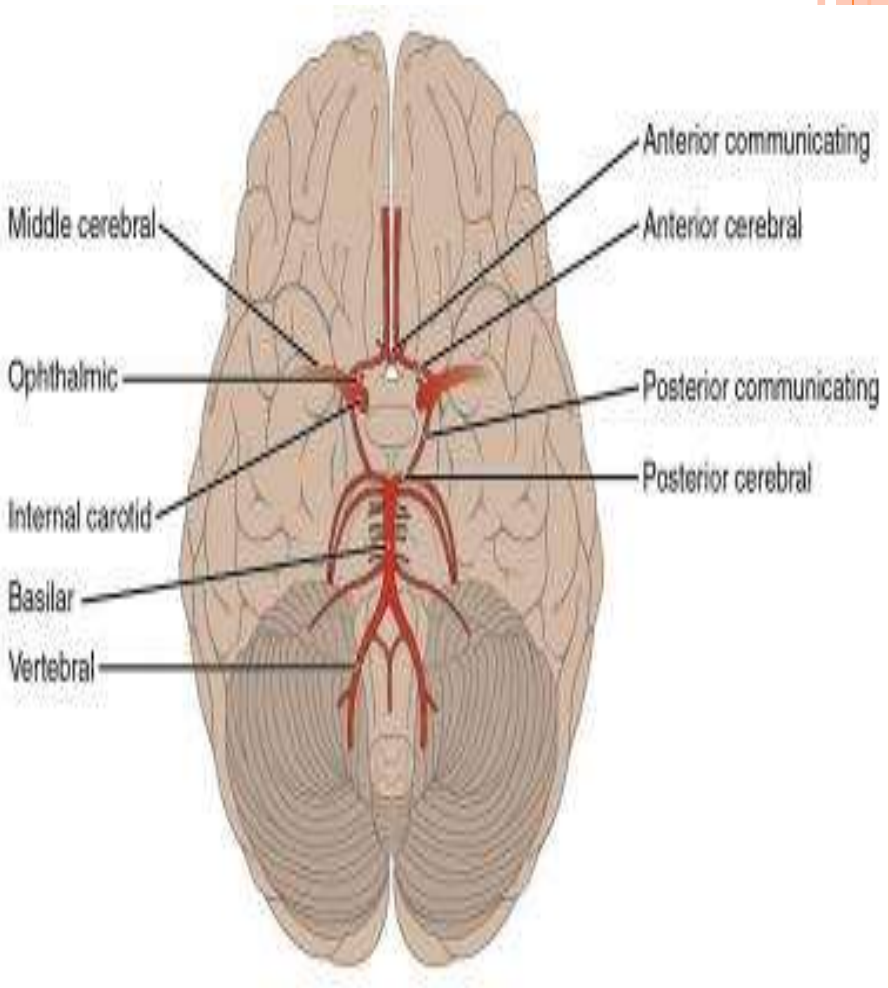


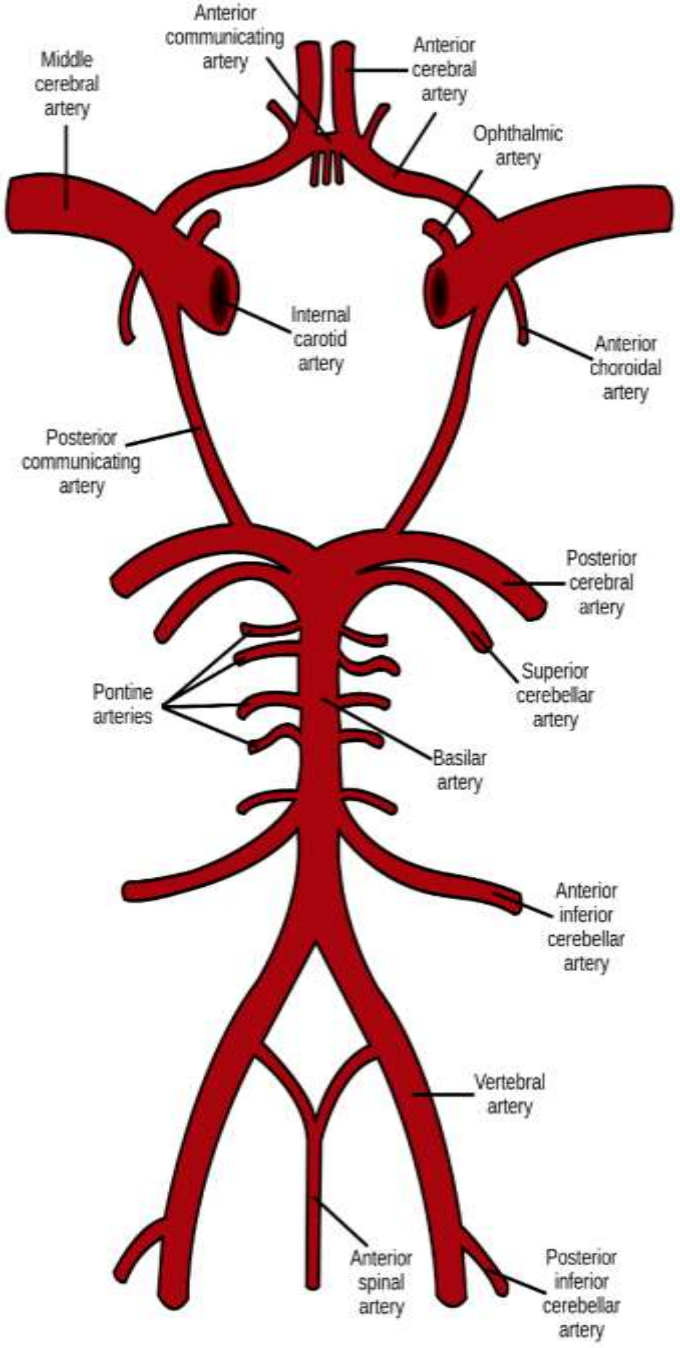
INCIDENCE

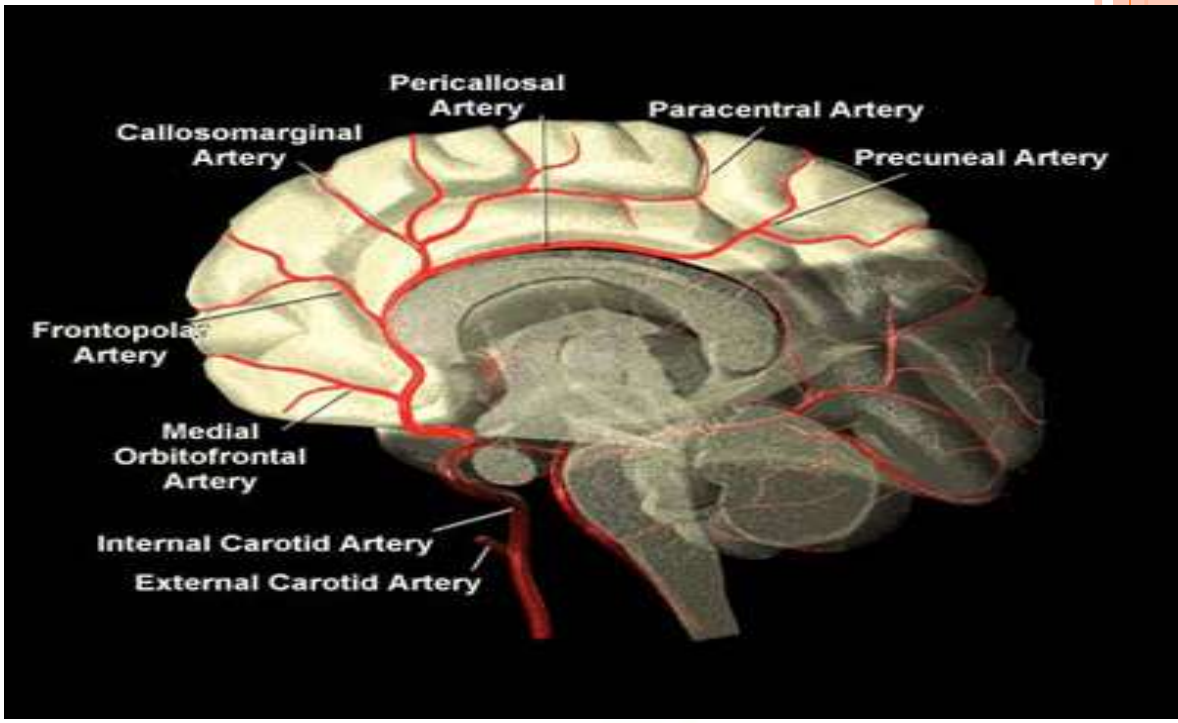
- In 1972, out of total population of 100,000 15.2 % males and 10.8 % in females incidences were seen.
- In 2012-2013 overall 3126 patients in India were identified with stroke with an annual incidence rate of 140/100,000.
- According to WHO, 15 million people suffer from stroke worldwide each year.

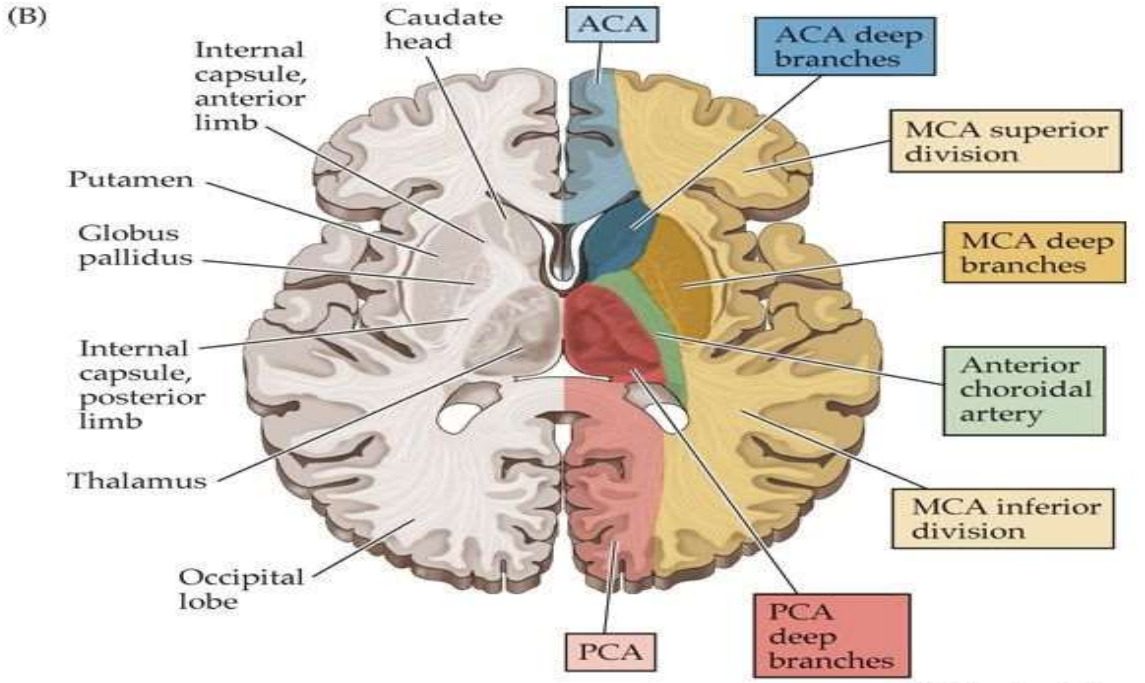
CIRCULUS ARTERIOSUS

- Greater part of brain is supplied by Circle of Willis.
- Anastomosis between the branches of vertebral artery and internal carotid artery.
- Cerebral flow represents approx 70 % of cardiac output.
- Normal flow of blood to brain is 50-60 mL/100 mg of brain tissue/ min.
- Requires continuous blood flow to deliver oxygen and glucose to the tissues.




Anterior communicating artery
Middle cerebral artery
Anterior cerebral artery
Ophthalmic artery
Internal carotid artery
Anterior choroidal artery
Posterior communicating artery
Posterior cerebral artery
Superior cerebellar artery
Pontine arteries
Basilar artery
Anterior inferior cerebellar artery
Vertebral artery
Anterior spinal artery
Posterior inferior cerebellar artery


Pericallosal Artery
Paracentral Artery
Callosomarginal Artery
Precuneal Artery
Frontopolar Artery
Medial Orbitofrontal Artery
Internal Carotid Artery
External Carotid Artery


(B)
Caudate head
ACA
ACA deep branches
Internal capsule, anterior limb
MCA superior division
Putamen
Globus pallidus
MCA deep branches
Internal capsule, posterior limb
Anterior choroidal artery
Thalamus
MCA inferior division
Occipital lobe
PCA
PCA deep branches
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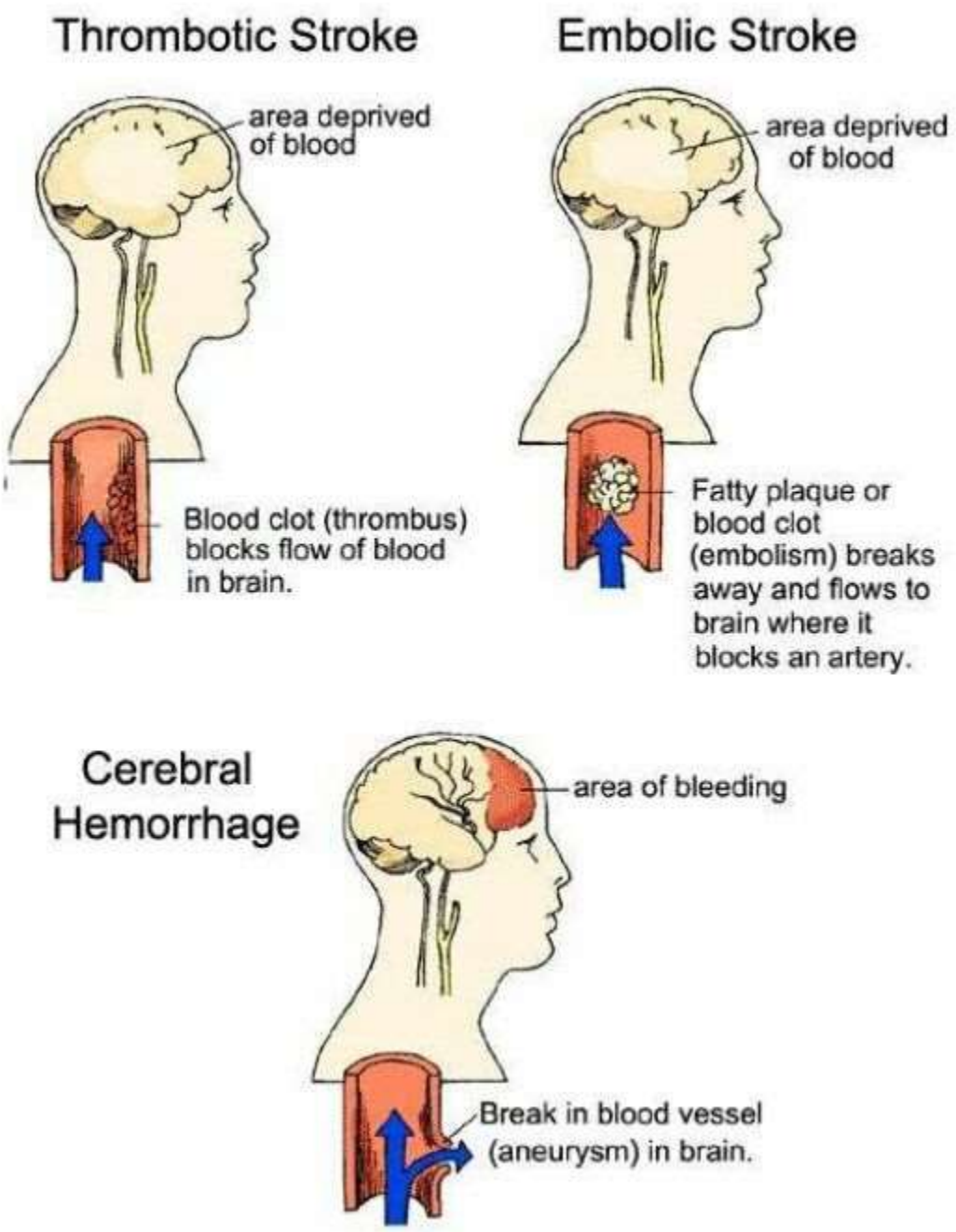
ETIOLOGY

- PRIMARY ETIOLOGIES:
  A. Atherosclerosis
  B. Emboli
  C. Haemorrhage
  D. Hypertension
  E. Ischemia
- SECONDARY ETIOLOG
  A. Smoking
  B. Alcohol consumption
  C. Brain tumour
  D. Trauma
  E. Obesity



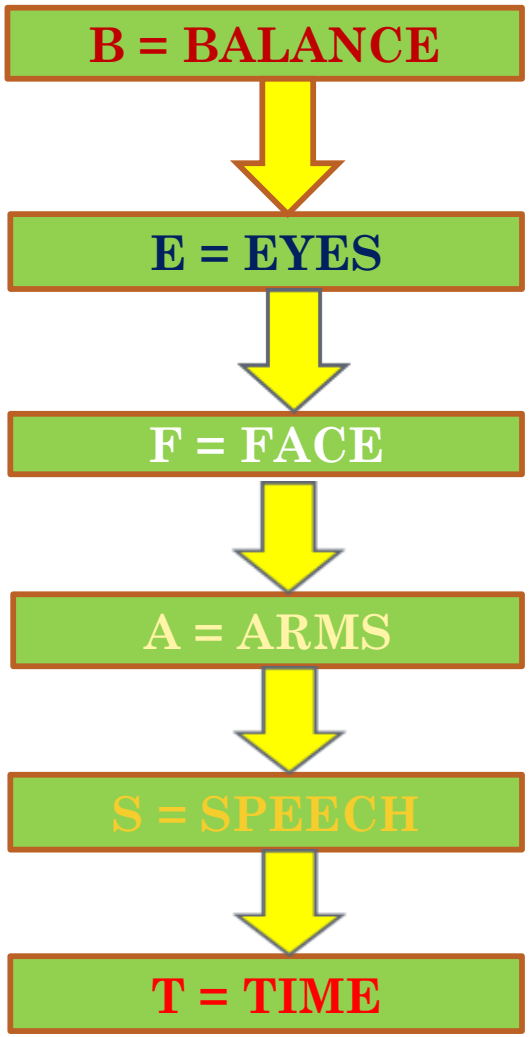
PATHOPHYSIOLOGY

- Basic 2 mechanism causing brain damage in stroke are ischemia and haemorrhage.

- Ischemic stroke is when the blood supply is decreased or ceased.
- Haemorrhagic stroke is when there is vascular compromise.

SIGNS AND SYMPTOMS

- Sudden numbness
- Weakness of unilateral face, arm or leg.
- Sudden confusion.
- Trouble in speaking or understanding.
- Dizziness.
- Loss of balance and coordination.
- Sudden, severe headache.
- Sudden nausea.

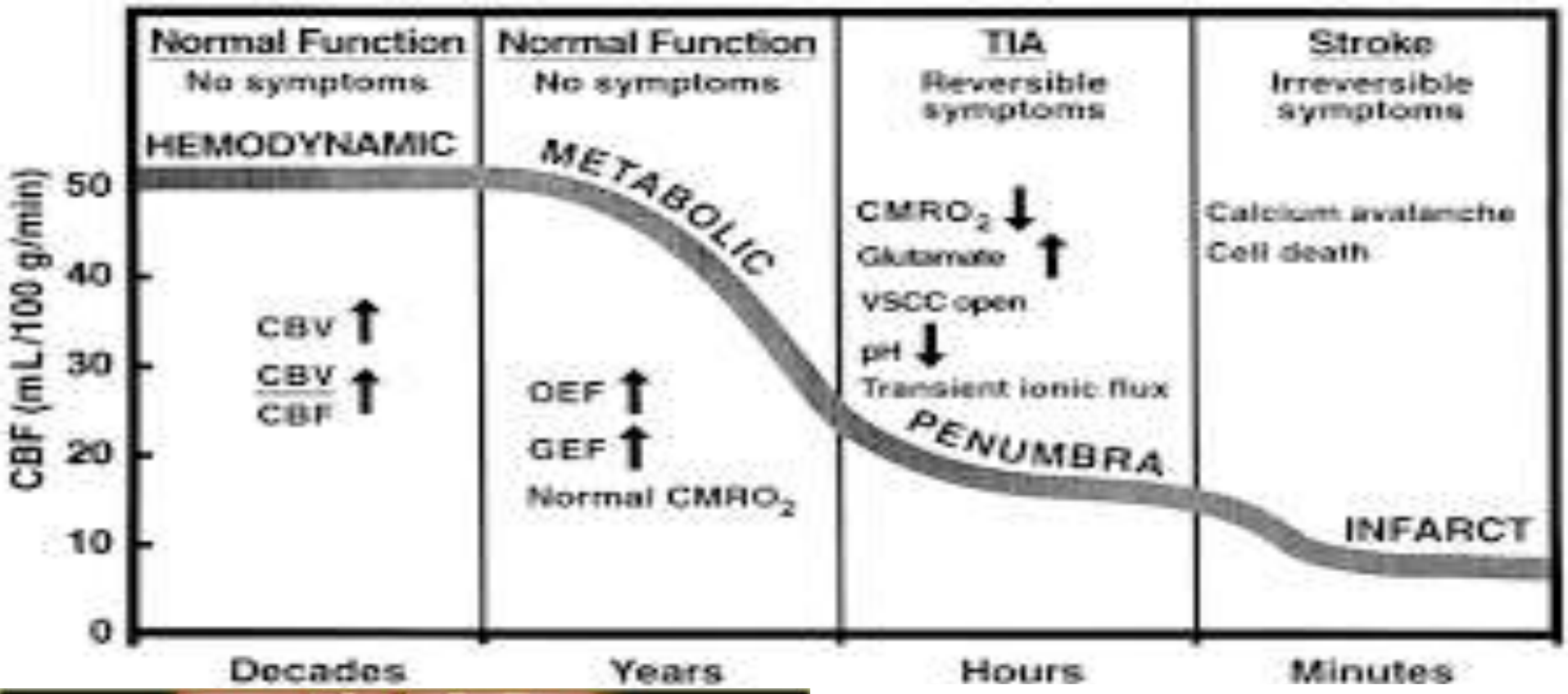


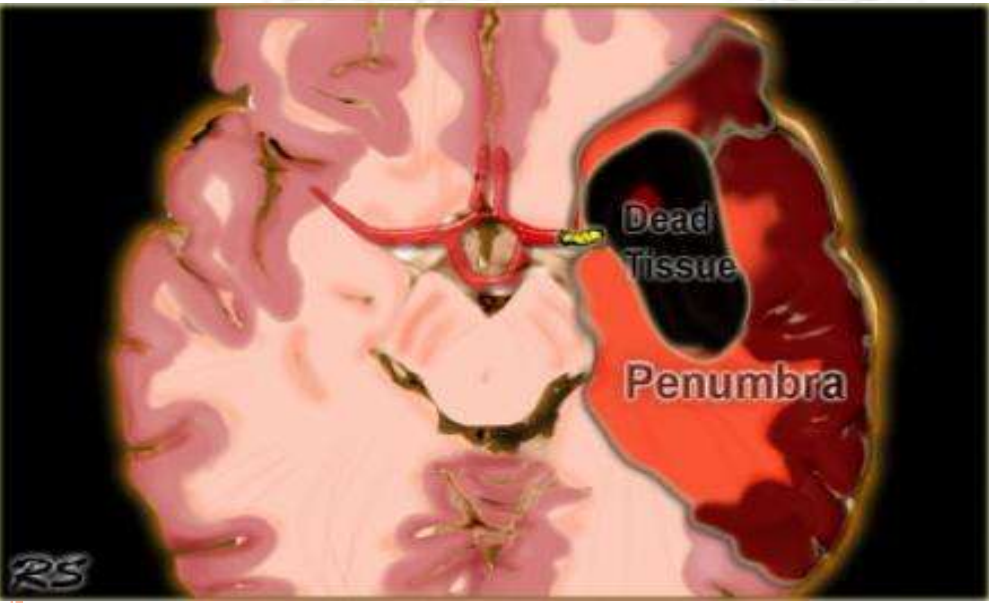
RISK FACTORS

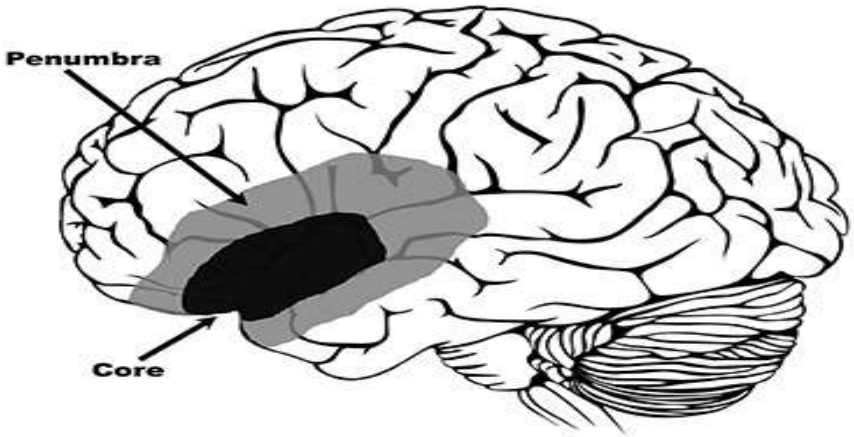
- Hypertension
- Diabetes
- Elevated total cholesterol level
- Low density lipoprotein elevated levels
- Decreased HDL
- Elevation of haematocrites
- Rheumatic heart disease
- Valvular diseases
- Endocarditis
- CABG

ISCHEMIC PENUMBRA

- Within an hour of hypoxic-ischemic insult, there is a cone of infarction surrounded by oligemic zone called Ischemic Penumbra.
- Critical time period during which the IP zone is at risk is referred to as the window of opportunity.
- Critical time period may be 2-4 hours of onset of ischemia.
- Cerebral oedema begins within a few minutes and reaches a maximum for about 4 days and mostly disappears by 3 weeks.


Normal Function
No symptoms
HEMODYNAMIC
Normal Function
No symptoms
METABOLIC
TIA
Reversible symptoms
Stroke
Irreversible symptoms
CBF (mL/100 g/min)
50
40
30
20
10
0
CBV ↑
CBV / CBF ↑
OEF ↑
GEF ↑
Normal CMRO2
CMRO2 ↓
Glutamate ↑
VSCC open
pH ↓
Transient ionic flux
PENUMBRA
Calcium avalanche
Cell death
INFARCT
Decades
Years
Hours
Minutes


Dead Tissue
Penumbra


Penumbra
Core

TYPES OF STROKE

- According to etiology
- According to management category
- According to clinical category

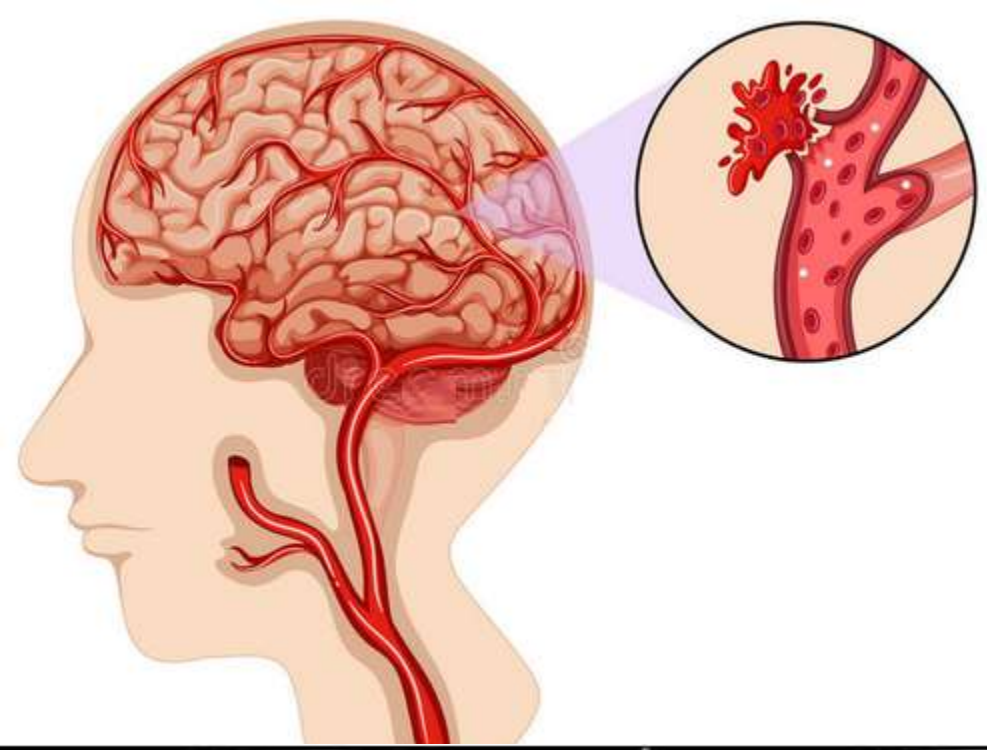
ACCORDING TO ETIOLOGY

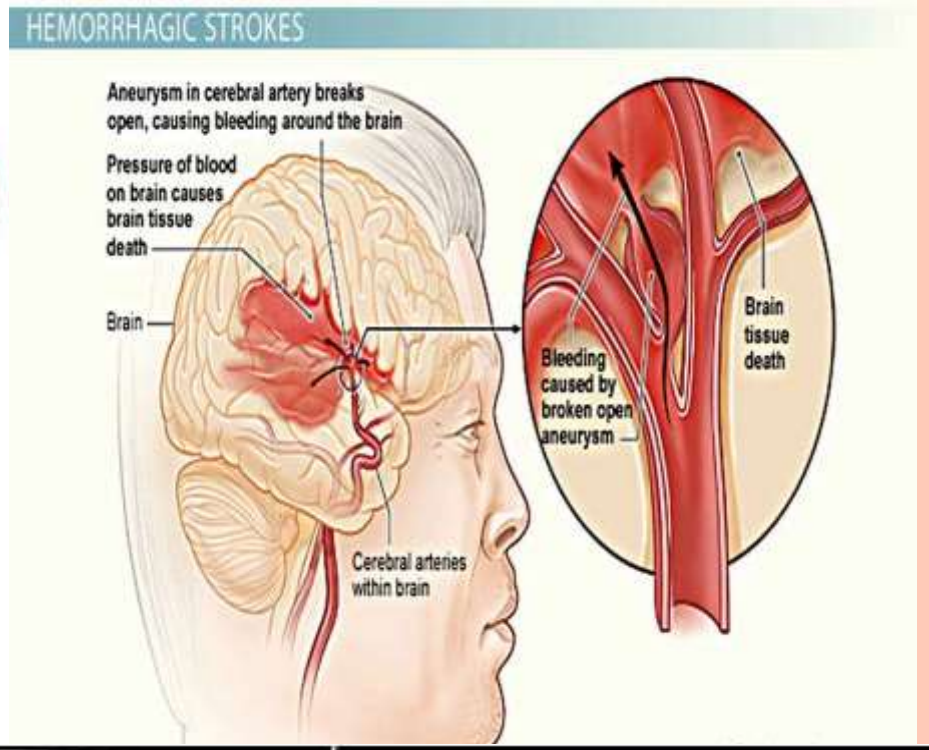
ISCHEMIC STROKE

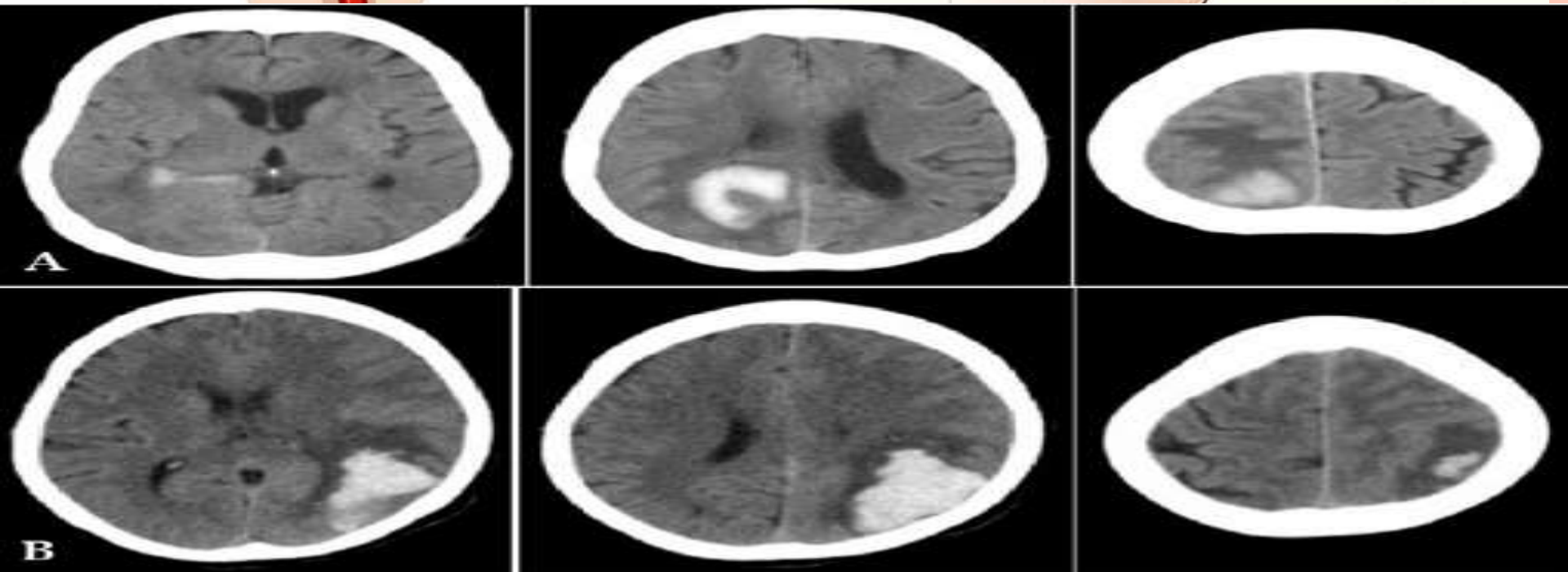
- Result of thrombus embolus occlusion, cerebral infarction, etc.
- Blood clot or plaque formation.
- Majority of emboli are showered from the heart affected by rheumatic heart disease.

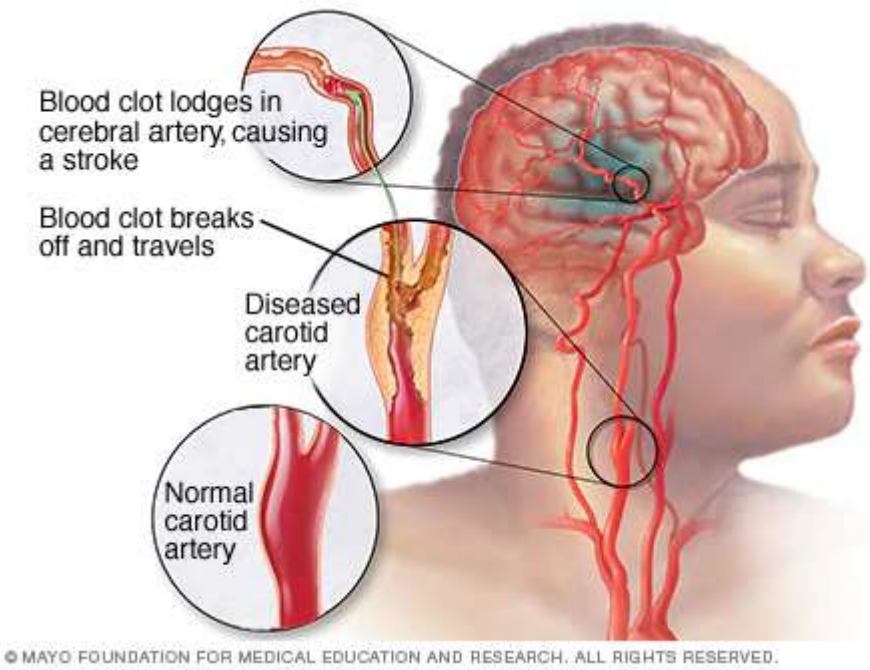
HAEMORRHAGIC STROKE

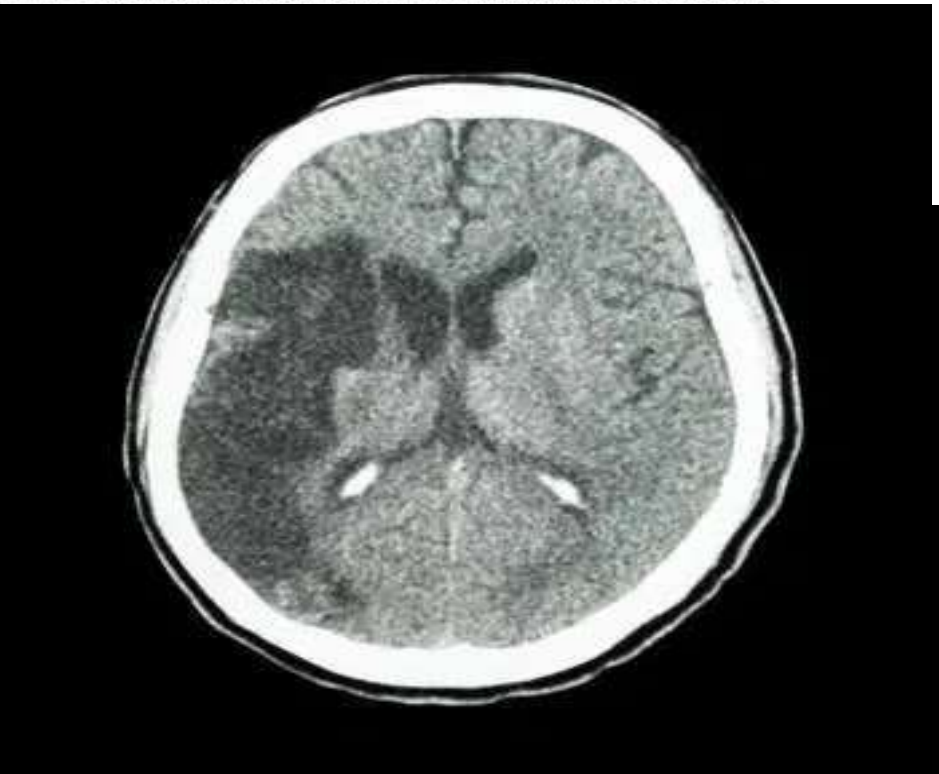
- Rupture or trauma of intra-cerebral vessels leading to abnormal bleeding.
- Bleeding into extra-vascular areas of the brain result into increased preesure, i.e. ICP.
- Can be of three types intracerebral, primary cerebral and subarachnoid haemorrhage.

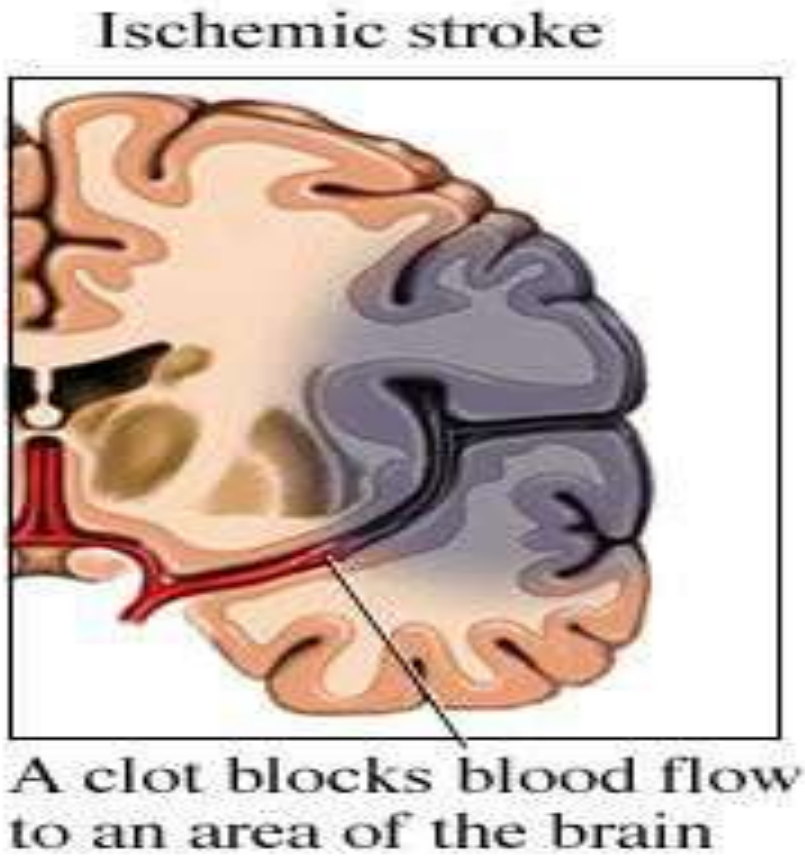


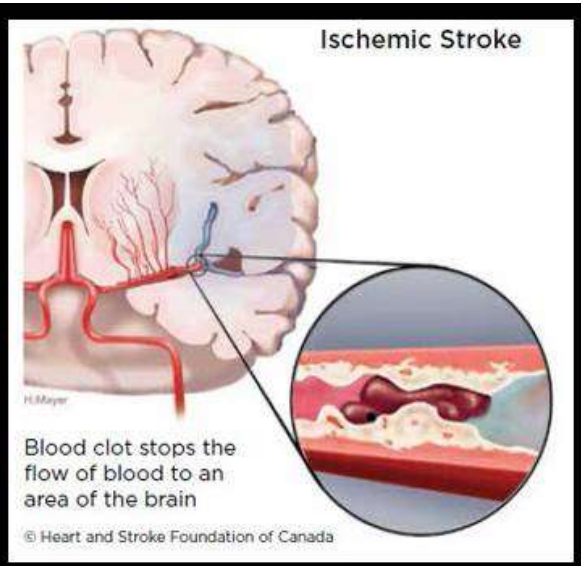

HEMORRHAGIC STROKES
Aneurysm in cerebral artery breaks open, causing bleeding around the brain
Pressure of blood on brain causes brain tissue death
Brain
Cerebral arteries within brain
Bleeding caused by broken open aneurysm
Brain tissue death


A
B









ACCORDING TO MANAGEMENT CATEGORY

TRANSIENT ISCHEMIC ATTACK

- **If the patient recovers from neurological dysfunction within 24 hours of attack, then termed as TIA.**

MINOR STROKE

- **It is the one in which there is no neurological deficit with good rehabilitation outcome.**

MAJOR STROKE

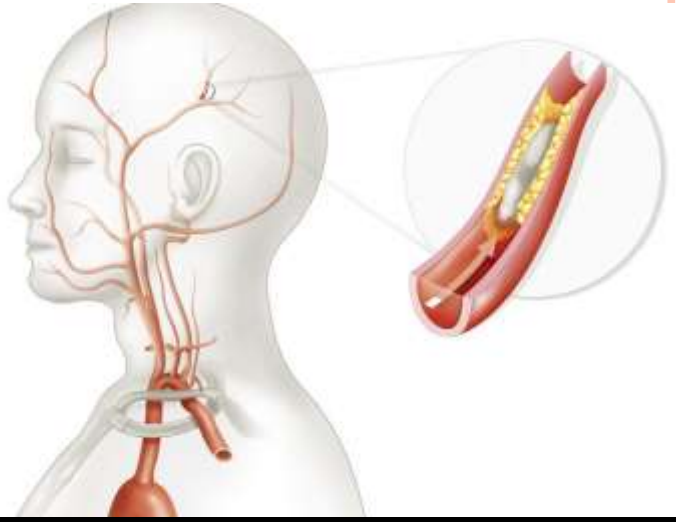
- **This is compiled of neurological deficit with bad rehabilitation outcome.**

DETERIORATING STROKE

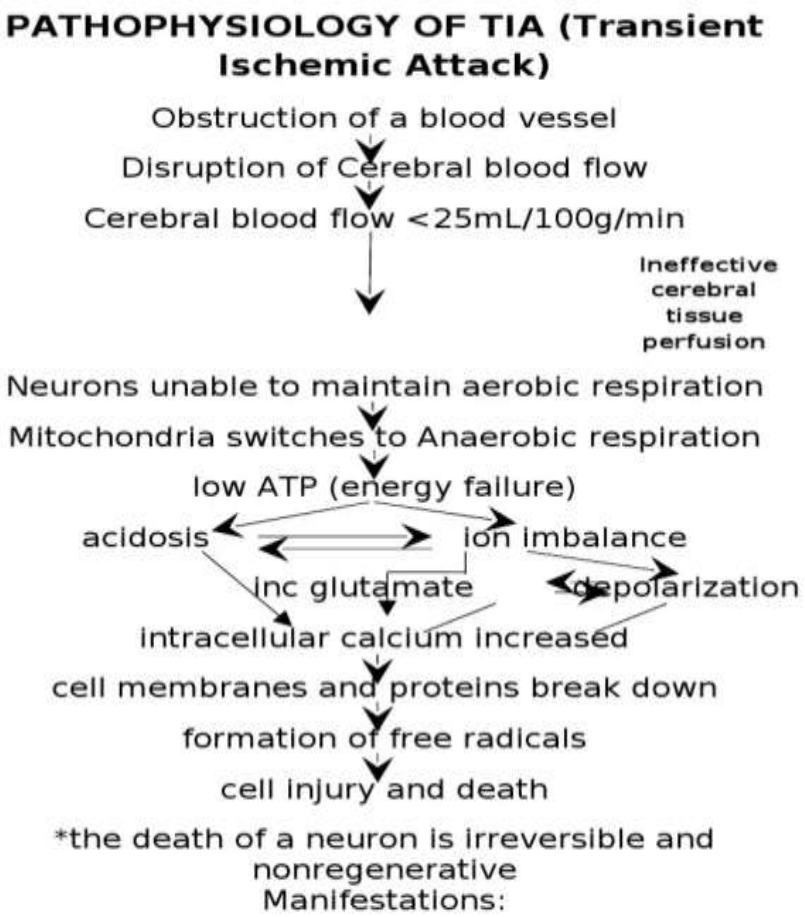
- Refers to the patient whose neurological status is deteriorating after admission into the hospital

TRANSIENT ISCHEMIC ATTACK

- Mostly lasts for only 5 to 30 minutes.
- CITS- cerebral infarction with transient symptoms and signs resolving within 24 hours despite residual brain infarction.
- Men are affected twice as frequently as women.
- Onset occurs most often in 50-70 years of age.
- About 40% of those affected are hypertensive, 50% have ischemic heart disease, 10% have diabetes mellitus.
- About 90% of TIAs occur in the carotid distribution, 7% in the vertebral-basilar distribution and 3% in both.



PATHOGENESIS OF TIA



MICRO-EMBOLISM

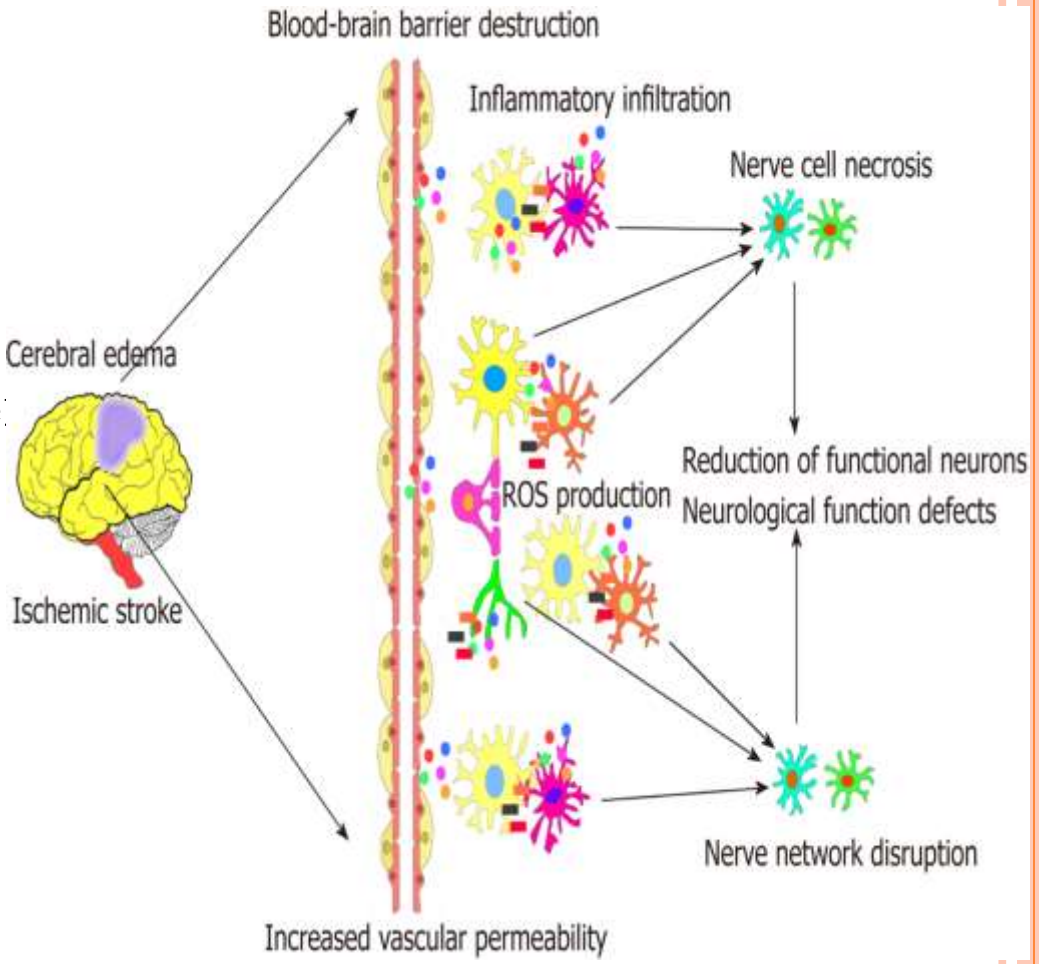
VASOSPASM

HYPOTENSION

REVERSAL OF CEPHALIC BLOOD FLOW

SYMPTOMS

- Unconsciousness
- Dizziness
- Sensory symptoms
- Tonic seizures
- Bowel or bladder incontine
- Loss of vision
- Vertigo
- Headache
- Dysarthria
- Dysphagia
- Diplopia
- Confusion



ACCORDING TO CLINICAL CATEGORY

ANTERIOR CEREBRAL ARTERY SYNDROME

MIDDLE CEREBRAL ARTERY SYNDROME

POSTERIOR CEREBRAL ARTERY SYNDROME

INTERNAL CAROTID ARTERY SYNDROME

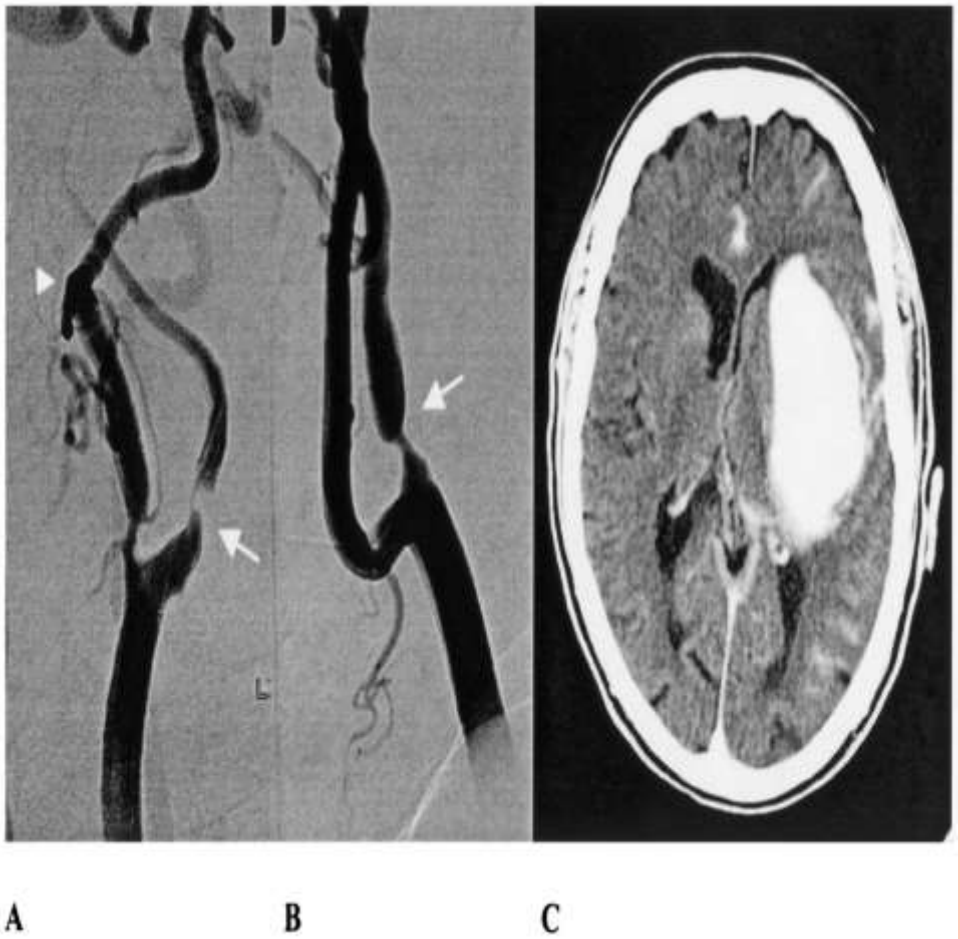
VERTEBRO-BASILAR ARTERY SYNDROME

COMMON STROKE SYNDROMES

OCCLUDED ARTERY	CLINICAL MANIFESTATIONS
1. Internal carotid artery syndrome	Transient monocular blindness, Contralateral hemiparesis.
2. Middle cerebral artery syndrome	Contralateral hemiparesis, Sensory loss of face, arm and wrist, Motor aphasia, Spatial disorientation.
3. Anterior cerebral artery syndrome	Contralateral hemiparesis, sensory loss(worsened in legs)
4. Posterior cerebral artery syndrome	Contralateral hemianopia.
5. Vertebro-basilar artery syndrome	Bilateral blindness, quadriparesis, ipsilateral loss of facial sensation, ataxia, dysphagia, hemiparesis.

I.C.A SYNDROME

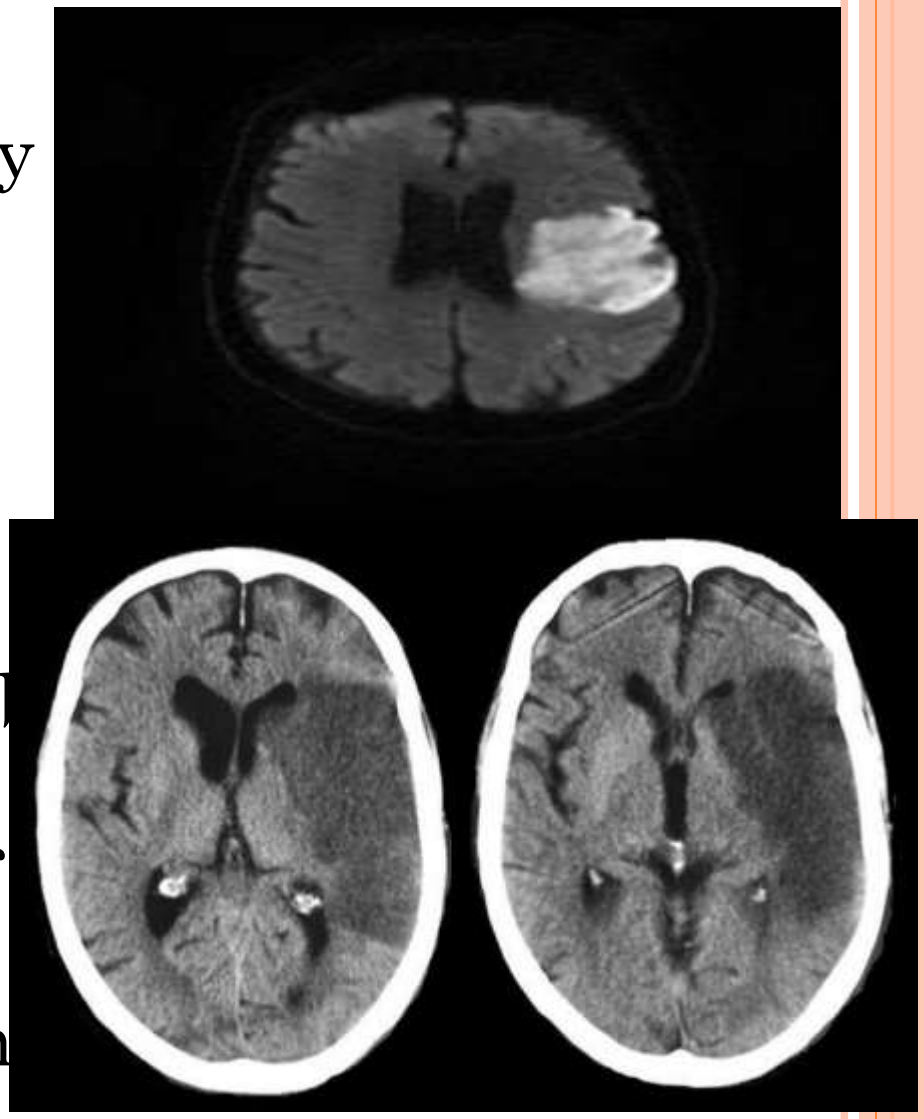
- Transient monocular blindness
- Contralateral hemiparesis.
- Significant edema
- Coma
- Death



A B C

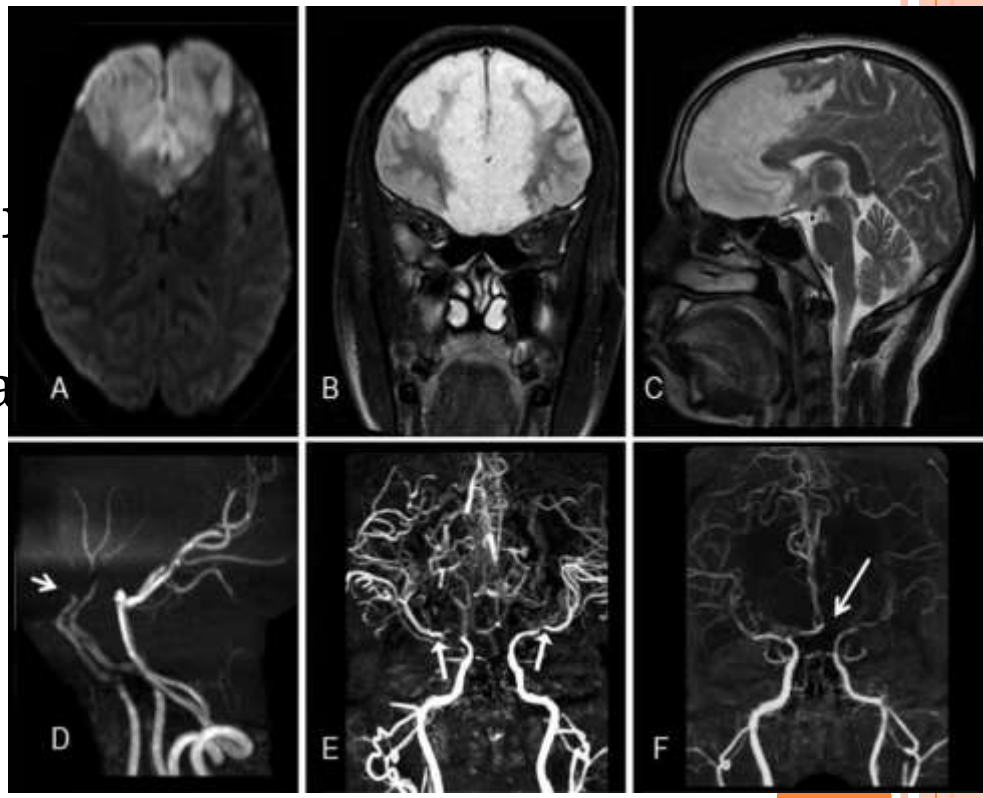
M.C.A SYNDROME

- Contralateral hemiparesis
- Contralateral hemi-sensory loss
- Motor speech impairment
- Impaired auditory comprehension
- Unilateral neglect
- Ataxia of contralateral limb
- If lesion present at upper portion of posterior limb of internal capsule then pure motor hemiplegia is evident



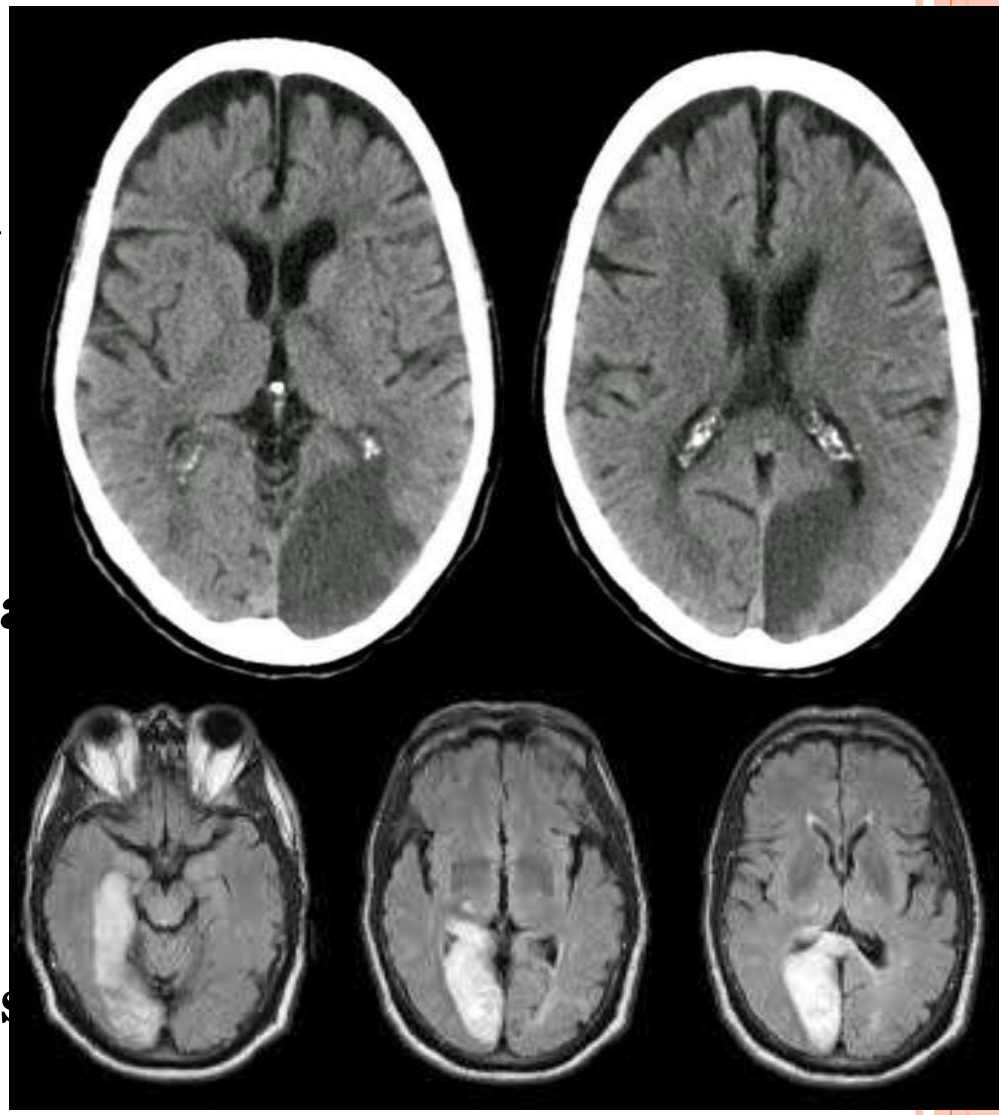
A.C.A SYNDROME

- Contralateral hemiparesis
- Lower extremities more involved than upper extremities.
- Proximal muscles are mo involved than distal.
- Problematic immitation a bimanual tasks
- Apraxia
- Slowness
- Lack of spontanity
- Urinary incontinence



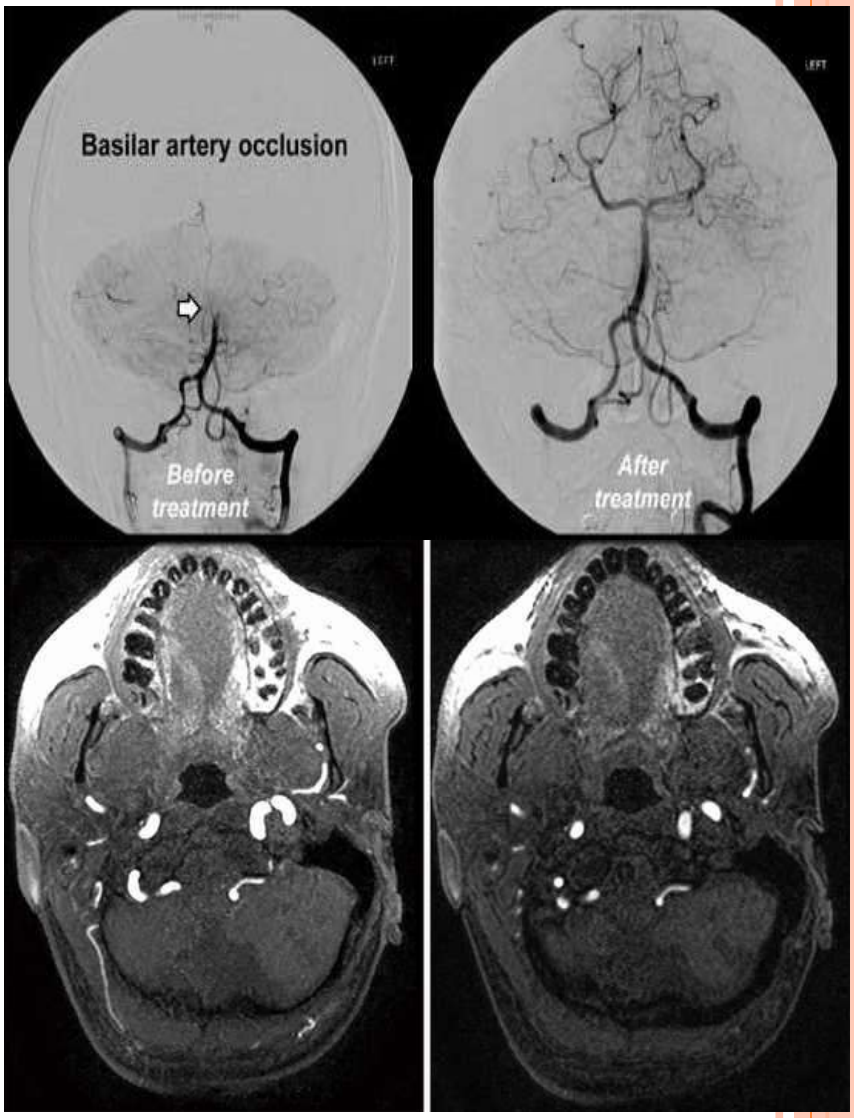
P.C.A SYNDROME

- Memory defect
- Central post stroke pain
- Involuntary movements
- Intention tremors
- Contralateral hemiplegia
- Occular nerve palsy
- Visual agnosia
- Contralateral hemianops



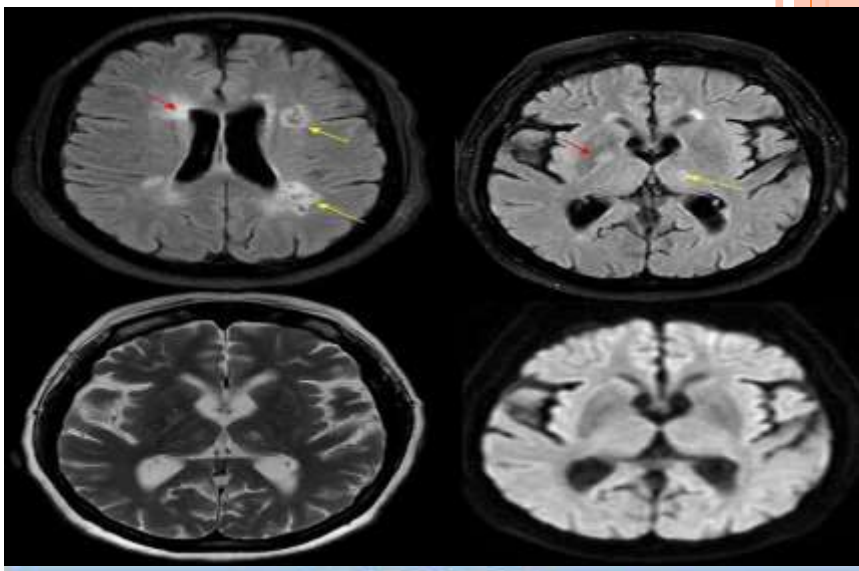
VERTEBRO-BASILAR ARTERY SYNDROME

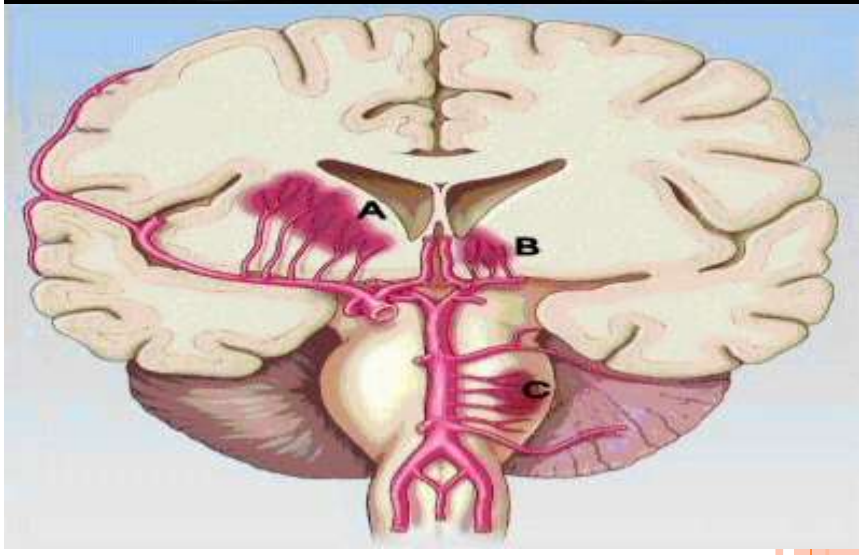
- Complete occlusion can be fatal due to supply to the pons, inner ear and cerebellum.
- Occipital headache
- Diplopia
- Hemiplegia
- Quadriplegia
- Coma
- Locked In syndrome- anarthria, quadriplegia, preserved consciousness, alertness and vertical eye movement.
- Loss of pain and temperature sensation



LACUNAR SYNDROMES

- Caused by small vessel disease deep in cerebral white matter.
- Pure lacunar syndrome is due to involvement of ventrolateral thalamus.
- Dysarthria/ clumsy hand syndrome due to involvement of base of pons, internal capsule.
- Ataxic hemiparesis due to involvement of pons, genu, corona radiata or cerebellum.
- Sensory / motor involvement due to lession at the junction of internal capsule and thalamus.
- Dystonia/ involuntary movements due to infarction of the putamen or globus pallidus.





PRIMARY IMPAIRMENTS

- Altered consciousness.
- Motor deficits.
- Sensory deficits
- Presence of abnormal reflexes.
- Cognitive dysfunctions.
- Presence of language disorders.
- Perceptual problems.
- Seizures
- Bladder and bowel dysfunction

ALTERED CONSCIOUSNESS

- Coma or decreased arousal level may occur in extensive brain damage as in large MCA occlusion.
- Glasgow coma scale.
- Levels of consciousness:

a) Normal
b) Lethargy
c) Obtunded
d) Stupor
e) Coma

Feature	Response	Score
Best eye response	Open spontaneously	4
	Open to verbal command	3
	Open to pain	2
	No eye opening	1
Best verbal response	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No verbal response	1
Best motor response	Obeys commands	6
	Localising pain	5
	Withdrawal from pain	4
	Flexion to pain	3
	Extension to pain	2
	No motor response	1

Descriptor	Characteristics
Awake	Attends to the environment; responds appropriately to verbal instruction or gesture
Lethargic	Oriented when awake but will sleep if left alone; requires verbal or gentle touch stimulation to initiate a response
Obtundation	Requires repeated moderate stimulation to maintain attention; responds slowly and goes back to sleep
Stupor	Can be aroused by vigorous stimuli; decreased reaction to external stimuli
Coma	Cannot be aroused, no response to external stimuli, no voluntary movements

MOTOR DEFICITS

- Symptoms seen are flaccidity followed by spasticity and then stage of spontaneous recovery.
- Immediately after disruption o blood flow there is stage of flaccidity and areflexia.
- Replaced by development of spasticity, hyperreflexia and synergies.
- Symptoms seen in opposite side of lession.
- Variability depends upon the location and severity of the lesion and potential for adaptation.

Brunnstrom's Stages of Stroke Recovery	
Stage 1: Flaccidity	Arm is flaccid; complete lack of voluntary movement
Stage 2: Spasticity Appears	Arm begins to make small, jerky movements
Stage 3: Increased Spasticity	Arm becomes spastic; muscles feel stiff and tight
Stage 4: Decreased Spasticity	Arm spasticity decreased; arms begins to move easier
Stage 5: Complex Movement Combinations	Coordinated, typical arm movement begins to return
Stage 6: Spasticity Disappears	Spasticity disappears and movements are voluntary
Stage 7: Normal Function Return s	Voluntary and controlled arm function returns

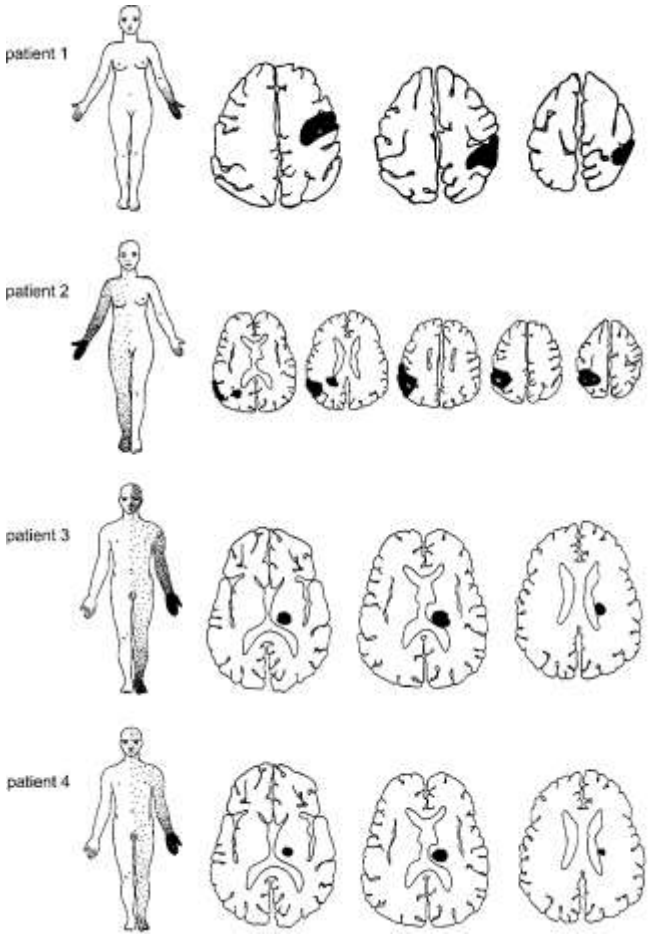
STAGE	CHARACTERISTICS
Stage 1	•Period of flaccidity •Neither reflex nor voluntary movements are present
Stage 2	•Basic limb synergies may appear as associated reactions •Spasticity begins mostly evident in strong components (flexor synergy appear prior to extensor synergy) •Minimal voluntary movement responses may be present
Stage 3	•Patient starts to gain voluntary control over movement synergies •Spasticity reaches its peak •Semi-voluntary stage as individual is able to initiate movement but unable to control it

	Flexion Synergy Components	Extensor Synergy Components
Upper Extremity	Scapular retraction and elevation Shoulder abduction, ext rot Elbow flexion Forearm supination Wrist/finger flexion	Scapular protraction Shoulder adduction, internal rotation Elbow extension Forearm pronation Wrist/finger flexion
Lower Extremity	Hip flexion, abduction, external rotation Knee flexion Ankle DF, inversion Toe DF	Hip extension, adduction, internal rotation Knee extension Ankle PF, inversion Toe PF

O'Sullivan & Schmitz. *Physical Rehabilitation*. 2007. Chapter 18.

SENSORY DEFICITS

- Depend upon the site and extent of lession.
- Lession involving thalamus leads to contralateral hemianesthesia.
- Lession involving the somatosensory cortex then cortical sensations are impaired like tactile localization, 2-point discrimination, stereognosis and tactile extinction.
- Visual disturbances seen in the involvement of visual cortex.
- Internal capsule or optic radiation involvement gives rise to hemianopia.
- Involved upper part of pons results in crossed anesthesia.

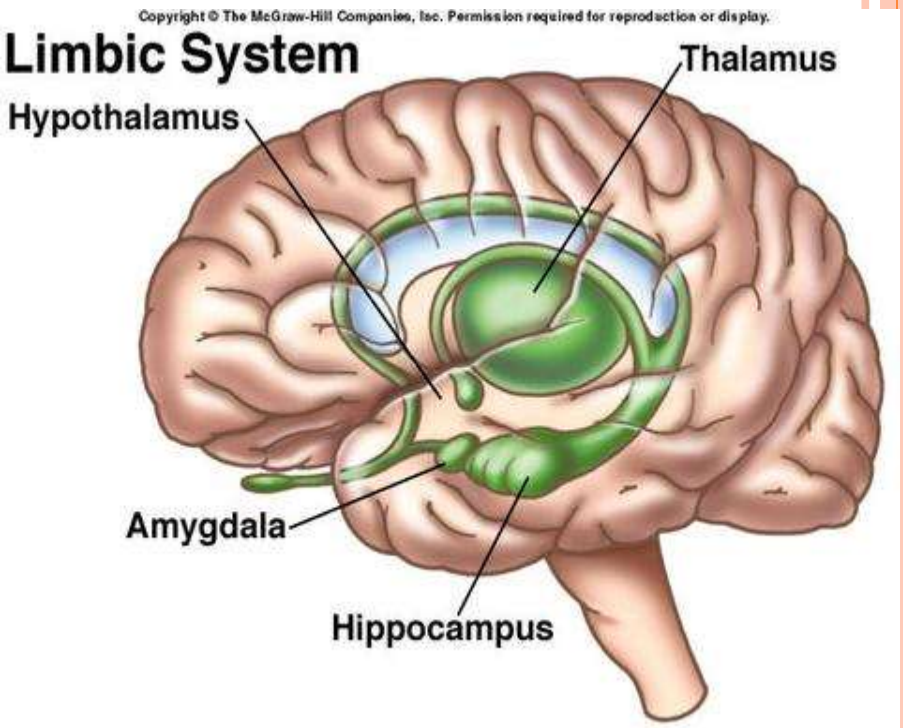


ABNORMAL REFLEXES

- Initially all the deep tendon reflexes absent or suppressed.
- Exaggerated deep tendon reflexes on development of spasticity.
- Babinski positive with extensor response.
- Superficial reflexes diminished or absent.

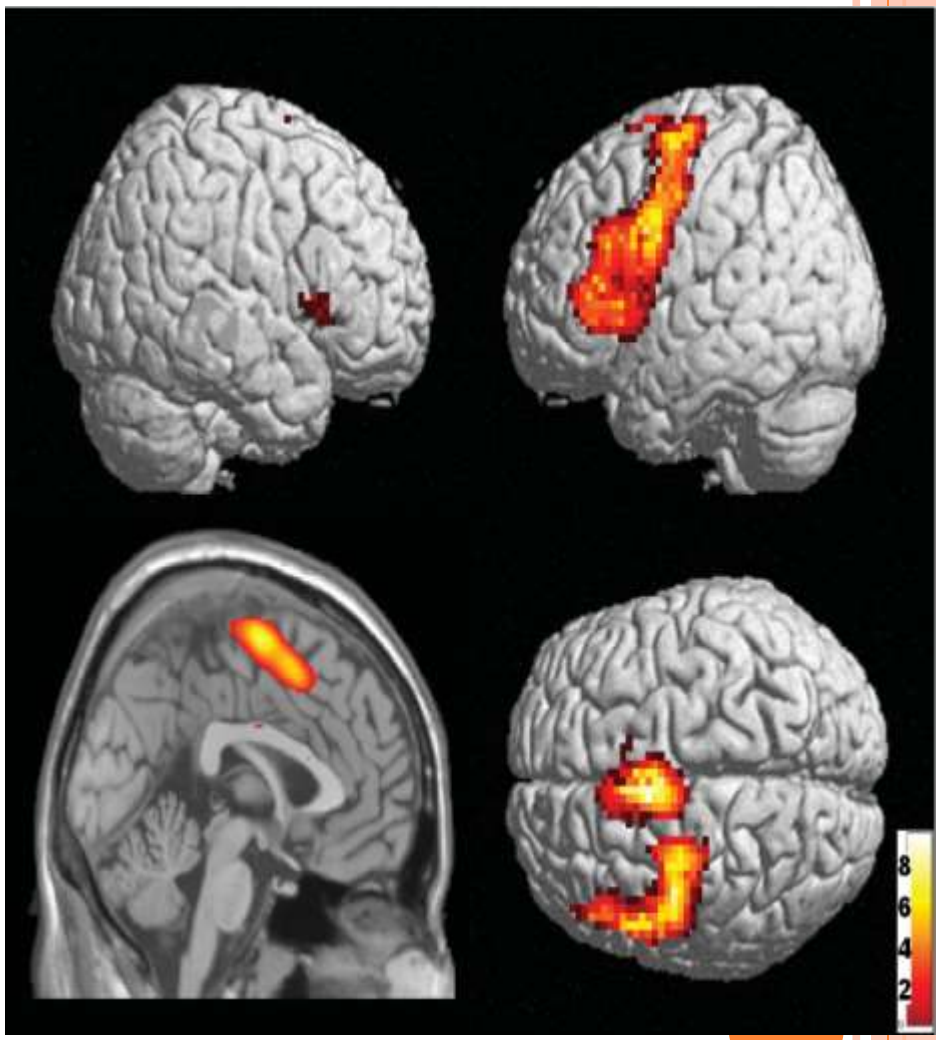
COGNITIVE DYSFUNCTION

- Present with lessions of the cortex.
- Impairments in alertness, attention, orientation, memor and executive functions.
- Confabulations result due to lession of the prefrontal corte
- Short term memory loss is associated with limbic system and limbic association cortex.
- Long term memory impairments is associated with the hippocampus of the limbic



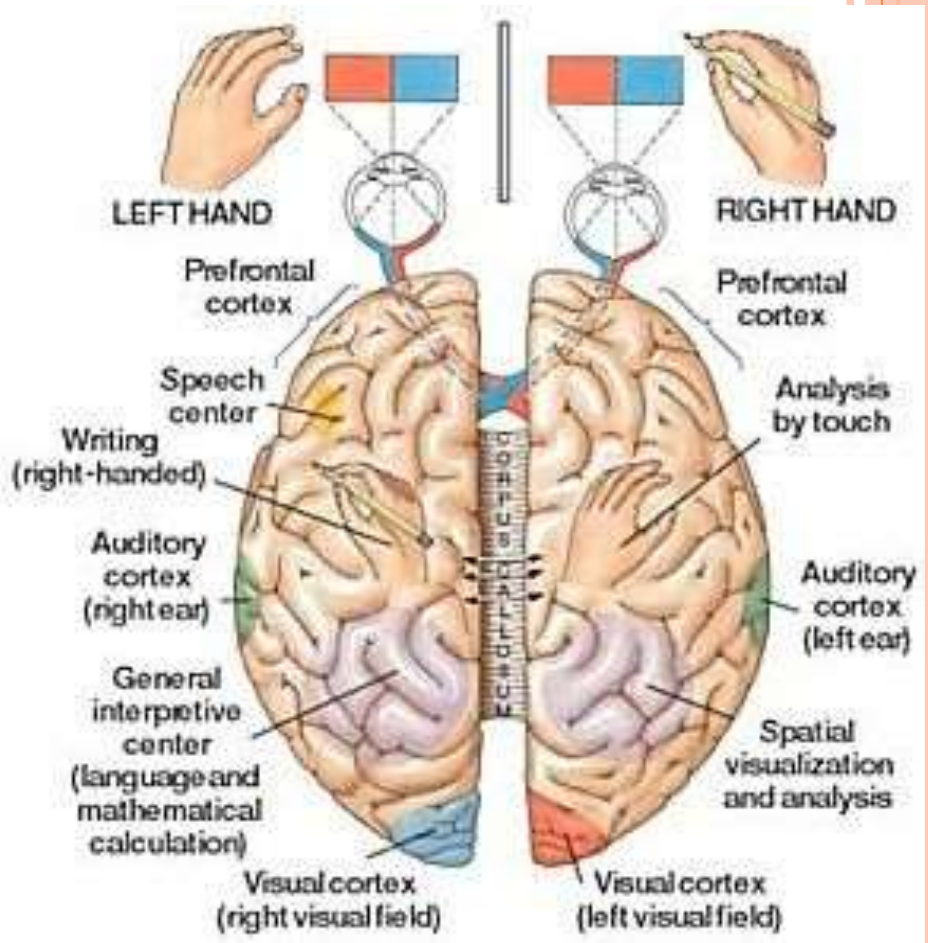
SPEECH AND LANGUAGE

- Due to involvement of cortex of dominant hemisphere.
- Patients present with aphasia and dysarthria.
- Respiration, articulation of voice box, phonation, resonance and/or sensory feedback may be affected.
- Lesion can be located in the primary motor cortex in frontal lobe, primary sensory cortex in parietal lobe or the cerebellum.



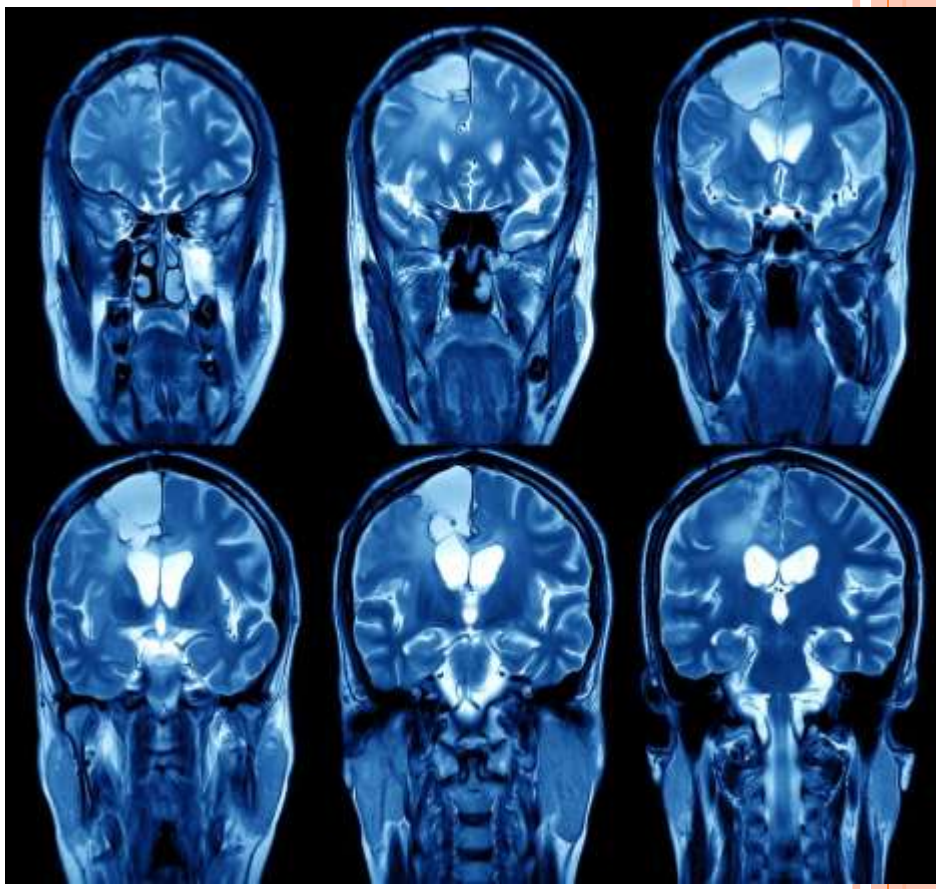
PERCEPTUAL DISORDERS

- Presence of apraxia, due to lesion on the dominant parietal lobe.
- Presence of agnosia due to disorders in the association fibers or association pathways disorders.
- Visuo-spatial relation disorder due to lesion of the non-dominant parietal lobe.



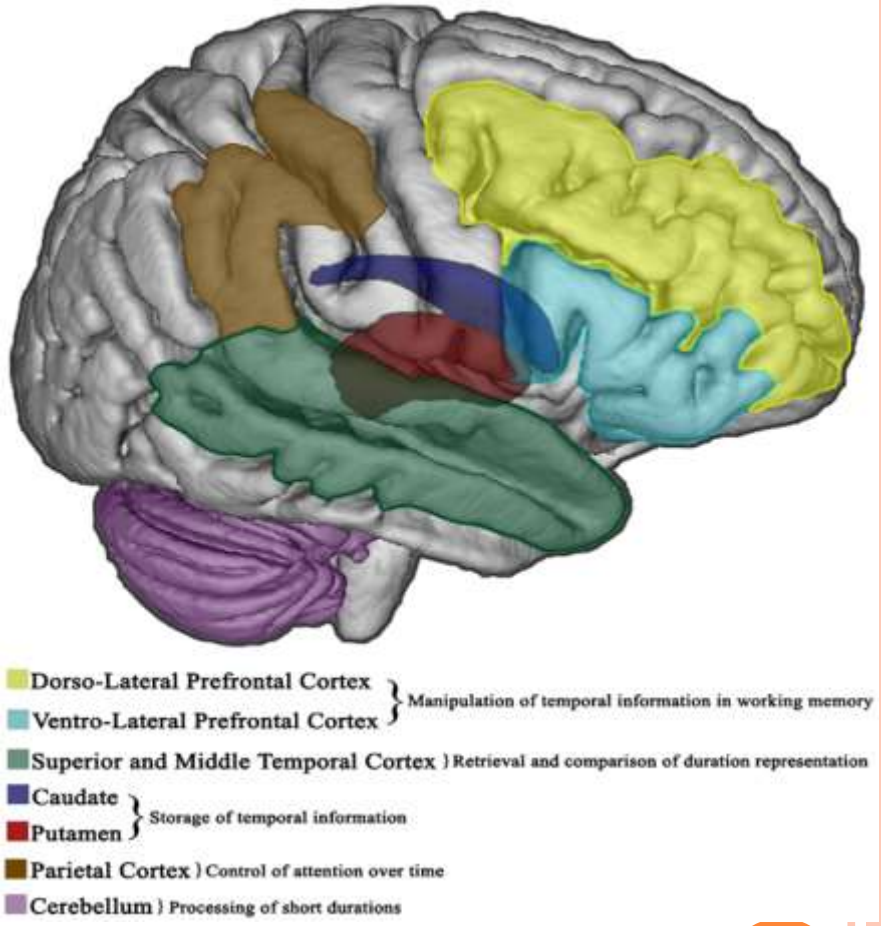
SEIZURES

- Occur in small percentage.
- More common in occlusive carotid disease.
- Less commonly in the MCA stroke.
- Potentially life threatening if not controlled.



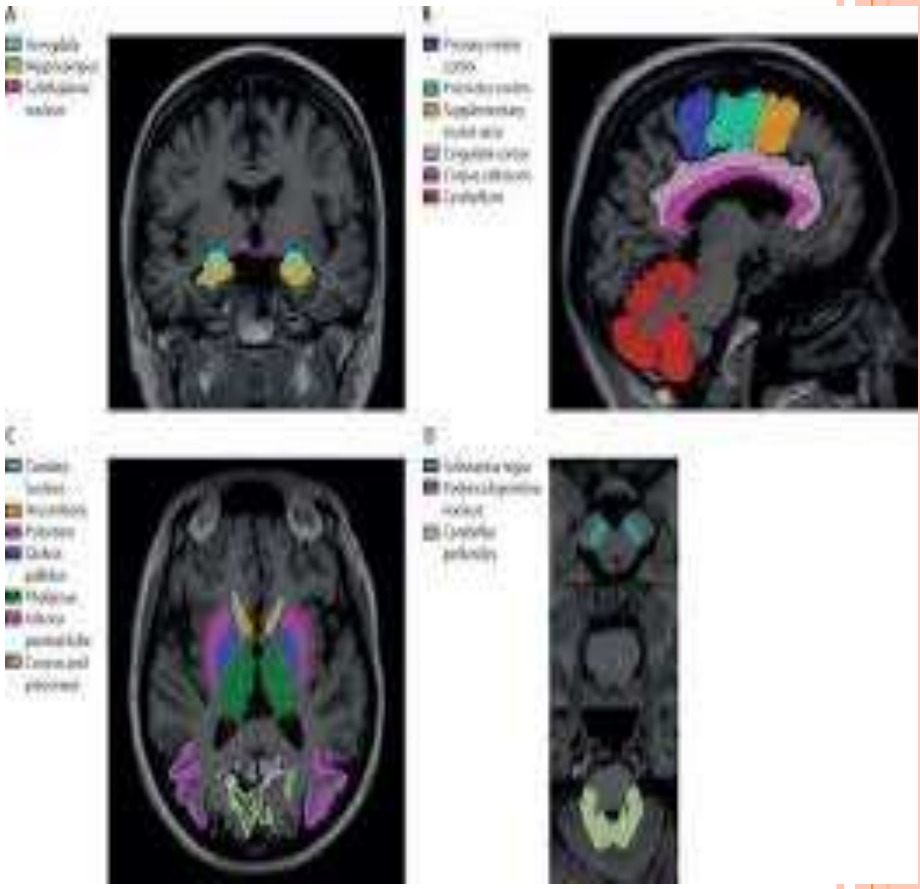
BLADDER AND BOWEL

- Urinary incontinence can result from the bladder hyperreflexia or hyporeflexia disturbances of sphincter control, and/or sensory loss.
- Anterior communicating artery blockage results in ischemia of paracentral lobule resulting in loss of voluntary control of micturition.



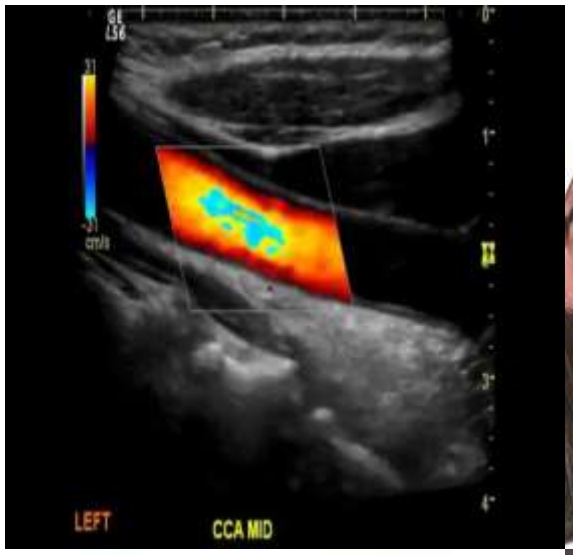
GAIT IMPAIRMENTS

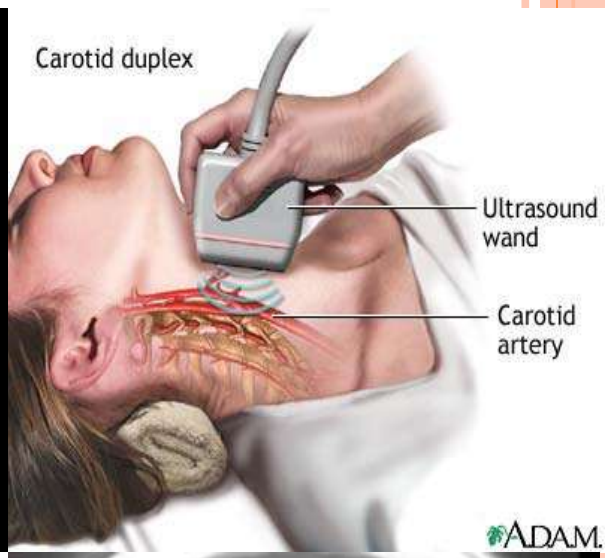
- Mostly upper limb goes into flexion and lower limb in extension synergy.
- Circumductory or swing gait.
- Missing of heel strike component.
- In flaccid stge stage there is buckling of knee on weight bearing.
- Slowness of walking due to quadriceps and planter flexors spasticity.
- Trendelenburg sign positive.

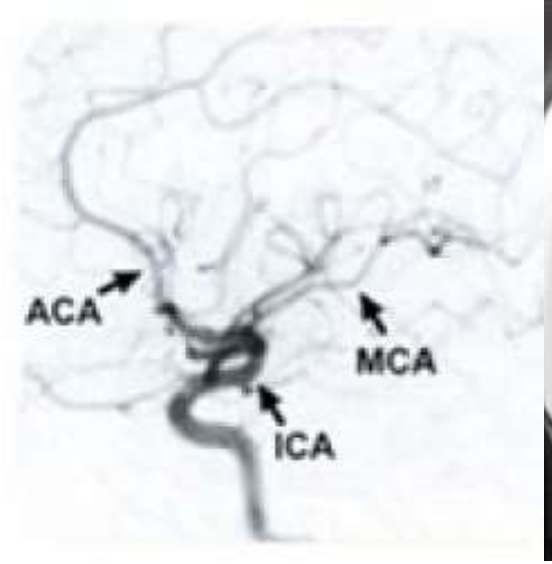


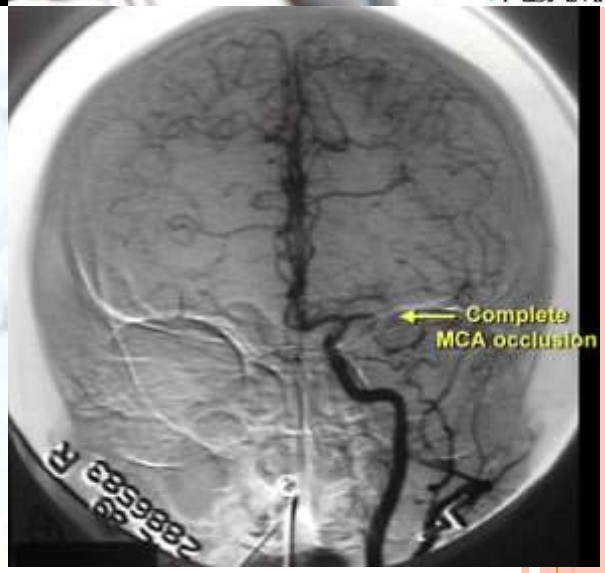
INVESTIGATIONS

- Carotid duplex
- CT Scan
- Electrocardiogram
- MRI Scan
- Cerebral arteriogram









THANK YOU