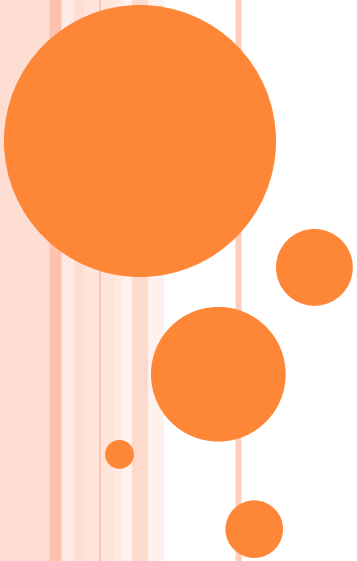


# CNS

## REVISION LECTURES

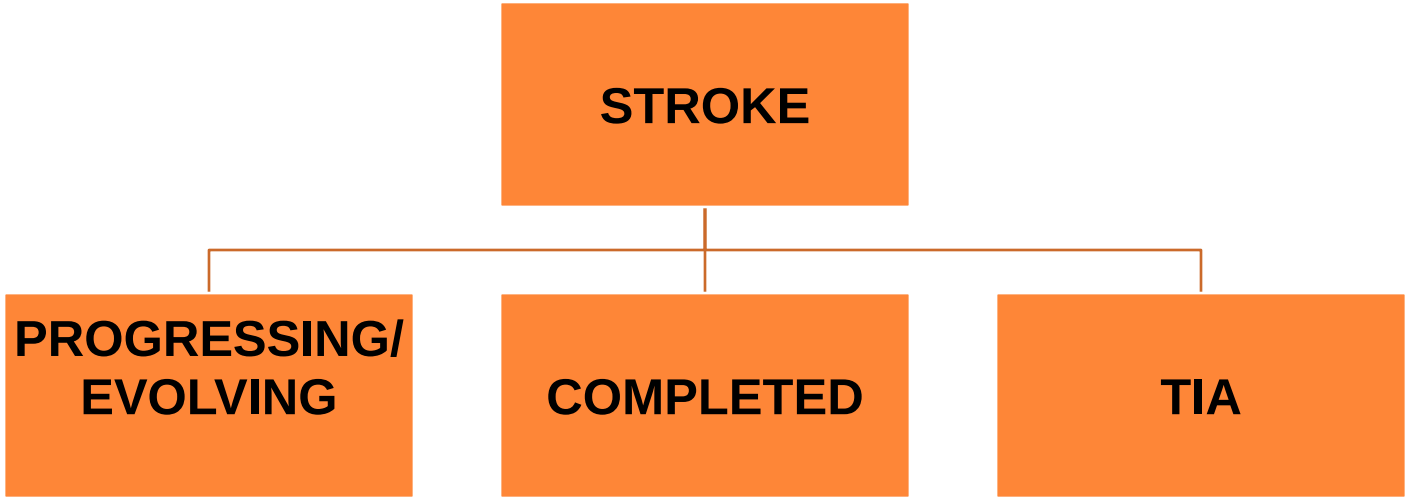
# STROKE

DR. SURABHI NAGARE

# INTRODUCTION

- Stroke is defined as
  - ➢ Abrupt onset of neurological deficit
  - ➢ Persists more than 24 hour.
  - ➢ With no apparent case other than that of vascular origin

# CLINICAL CLASSIFICATION



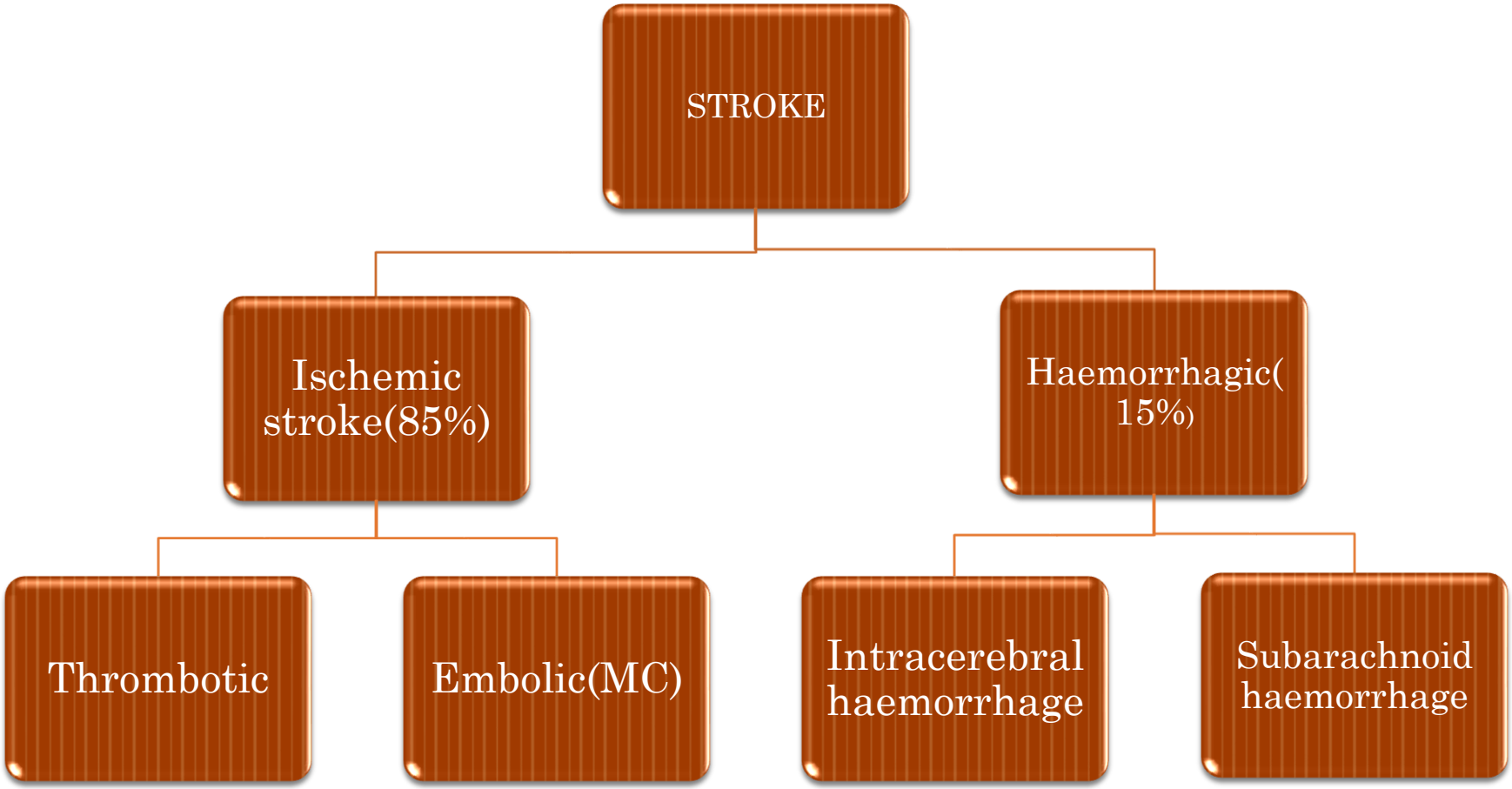
- TIA(Transient ischemic attack):A clinical syndrome of rapid onset of focal deficits of brain function, which resolves with in 24 hours,regardless of whether there's imaging evidence of new permanent brain injury.
- PROGRESSIVE STROKE:A stroke in which focal neurological deficits worsen with time.
  - Also called stroke in evolution.
- COMPLETED STROKE:A stroke in which the focal neurological deficits persists & donot worsen with time.

# Types & risk factors

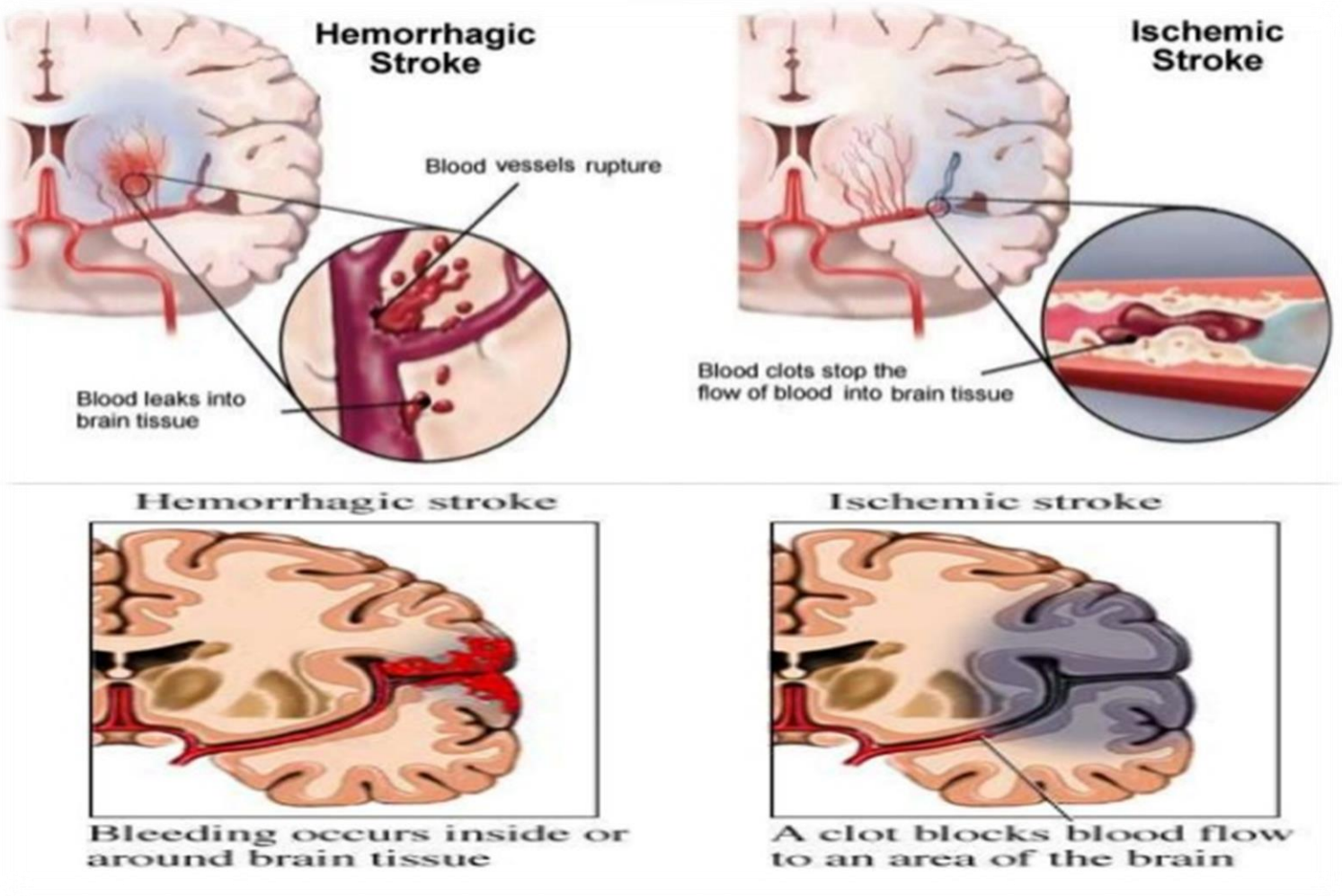
## Risk factors

- Fixed
  - Age
  - Gender( male> female)
  - Race(Asian>european)
  - Heredity
  - Previous Vascular event.eg: MI, peripheral embolism
  - High fibrinogen
- Modifiable
  - High blood pressure
  - Heart disease(atrial fibrillation, heart failure, endocarditis)
  - Diabetes mellitus
  - Hyperlipidaemia
  - Smoking
  - Excess alcohol consumption
  - Oral contraceptives
  - Social deprivation
  - Obesity, sedentary lifestyle

# Types of stroke



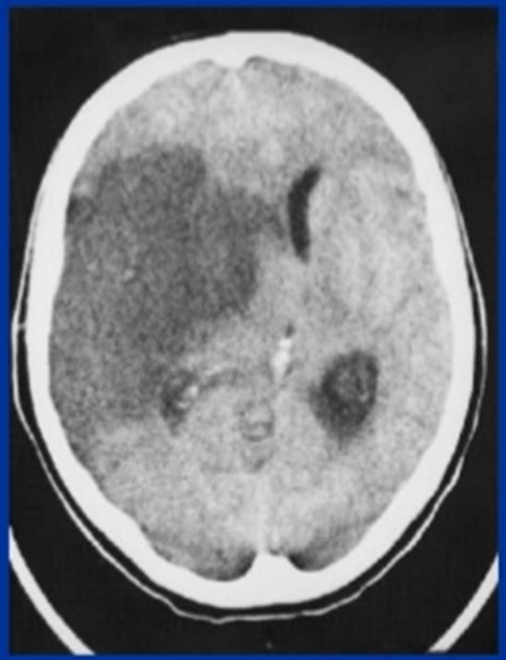
# Types of stroke

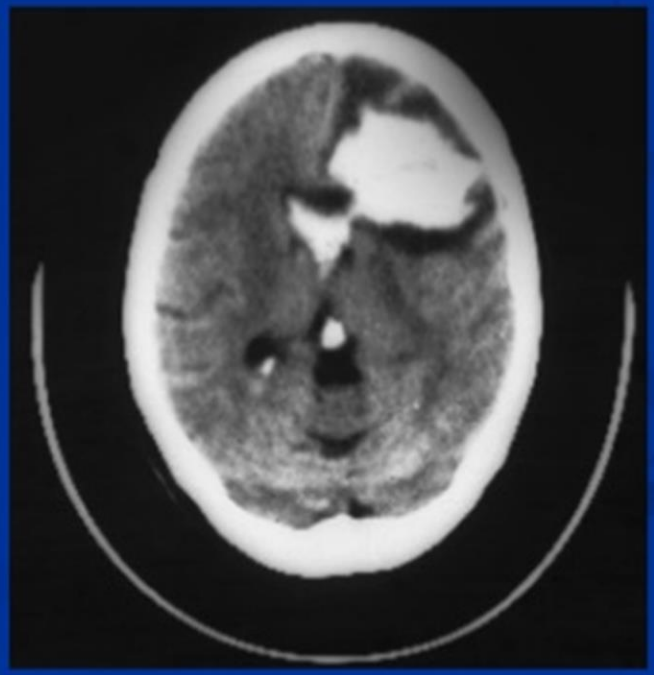


# Types of Stroke

85%
Ischemic

15 %
hemorrhagic



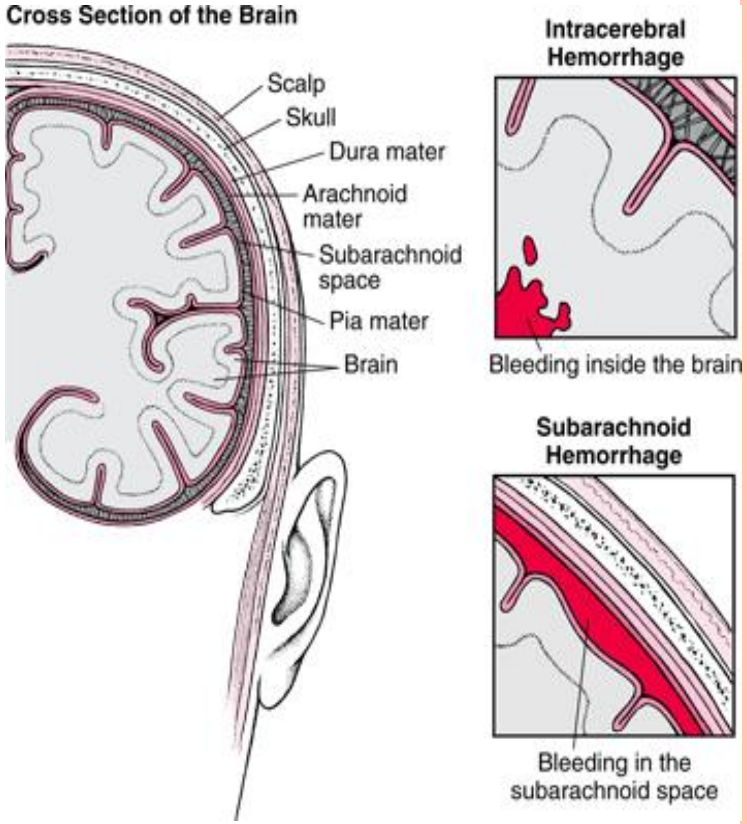


# Pathophysiology of stroke

- Brain requires constant supply of glucose & oxygen, delivered by blood.
- Brain receives 15% of resting output & accounts for 20% of total body oxygen consumption.
- Cerebral blood flow is maintained via auto regulation. Thus the brain is highly aerobic tissue where oxygen is limiting factor.
- Blood flow
  - If zero leads to death of brain tissue within 4-10 mins
  - <16-18ml/100g tissue/min ⟶ infarction with in an hour.
  - <20ml/100gm tissue/min ⟶ ischemia without infarction unless prolonged for several hours or day.

# Hemorrhagic Stroke

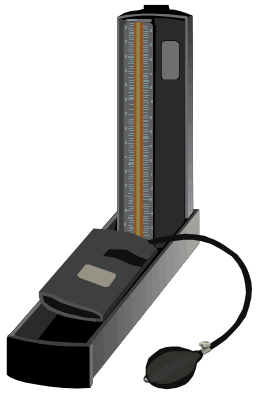
- Two types
  - Intracerebral hemorrhage(ICH)
  - Subarachnoid hemorrhage(SAH)
- Higher mortality rates when compared to ischemic stroke



# Pathophysiology Of Hemorrhagic Stroke

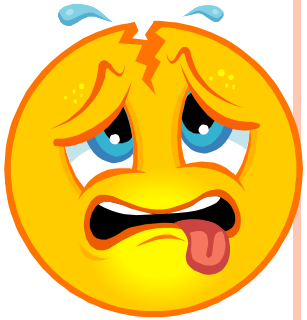
- Explosive entry of blood into the brain parenchyma structurally disrupts neurons.
- White matter fibre tracts are split.
- Immediate cessation of neuronal function.
- Expanding hemorrhage can act as a mass lesion and cause further progression of neurological deficits.
- Large hemorrhages can cause transtentorial coning and rapid death.

# Intracerebral Hemorrhage



- Result of chronic hypertension
- Small arteries are damaged due to hypertension
- In advanced stages vessel wall is disrupted and leads to leakage

## Subarachnoid Hemorrhage



- Most common cause is rupture of saccular or Berry aneurysms
- Other causes include arteriovenous malformations, angiomas, mycotic aneurysmal rupture etc.
- Associated with extremely severe headache

# Etiology of ischemic stroke

## Thrombotic

- Lacunar stroke
- Large vessel thrombosis
- Hypercoagulable disorders

## Embolic

- Artery to artery
  - Carotid bifurcation
  - Aortic arch
- Cardioembolic
  - Atrial fibrillation
  - Myocardial infarction
  - Mural thrombus
  - Bacterial endocarditic
  - Mitral stenosis
  - Paradoxical embolus

# Thrombotic Stroke

- **Atherosclerosis** is the most common pathology leading to thrombotic occlusion of blood vessels
- **Lacunar stroke**
- Accounts for 20% of all strokes
- Results from occlusion of small deep penetrating arteries of the brain
- Thrombosis leads to small infarcts known as lacunes
- Clinically manifested as lacunar syndromes

# Embolic Stroke

- **Cardioembolic stroke**
  - Embolus from the heart gets lodged in intracranial vessels
  - MCA most commonly affected
  - Atrial fibrillation is the most common cause
  - Others: MI, prosthetic valves, rheumatic heart disease
- **Artery to artery embolism**
  - Thrombus formed on atherosclerotic plaques gets embolized to intracranial vessels
  - Carotid bifurcation atherosclerosis is the most common source
  - Others: aortic arch, vertebral arteries etc.

# Ischemic penumbra

- Tissue surrounding the core region of infraction which is ischemic but reversibly dysfunctional.
- Maintained by collaterals.
- Can be salvaged if reperfused in time
- Primary goal of revascularization therapies.

# Signs & symptoms of stroke

# History

- Ask for onset and progression of neurological symptoms – completed stroke or stroke in evolution
- History of previous TIAs
- History of hypertension & diabetes mellitus
- History of heart conditions like arrhythmias, RHD & prosthetic valves
- History of seizures & migraine
- History of anticoagulant therapy
- History of oral contraceptive use
- History of any hypercoagulable disorders like sickle cell anemia & polycythemia vera
- Substance abuse: cocaine, amphetamines

# Examination of a stroke patient

- The neurological examination is highly variable and depends on the location of the vascular lesion.
- Skin: look for xanthelasma,rashes,limb ischemia
- Eyes:look for diabetic changes,retinal emboli,hypertensive changes,arcus senilis
- CVS: hyper/hypotension, abnormal rhythm,murmursraised JVP, peripheral pulses and bruits Respiratory system: pulmonary edema, infection
- Abdomen: urinary retention
- Locomotor system: injuries sustained during collapse with stroke, comorbities which influence functional abilities.

# STROKE WARNING SIGNS

American Heart Association | American Stroke Association

SUDDEN NUMBNESS OR WEAKNESS OF THE FACE, ARM OR LEG, ESPECIALLY ON ONE SIDE OF THE BODY

SUDDEN CONFUSION, TROUBLE SPEAKING OR UNDERSTANDING

SUDDEN TROUBLE SEEING IN ONE OR BOTH EYES

SUDDEN TROUBLE WALKING, DIZZINESS, LOSS OF BALANCE OR COORDINATION

SUDDEN SEVERE HEADACHE WITH NO KNOWN CAUSE

**Immediately call 9-1-1** or the emergency medical services (EMS) number so an ambulance (ideally with advanced life support) can be sent for you.

**Also, check the time** so you'll know when the first symptoms appeared. It's very important to take immediate action. If given within 3 hours of the start of symptoms, a clot-busting drug called tissue plasminogen activator (tPA) may reduce long-term disability for the most common type of stroke.



www.strokeassociation.org

# Left and Right Hemisphere Stroke: Common Patterns

## Left (Dominant) Hemisphere Stroke: Common Pattern

- Aphasia
- Right hemiparesis
- Right-sided sensory loss
- Right visual field defect
- Poor right conjugate gaze
- Dysarthria
- Difficulty reading, writing, or calculating

## Right (Non-dominant) Hemisphere Stroke: Common Pattern

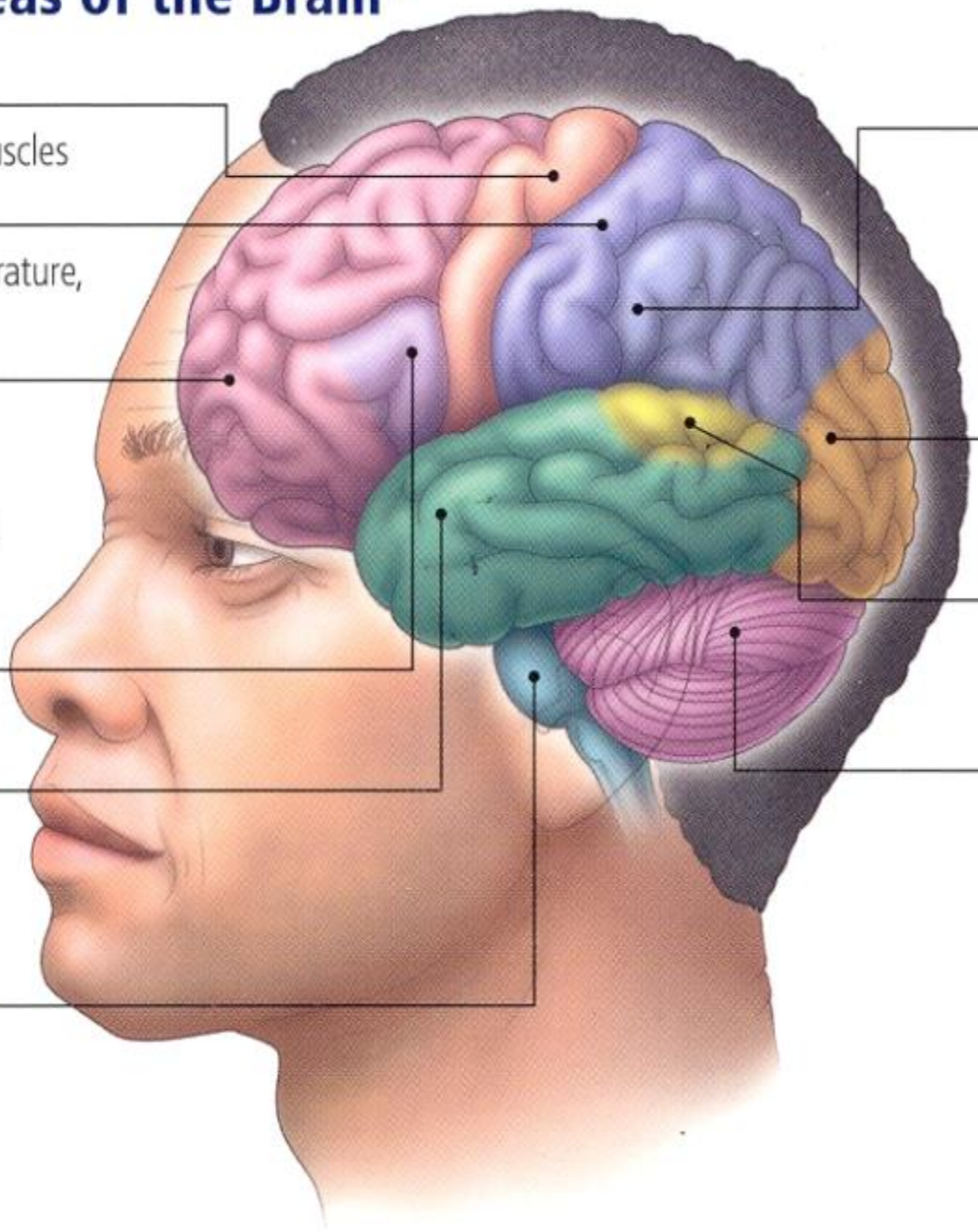
- Neglect of left visual field
- Extinction of left-sided stimuli
- Left hemiparesis
- Left-sided sensory loss
- Left visual field defect
- Poor left conjugate gaze
- Dysarthria
- Spatial disorientation

# Clinical localization of stroke syndromes

Prerequisites

- Functional anatomy of brain.
- Blood supply to the different parts of brain.

# Functional Areas of the Brain[1]



**Motor Area**
- control of voluntary muscles

**Sensory Area**
- skin sensations (temperature, pressure, pain)

**Frontal Lobe**
- movement
- problem solving
- concentrating, thinking
- behaviour, personality, mood

**Broca's Area**
- speech control

**Temporal Lobe**
- hearing
- language
- memory

**Brain Stem**
- consciousness
- breathing
- heart rate

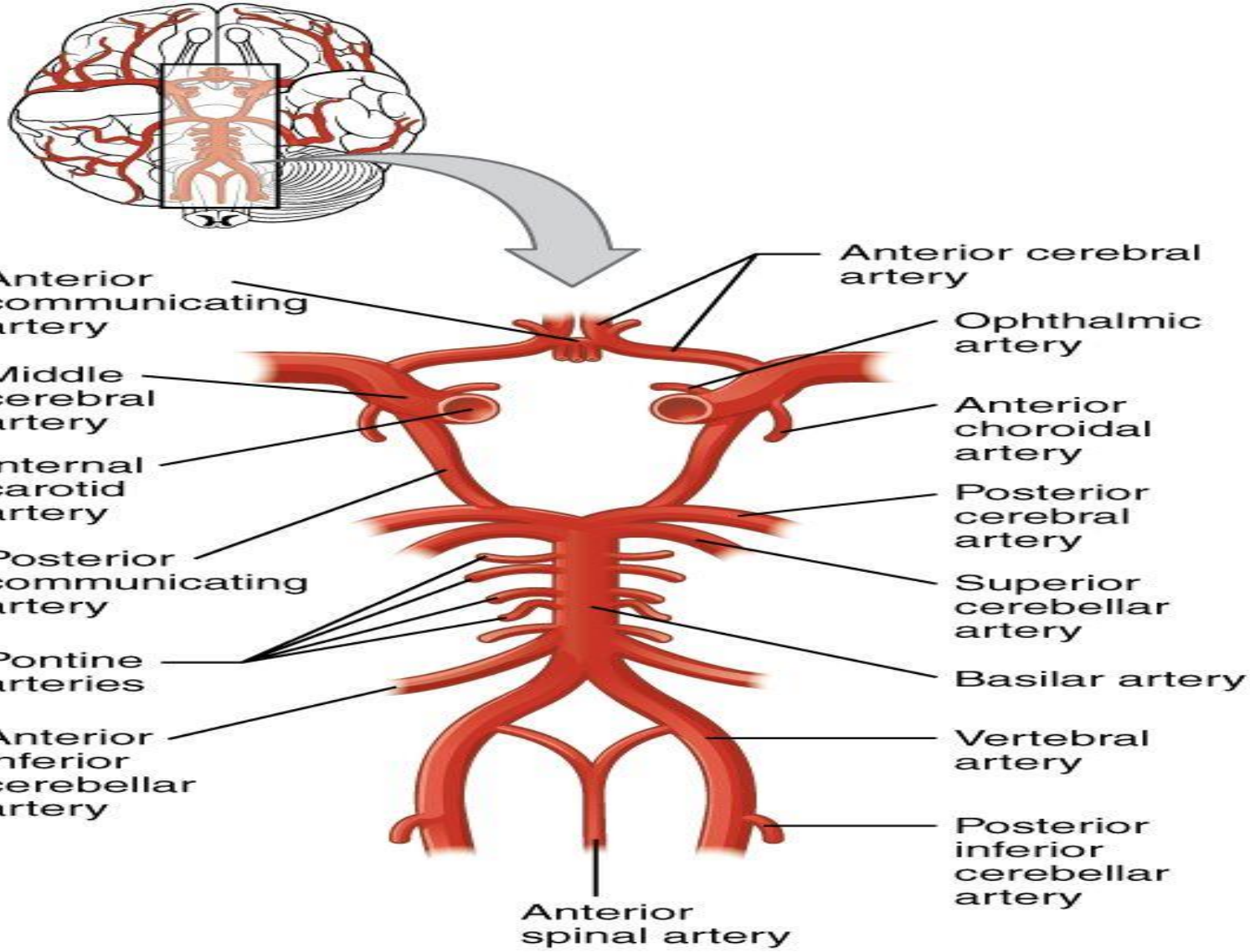
**Parietal Lobe**
- sensations
- language
- perception
- body awareness
- attention

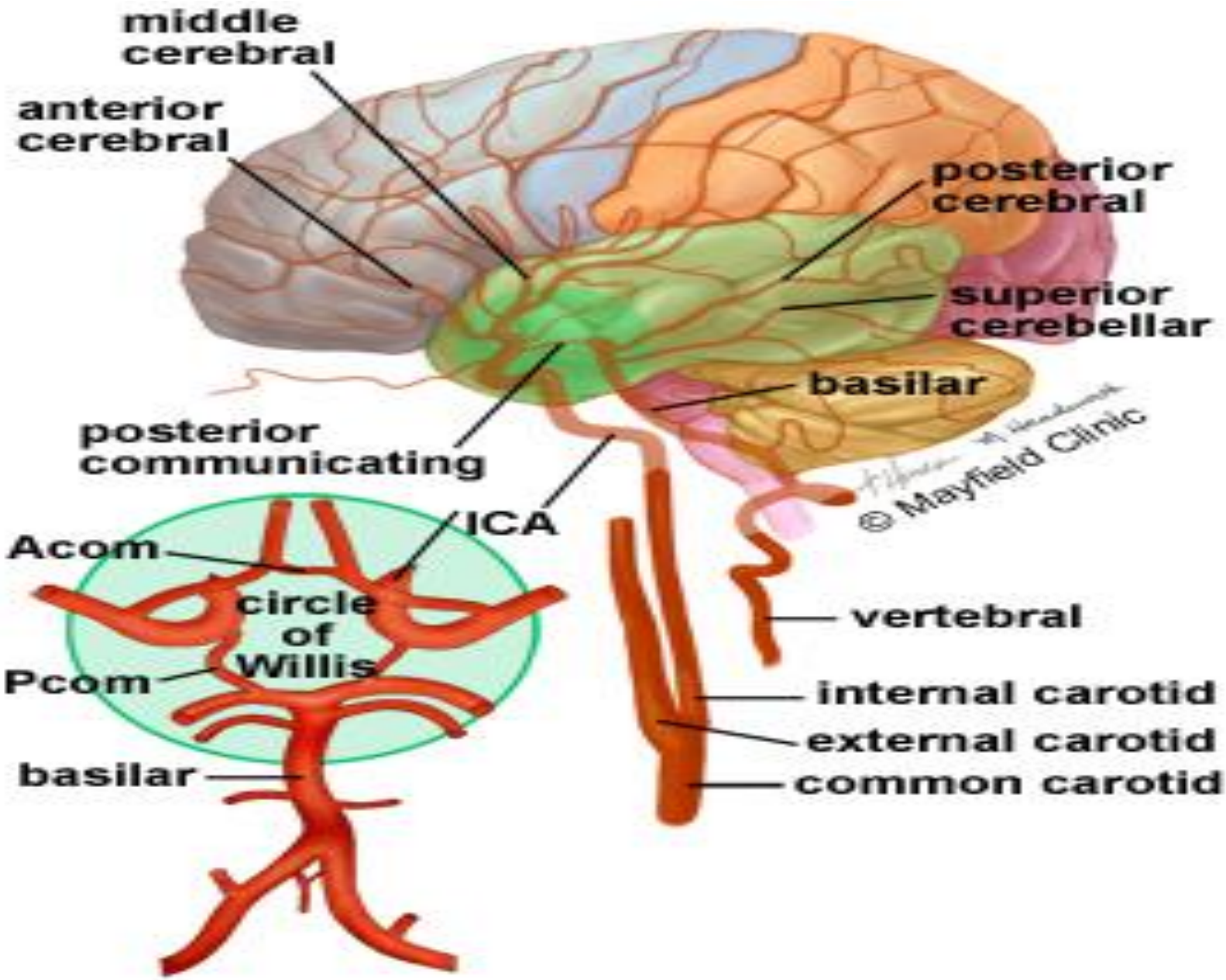
**Occipital Lobe**
- vision
- perception

**Wernicke's Area**
- language comprehension

**Cerebellum**
- posture
- balance
- coordination of movement

# Blood supply of brain




middle cerebral
anterior cerebral
posterior cerebral
superior cerebellar
basilar
posterior communicating
ICA
Acom
circle of Willis
Pcom
basilar
vertebral
internal carotid
external carotid
common carotid
© Mayfield Clinic

# Localization of stroke syndromes

- Clinical localization of the site of the lesion.
- Identifying the vascular territory and the vessel involved.
- Correlating with the imaging findings.

# CLASSIFICATION

- Large vessel stroke within the anterior circulation
- Large vessel stroke within the posterior circulation
- Small vessel disease of either vascular bed

# Cerebral circulation

- Anterior circulation- MCA, ACA, and Anterior choroidal artery

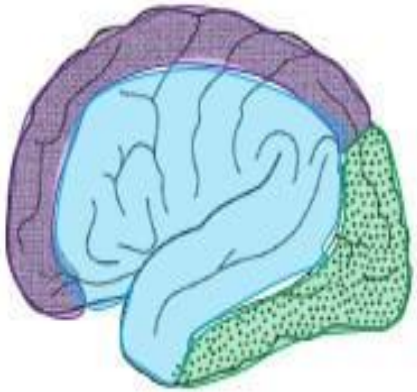
- Posterior circulation-Vertebral artery, Basilar artery and Posterior cerebral artery

# ANTERIOR CIRCULATION STROKE SYNDROMES

# *Stroke within the anterior circulation*

- Due to occlusion of Internal carotid artery and its branches

- Middle cerebral artery, Anterior cerebral artery and Anterior choroidal artery



# Blood Supply to the Brain



Anterior cerebral artery

Middle cerebral artery

Caudate nucleus

Putamen

Globus pallidus

Sylvian fissure

Internal capsule

Thalamus

Posterior cerebral artery

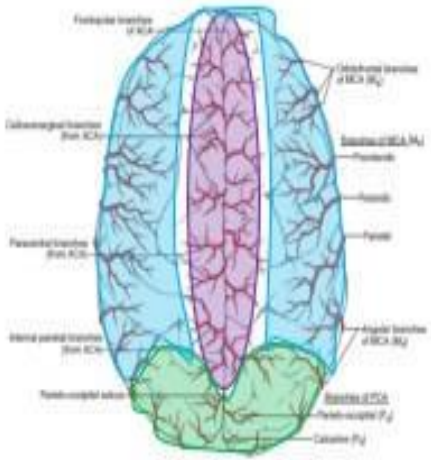
Carotid artery

ACA territory

MCA territory

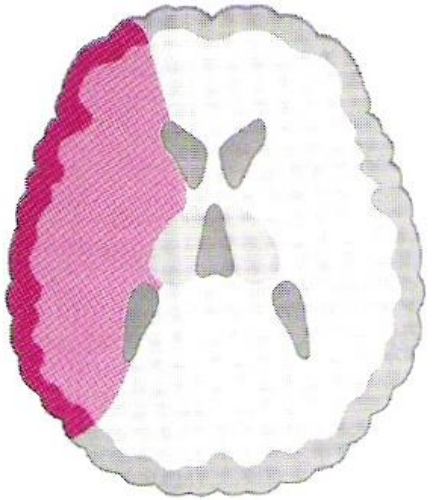
Anterior choroidal territory

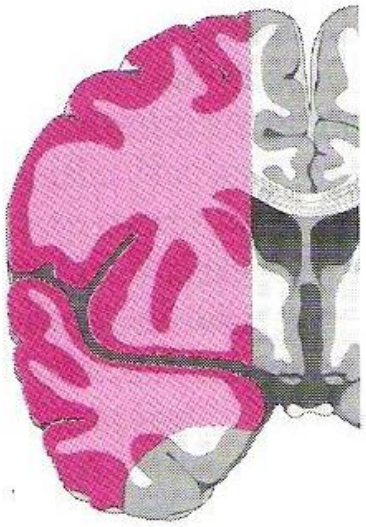
PCA territory



# Middle cerebral artery infarction - stem occlusion

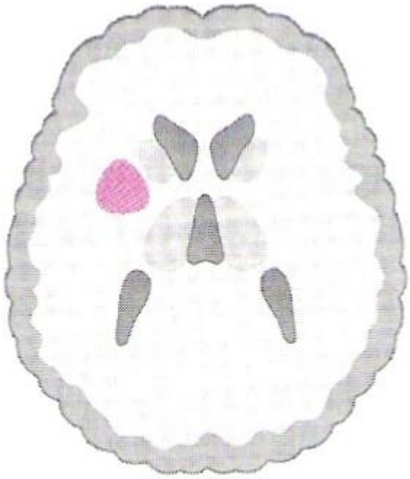
- Clinical features
  - Contralateral hemiplegia
  - Contralateral hemisensory loss
  - Contralateral gaze palsy
  - Contralateral hemianopia
  - Global dysphasia (Left sided lesion)
  - Anosognosia and amorphosynthesis (Right sided lesion)
  - Altered sensorium (due to edema)

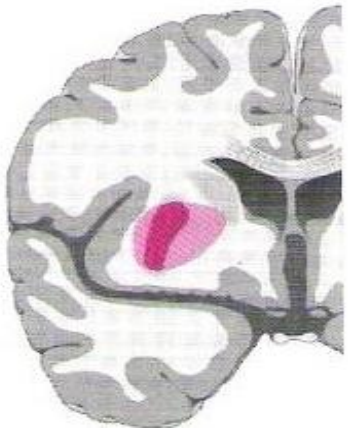




# Middle cerebral Artery infarction- lenticulostriatal occlusion

- Deep penetrating or lenticulostriate branches– Internal capsule, caudate nuclues, putamen and outer pallidus
- Occlusion of lenticulostriate branches-
- If ischemia of internal capsule produces pure motor or sensorymotor stroke contralateral to the side of lesion
- If ischemia of putamen, pallidus- predominantly parkinsonian features





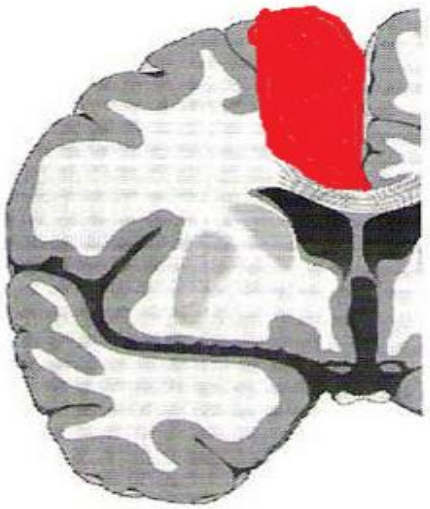
# Anterior cerebral artery infarction

- Clinical features
  - Contralateral

    a.paralysis of leg and foot with paresis of arm

    b.cortical sensory loss over leg and foot

    c.presence of primitive reflexes
  - Urinary incontinence
  - Gait apraxia
  - Mutism, delay and lack of spontaneity of motor acts
  - Apraxia of left sided limbs(with left sided lesion and corpus callosum involvement)



# *Anterior choroidal artery*

- Supplies posterior limb of internal capsule, retrolentiform and sublentiform parts

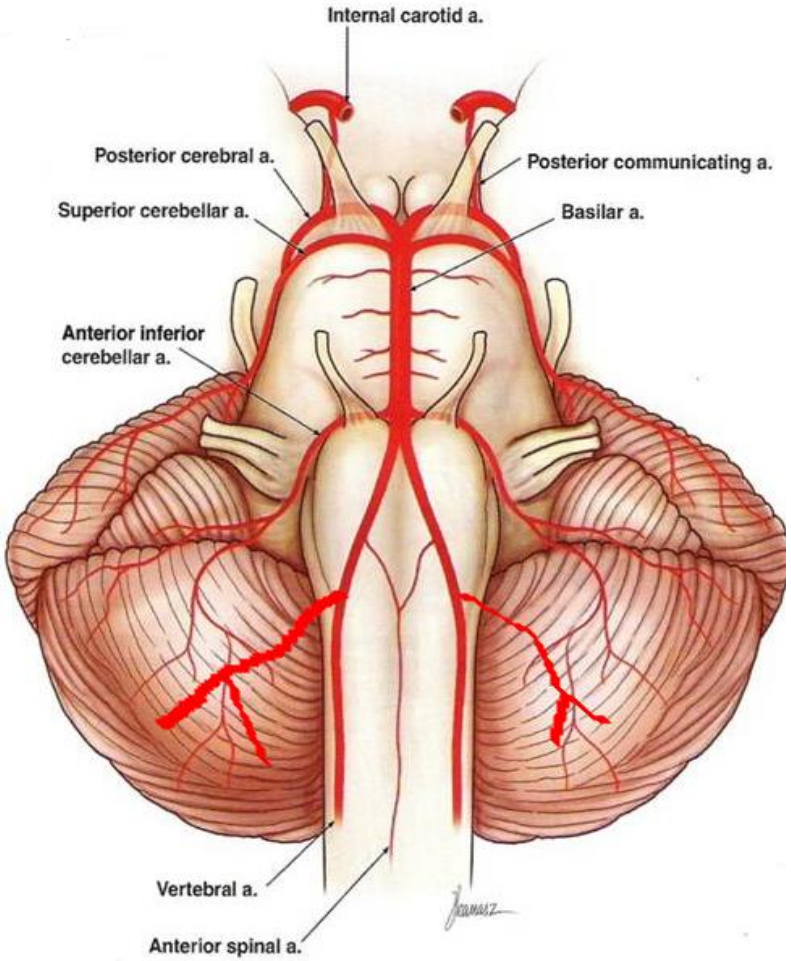
- Syndrome comprises
  - c/l hemiplegia
  - c/l hemianaesthesia
  - c/l homonymous hemianopia

# Posterior circulation stroke syndromes

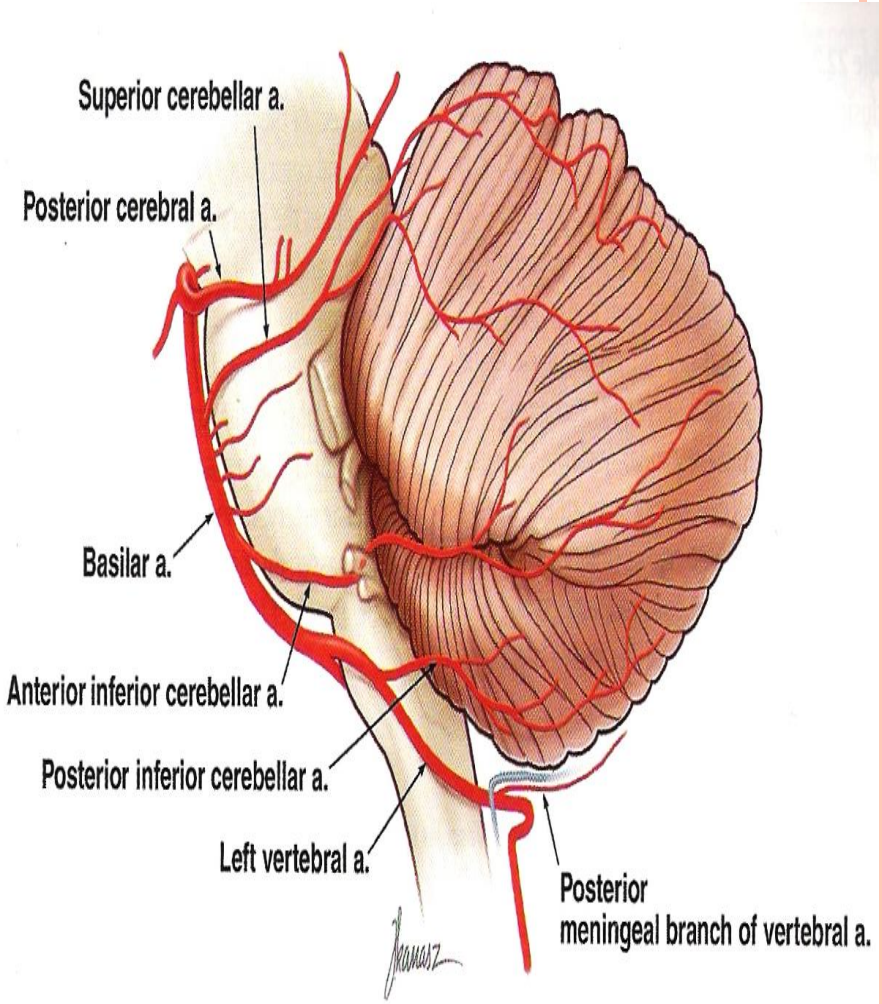
# Posterior circulation

- Supplies
  - Cerebellum
  - Medulla
  - Pons
  - Midbrain
  - Thalamus
  - Subthalamus
  - Hippocampus
  - Medial part of temporal lobe
  - Occipital lobe

# Posterior circulation




ventral view



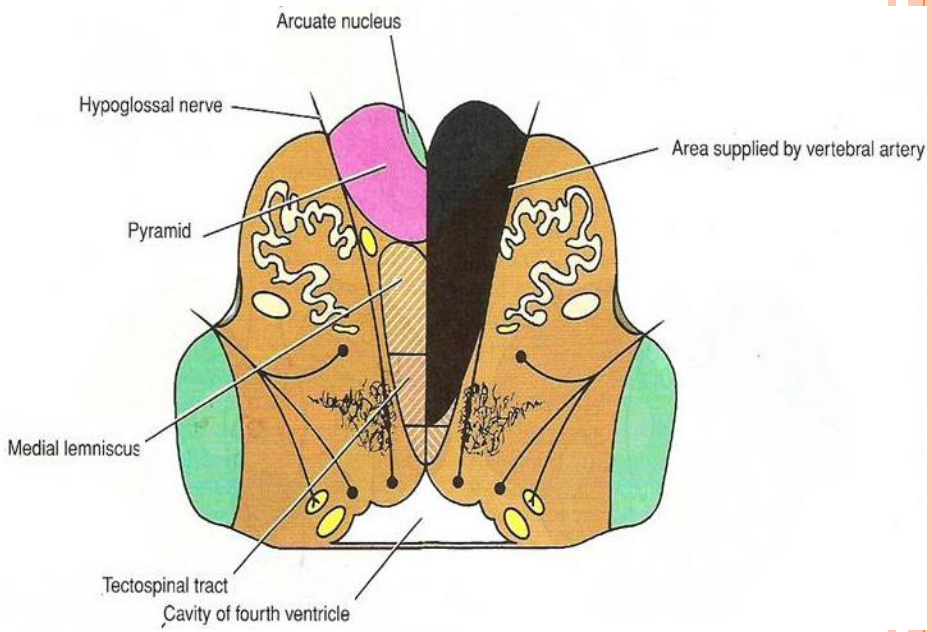
# LESIONS OF THE MEDULLA

- Medial medullary syndrome
- Lateral medullary syndrome

# MEDIAL MEDULLARY SYNDROME

A.IPSILATERAL

1.XII$^{th}$ nerve palsy




B.CONTRALATERAL

1.Hemiplegia – sparing the face

2.Hemianaesthesia sparing the face.

# LATERAL MEDULLARY SYNDROME

A. IPSILATERAL

1.X$^{th}$ cranial nerve palsy

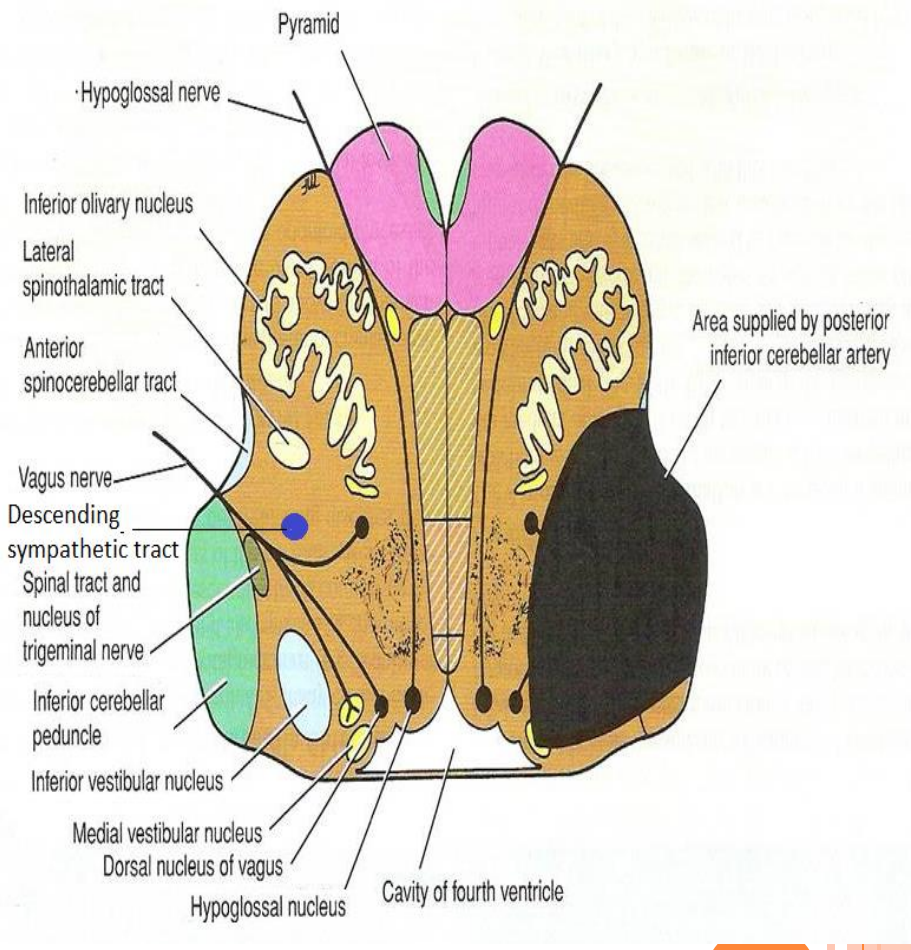
2.Cerebellar signs

3.Horner's syndrome

4.Impaired pain, temperature and touch On the upper half of face

B. CONTRA LATERAL

1.Impaired pain and temperature over the body



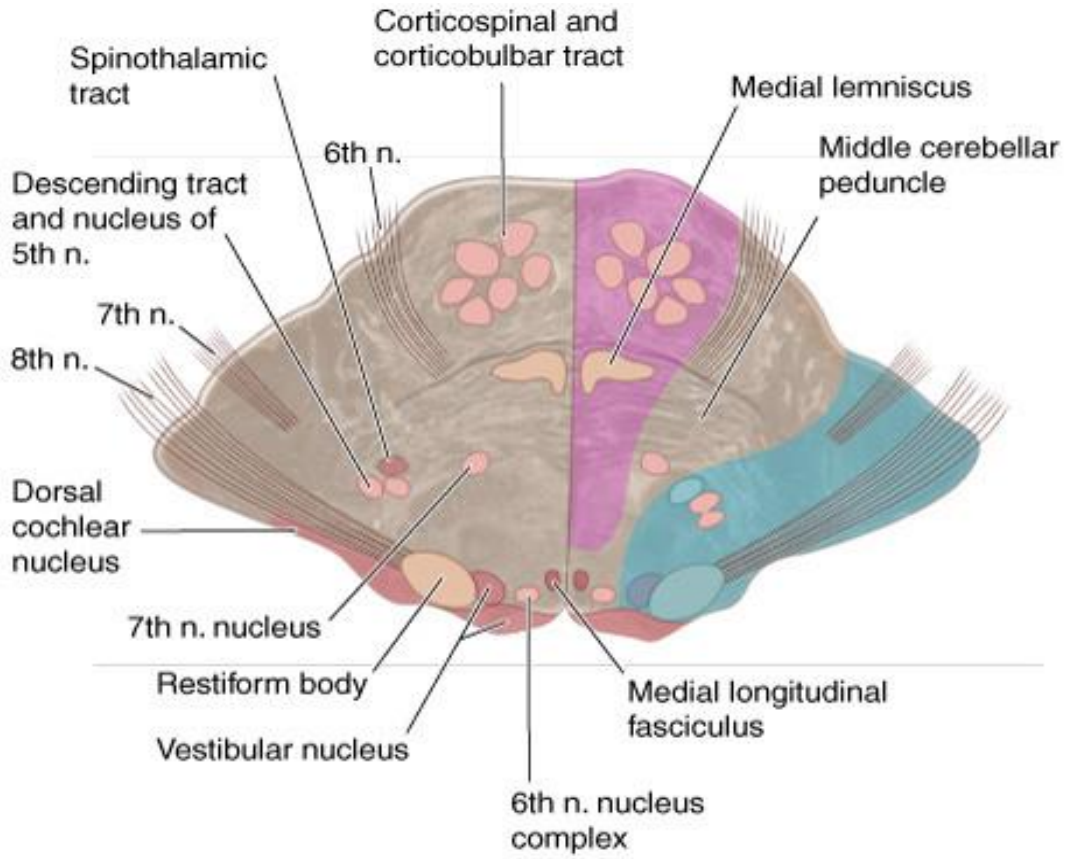
# *Basilar Artery*

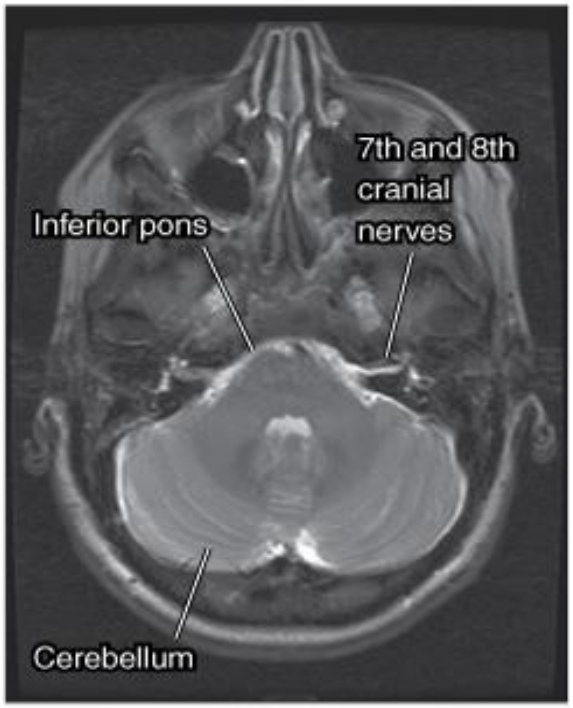
- Paramedian- wedge of pons in midline.
- Short circumerential- lateral two thirds of pons and middle and superior cerebellar peduncles.
- Long circumferential- Superior and anterior inferior cerebellar.

# *Basilar artery syndromes*

- Occlusion of basilar artery-b/l brainstem signs.
- Occlusion of basilar branch artery- unilateral motor, sensory and cranial nerves.
- Complete basilar artery occlusion(Locked in state)-b/l long tract(sensory/motor) with cranial nerve and cerebellar dysfunction- preserved consciousness, quadriplegia and cranial nerve signs.

# Inferior pontine syndrome



# Medial inferior pontine syndrome

- Results from thrombosis of the para median branches of the basilar artery. Affected structures--
- **Corticospinal tract**

Lesions result in

contralateral spastic hemiparesis.

- **Medial lemniscus**

Lesions result in

contralateral loss of tactile sensation from the trunk extremities.

- **Abducent nerve roots**

Lesions result in

ipsilateral lateral rectus paralysis.

# Lateral inferior pontine syndrome

- anterior inferior cerebellar artery (AICA) syndrome

Affected structures and resultant deficits include--

- **facial nucleus and intraaxial nerve fibers**

Lesions result in:

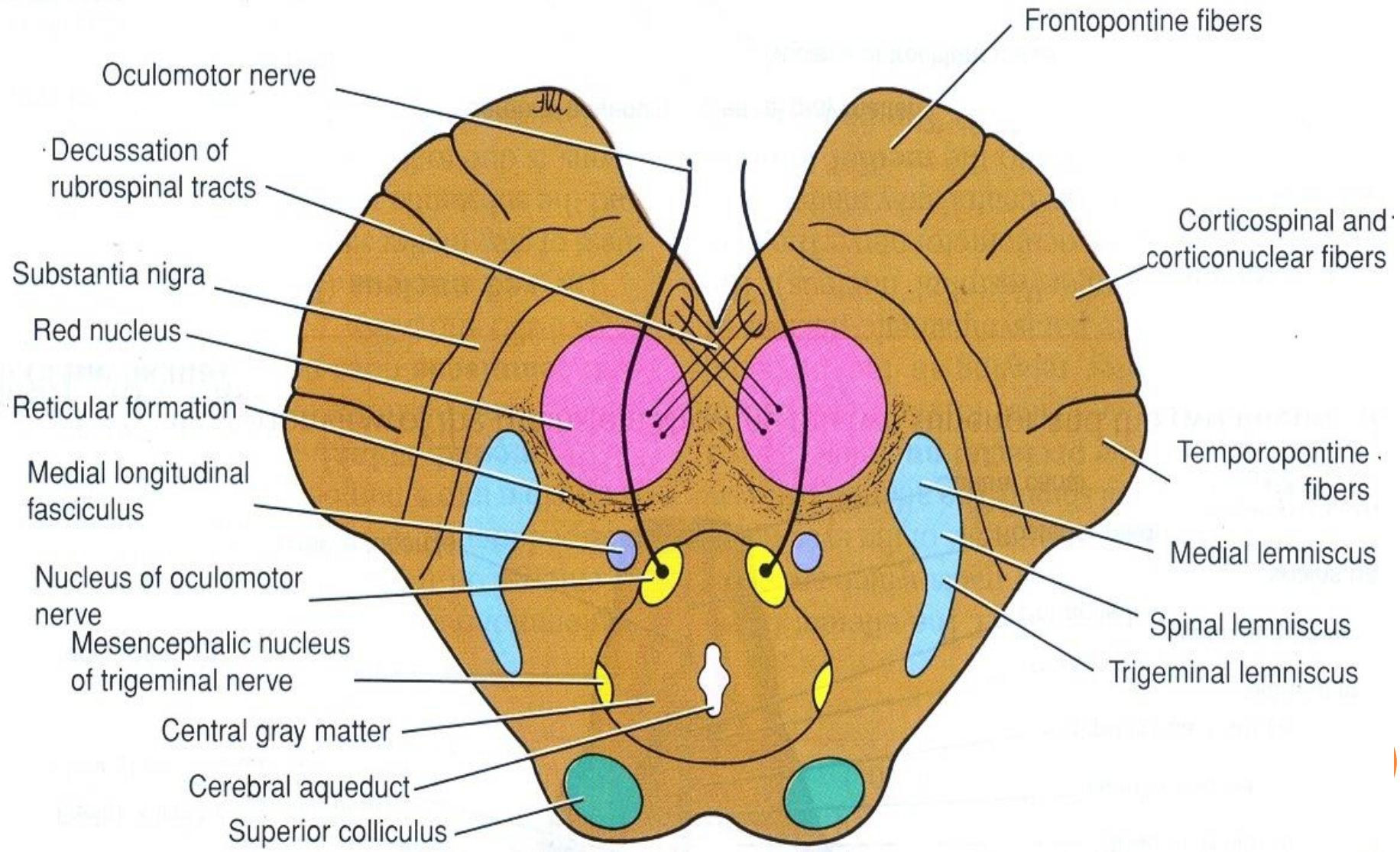
- Ipsilateral facial nerve paralysis
- Ipsilateral loss of taste from the ant. 2/3 of tongue
- Ipsilateral loss of lacrimation and reduced
- salivation
- Loss of corneal and stapedial reflexes

(efferent limbs).

# *Medial pontine syndromes*

- Caused due to occlusion of paramedian and short circumferential branches of basilar artery
- Corticobulbar and corticospinal-c/l face, arm and leg paralysis
- Cerebellar peduncles-ataxia of limb and gait

# MID BRAIN SYNDROME



# Weber syndrome-occlusion of perforating branch of posterior cerebral artery

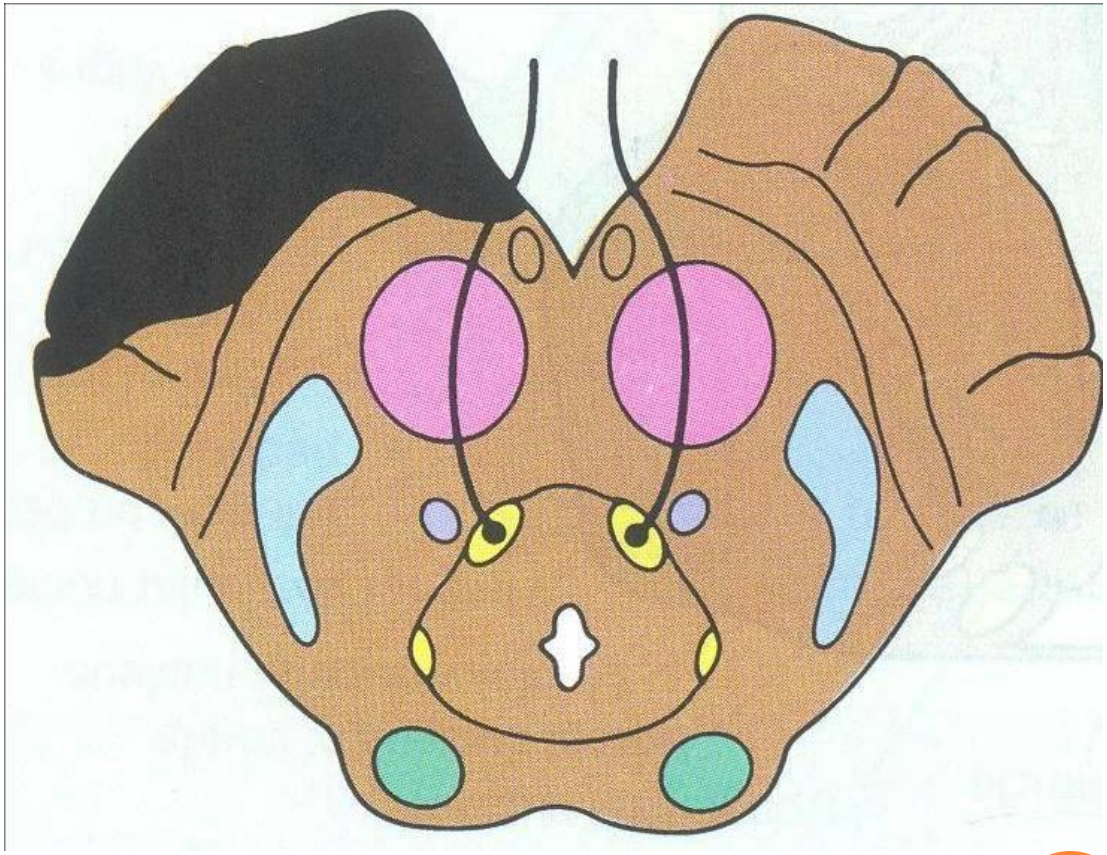
Clinical features

- 1.Ipsilateral

  a.3rd nerve palsy

- 2.Contralateral

  a.hemiplegia



# Benedikt syndrome-occlusion of perforating branch of posterior cerebral

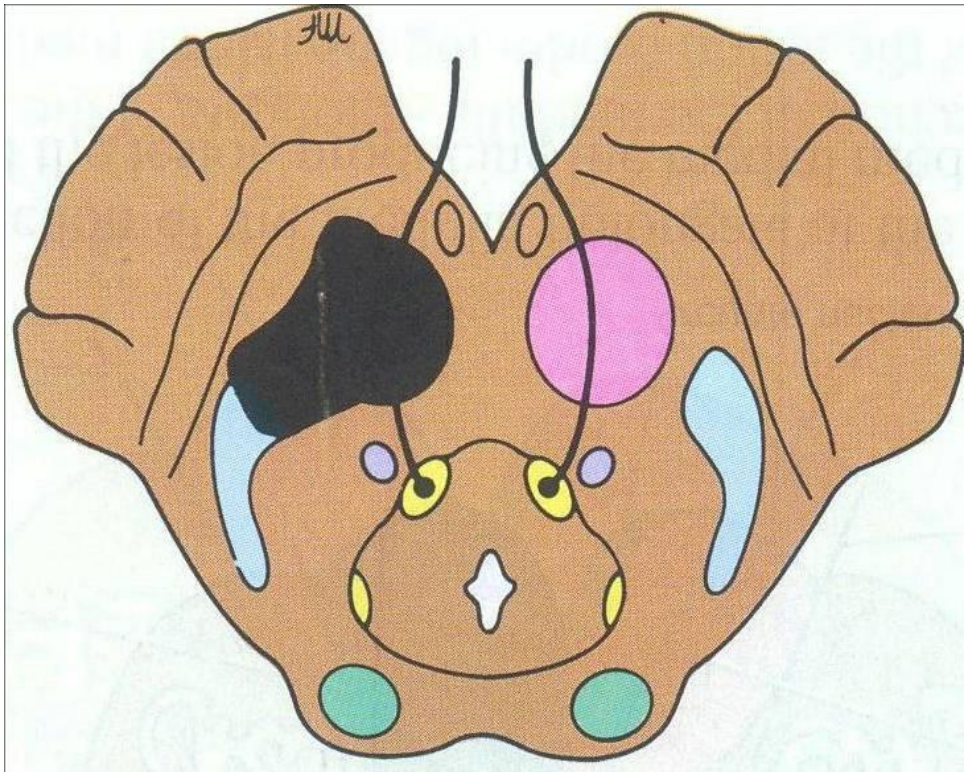
Clinical features

- 1.Ipsilateral

  a.3rd nerve palsy

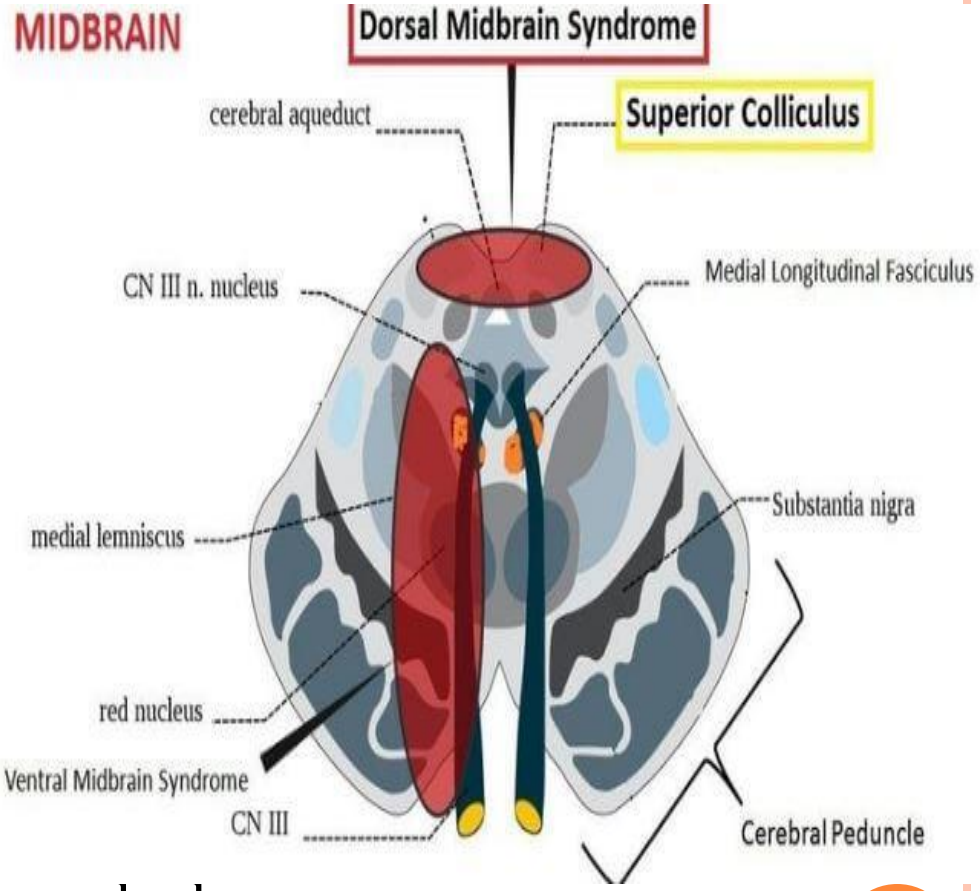
- 2.Contralateral

  a.cerebellar ataxia



# Dorsal midbrain (Parinaud's) syndrome

-paralysis of upward and downward gaze
-pupillary disturbances (Pseudo-Argyll Robertson pupils)
-absence of convergence (Convergence-Retraction nystagmus on Attempts at upward gaze)
-noncommunicating hydrocephalus



# Differentiating features between anterior and posterior circulation stroke

| Clinical features | Posterior circulation | Anterior circulation |
|---|---|---|
| **A.History** | | |
| 1.Vertigo | Present | Absent |
| 2.Unsteadiness | Present | Absent |
| **B.Physical findings** | | |
| 1.Crossed hemiplegia | Present | Absent |
| 2.Bilateral deficits | Present | Absent |
| 3.Cerebellar signs | Present | Absent |
| 4.Ocular findings(LMN/INO/Gaze deviation to paralysed side) | Present | Absent |
| 5.Dissociated sensory loss | Present | Absent |
| 6.Sensory loss over V1 and V2 | Present | Absent |
| 7.Horners syndrome | Present | Absent |

# Differential Diagnosis of Stroke

**Craniocerebral / cervical trauma**
**Meningitis/encephalitis**
**Intracranial mass**

- Tumor
- Subdural hematoma

**Seizure with persistent neurological signs**
**Migraine with persistent neurological signs**
**Metabolic**

- Hyperglycemia
- Hypoglycemia
- Post-cardiac arrest ischemia
- Drug/narcotic overdose

# INVESTIGATION OBJECTIVES

- To confirm the vascular nature of the lesion
- The pathological type of the vascular lesion
- The underlying vascular disease
- Risk factors present.

# Investigations in acute stroke:

| Diagnostic question | Investigation |
| --- | --- |
| Is it a vascular lesion? | CT/MRI |
| Is it ischaemic or haemorrhagic? | CT/MRI |
| Is it a subarachnoid haemorrhage? | CT<br>Lumbar puncture |
| Is there any cardiac source of embolism? | Electrocardiogram (ECG)<br>Echocardiogram |
| What is the underlying vascular disease? | Duplex ultrasound of carotids<br>Magnetic resonance angiography (MRA)<br>CT angiography (CTA)<br>Contrast angiography |
| What are the risk factors? | Full blood count<br>Cholesterol<br>Blood glucose |
| Is there an unusual cause? | ESR<br>Clotting/thrombophilia screen |

# General Investigations

- Identify conditions which may predispose towards premature cerebrovasculardisease.
- Full blood count – polycythemia, thrombocytopoenia.
- Blood glucose – diabetes mellitus.
- Serum lipids – hypercholesterolemia.
- Blood cultures – SBE.
- HIV screen – AIDS.
- Syphilis serology – VDRL.
- Clotting Screen.
- Thrombophilia Screen – Protein C, Protein S, AT- III.
- Anticardolipin antibodies – SLE.
- Lumbar Puncture – subarachnoid haemorrhage.

# INVESTIGATION MODALITIES: BRAIN

## NON-INVASIVE

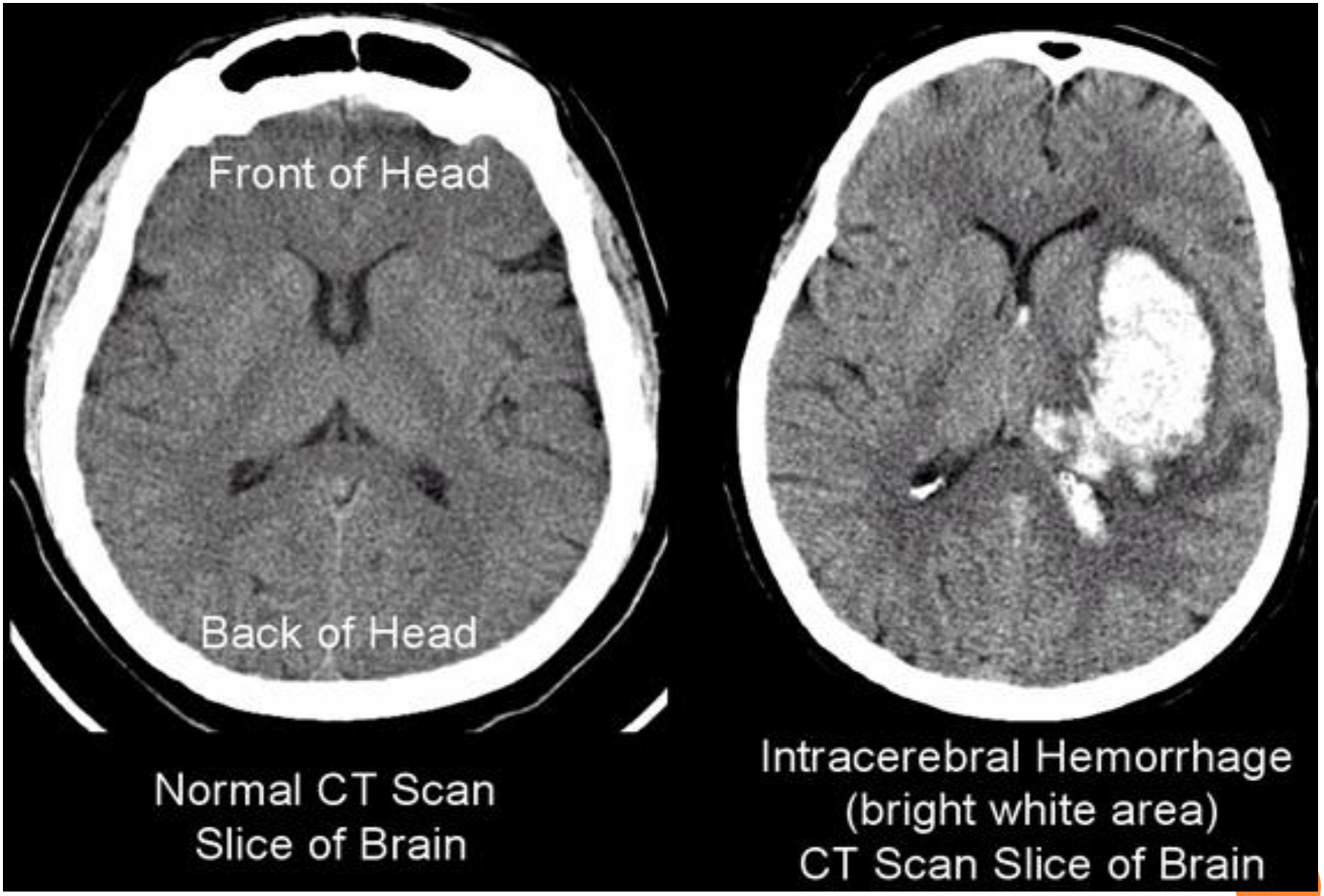
- CT Scan
- MRI Scan
- MR Angiography
- Doppler Ultrasound
- EEG
- PET
- SPECT

## INVASIVE

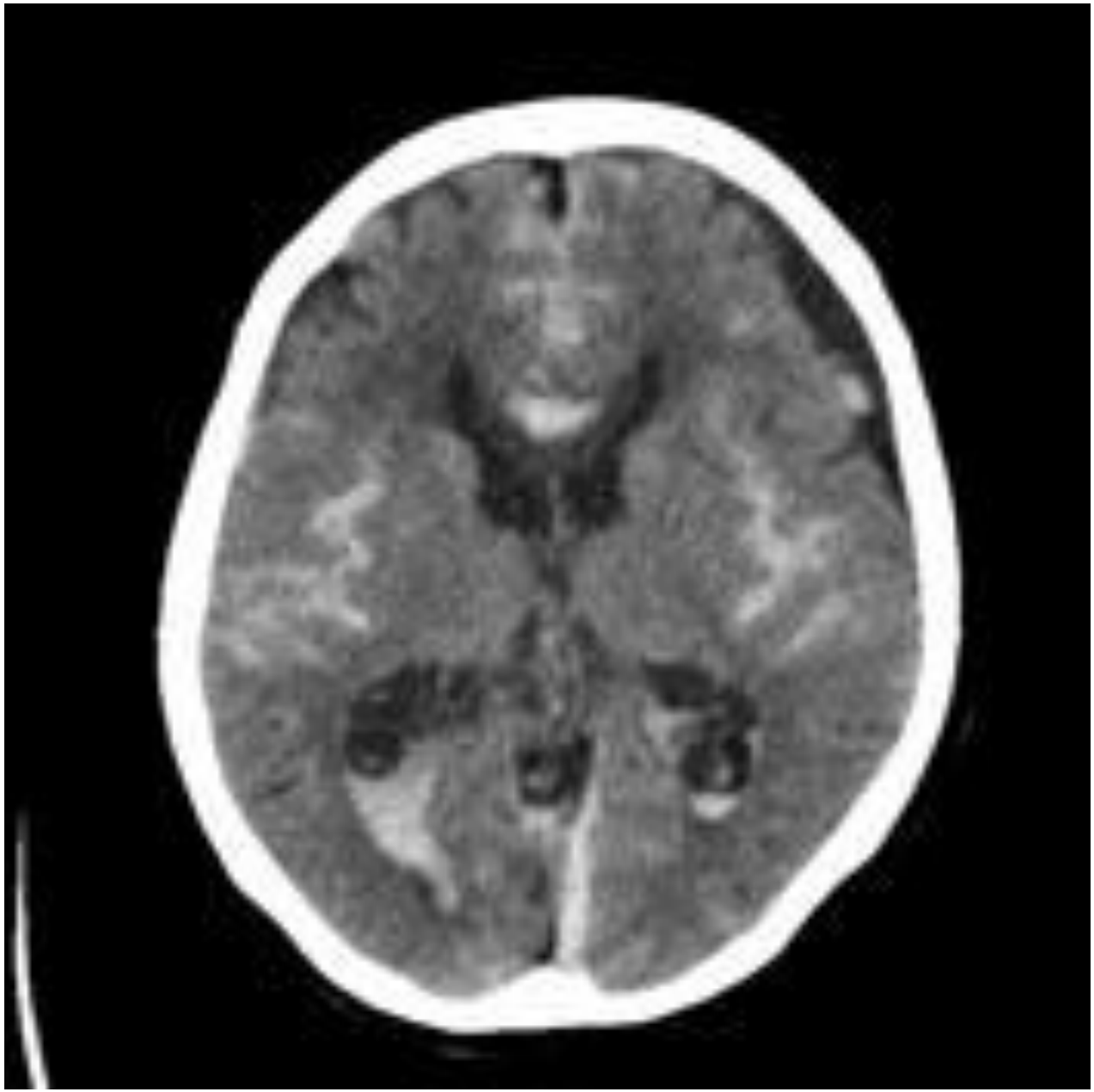
- Lumbar Puncture
- Contrast Angiography (Cerebral Arteriography)
- CT Angiography

# CT SCAN

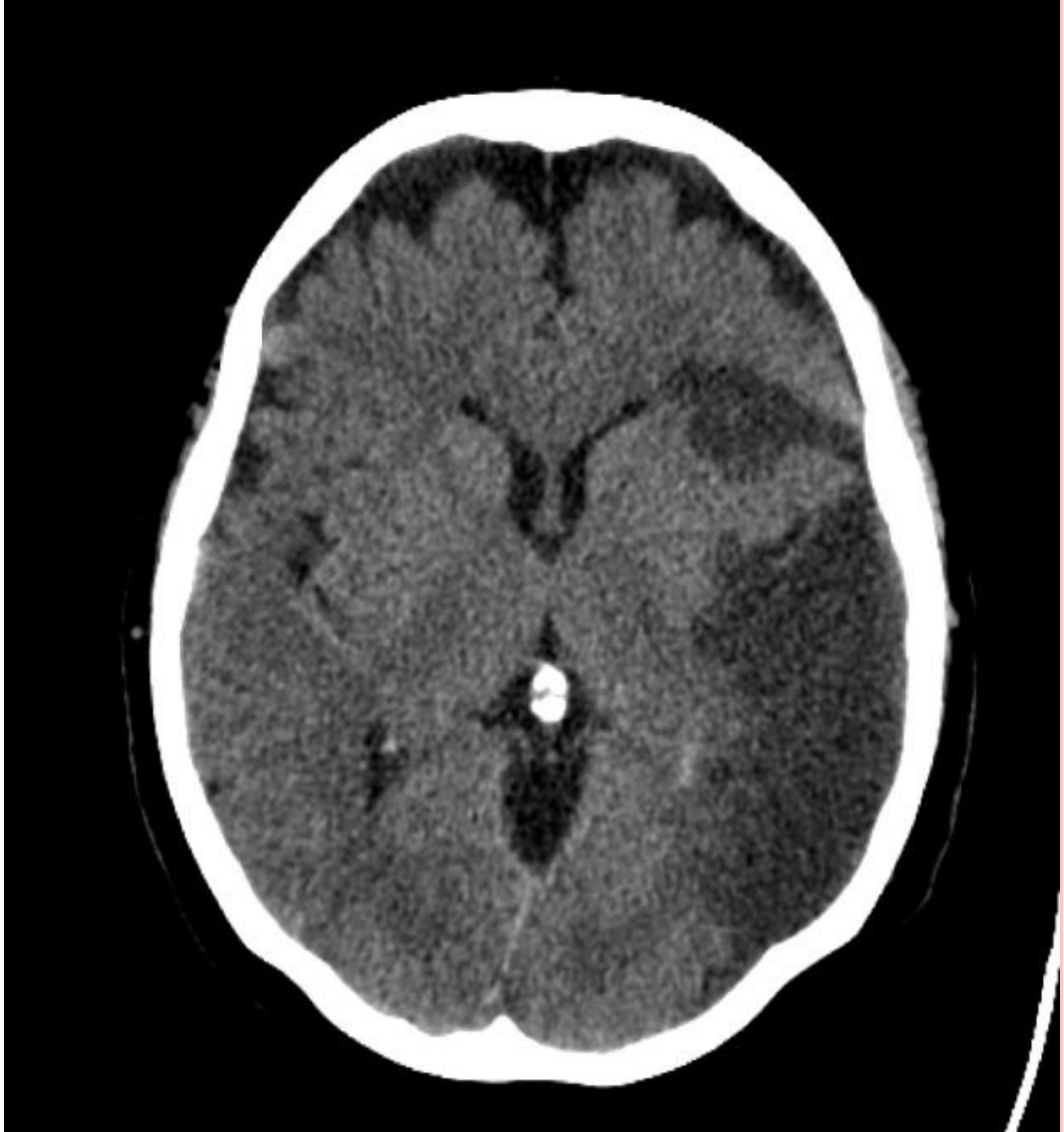
- Mandatory initial investigation
- **Haemorrhage** appears instantly as a **hyperdense** area
- **Infarct** appears as a **hypodense** area
- Infarct may not appear before 48 hrs
- MRI may be done instead but ct scan is more sensitive for detecting haemorrhage
- Diffusion weighted MRI is good for identifying ischaemic lesion.

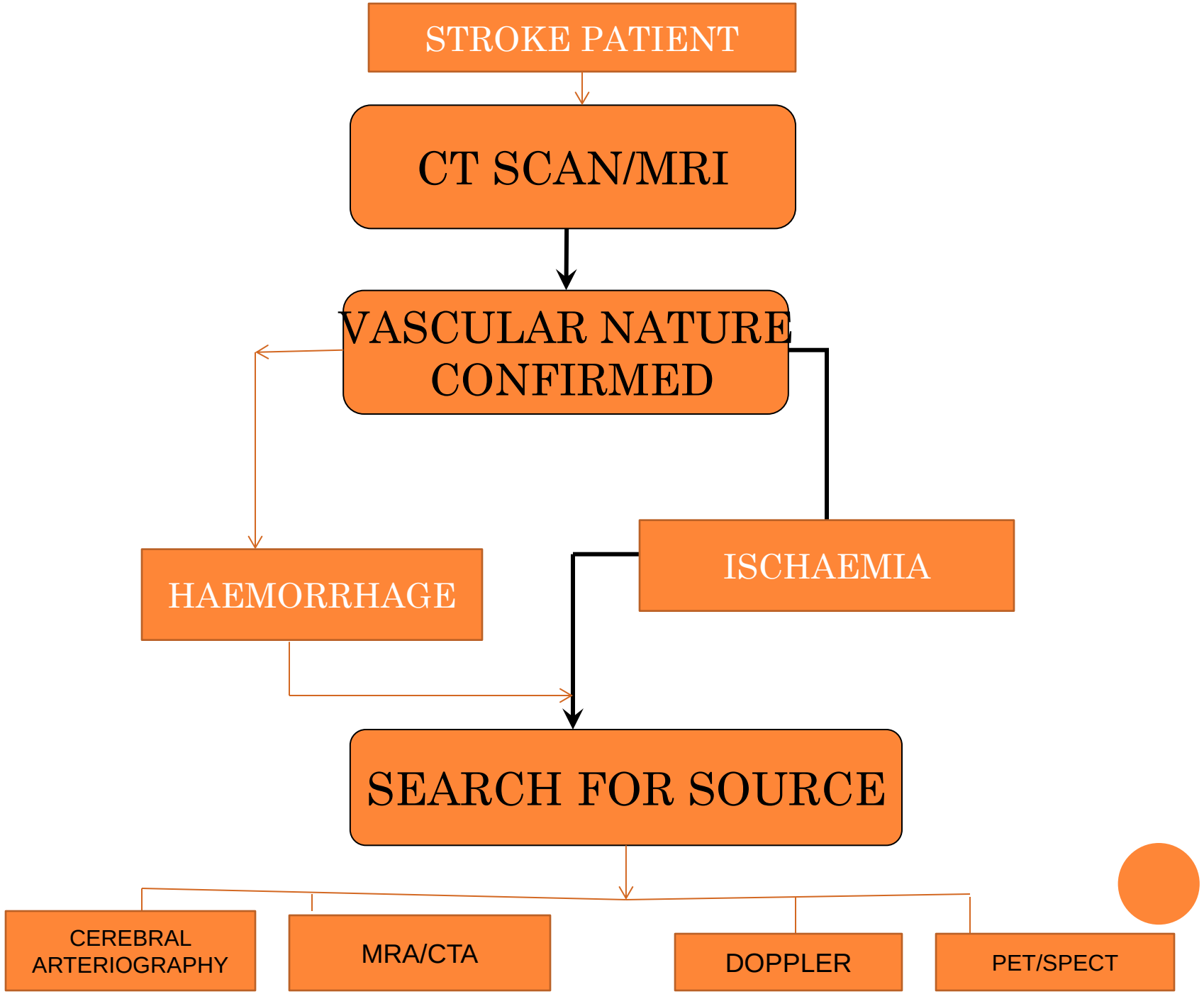

Front of Head
Back of Head
Normal CT Scan
Slice of Brain
Intracerebral Hemorrhage
(bright white area)
CT Scan Slice of Brain

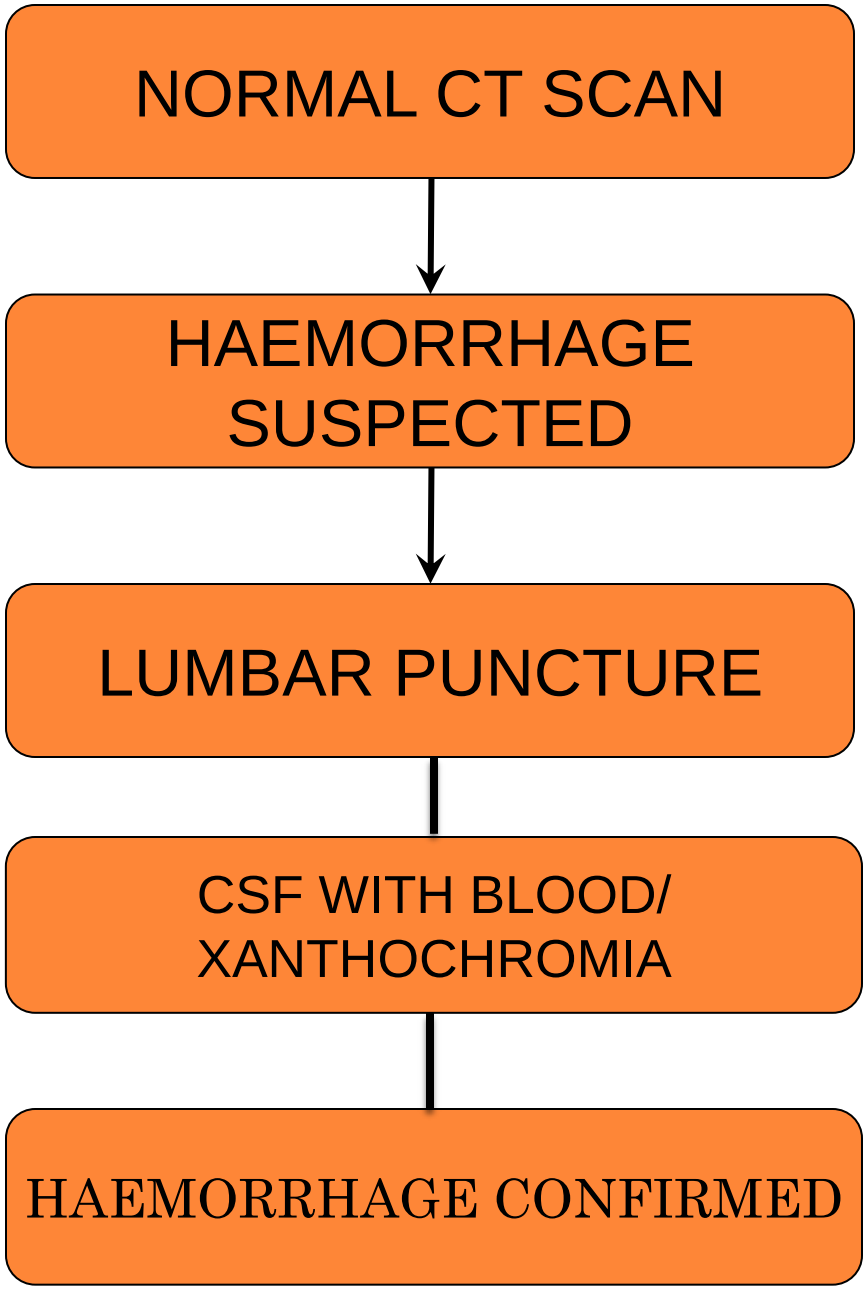
SAH



# ISCHEMIC STROKE




STROKE PATIENT
CT SCAN/MRI
VASCULAR NATURE CONFIRMED
HAEMORRHAGE
ISCHAEMIA
SEARCH FOR SOURCE
CEREBRAL ARTERIOGRAPHY
MRA/CTA
DOPPLER
PET/SPECT


NORMAL CT SCAN
HAEMORRHAGE SUSPECTED
LUMBAR PUNCTURE
CSF WITH BLOOD/ XANTHOCHROMIA
HAEMORRHAGE CONFIRMED

# TREATMENT OBJECTIVES

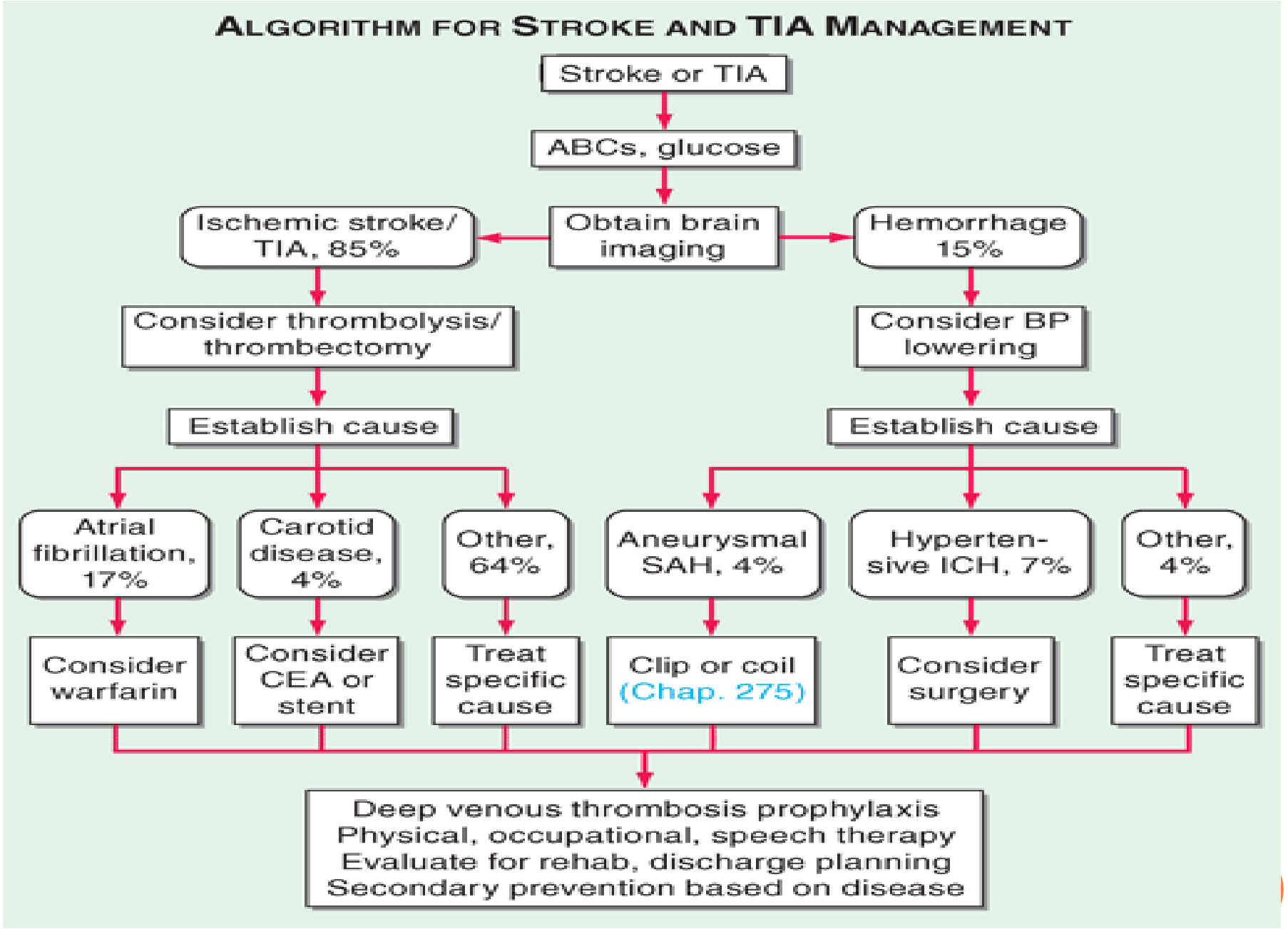
1. Minimize volume of brain reversibly damaged

2. prevent complications

3. Rehabilitation

4. reduce risk of reccurence

# Algorithm for Stroke and TIA Management




Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

# Assessment of a Person with Suspected Stroke & EMERGENCY SUPPORTIVE CARE

- EMS should be instructed in the rapid recognition, evaluation, treatment and transport
- Baseline assessment within minutes, CT scan ASAP)
- **Immediate evaluation of the following:**

  1. Airway
  2. Vital signs
  3. General medical assessment (including evidence of injury, cardiovascular abnormalities)
  4. Neurological assessment (frequent)

- Maintenance of adequate tissue oxygenation: protecting the airway, O2 inhalation
- Maintaining optimal blood pressure (autoregulation faulty or lost in stroke patients)

# Primary and secondary prevention

A- antiplatelet and anti coagulants

B- blood pressure lowering medication

C- cholesterol lowering, cessation of smoking

D- diet

E- exercise

# MANAGEMENT OF A TRANSIENT ISCHAEMIC ATTACK (TIA)

**MEDICAL MANAGEMENT**

(if diffuse atherosclerotic disease or poor operative candidates)

1. **Stop smoking**
2. Concurrent medical problems to be addressed:
   - Emboli from heart and other parts of cardiovascular system

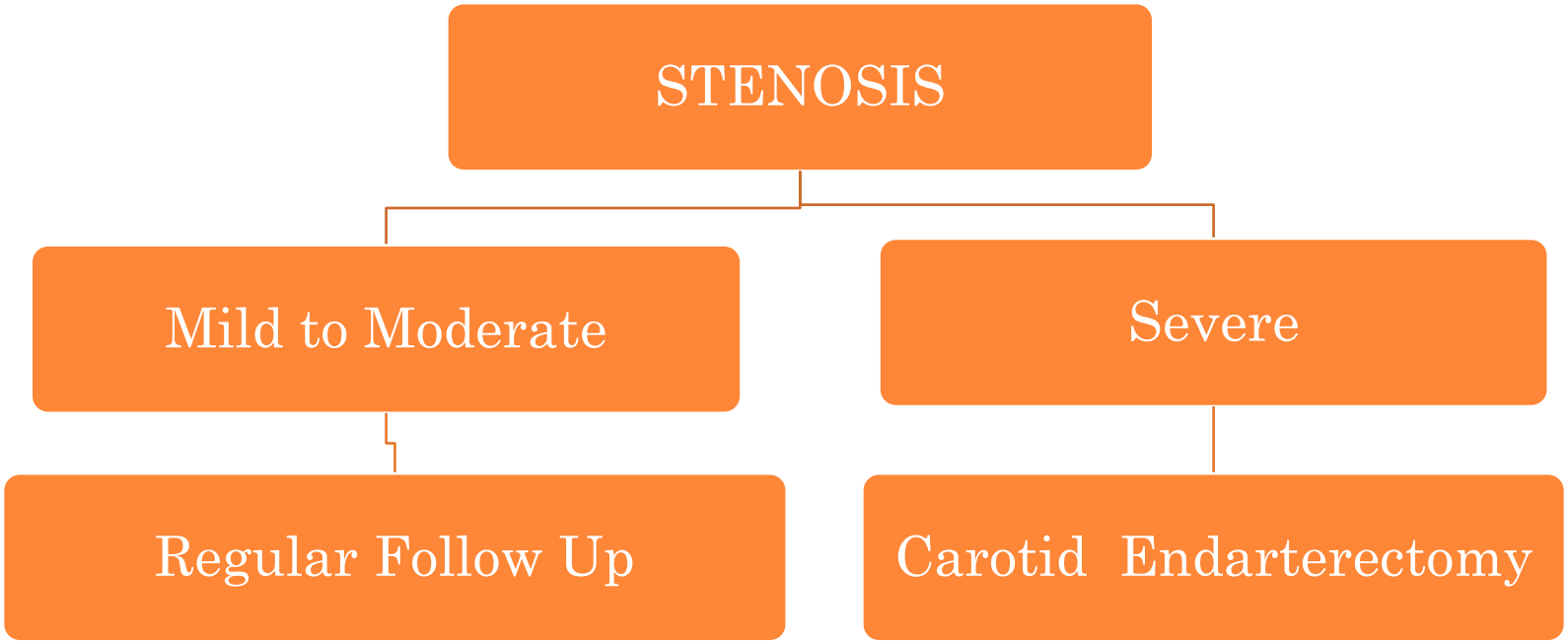
     (a) anti coagulants: Heparin(IV), Warfarin(oral)

     (b) anti platelet drugs: Aspirin(oral), Ticlopidine
   - Diabetes, Hypertension, Hyperlipidemia

# MANAGEMENT OF A TRANSIENT ISCHAEMIC ATTACK(TIA) – CONT'D

**SURGICAL MANAGEMENT**

- CAROTID AND CEREBRAL ARTERIOGRAPHY




- All above can be done only if there is relatively little atherosclerosis elsewhere in cerebrovascular system.

# MANAGEMENT OF AN ACUTE EPISODE OF STROKE

- AIRWAY - Maintain airway, prevent aspiration, keep nil per oral
- BREATHING - Maintain oxygen saturation > 97%
  - Supplementary oxygen
- CIRCULATION - Adequacy of pulse and BP
  - Fluid, Anti Arrhythmics, Ionotropes
- HYDRATION - Prevent dehydration ; give adequate fluids
  - Parenteral or via nasogastric tube
- NUTRITION - Nutritional supplements and Nasogatric feeding
- MEDICATION - Administer medication also by routes other than oral

# MANAGEMENT OF AN ACUTE EPISODE OF STROKE CONT'D

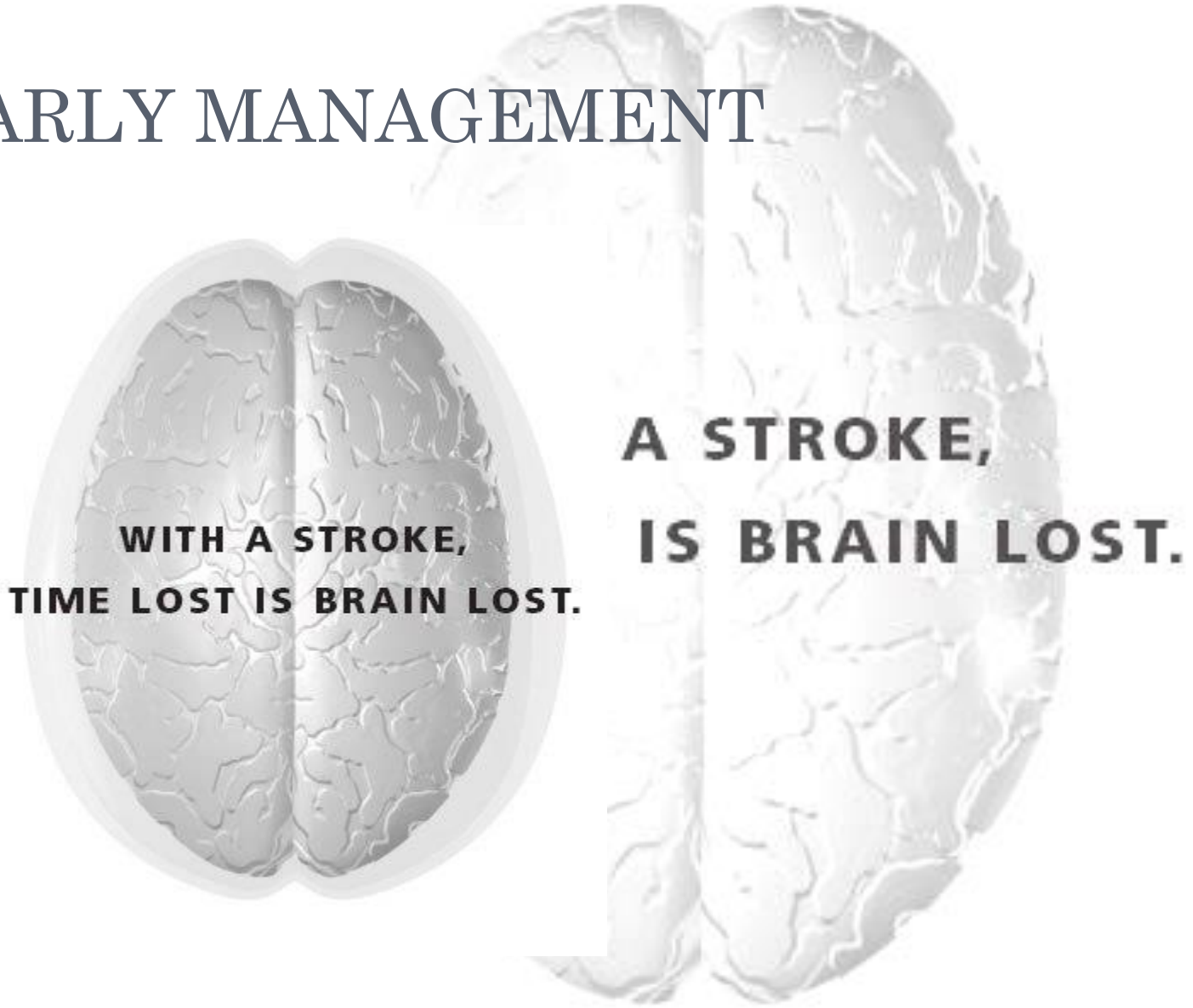
- BLOOD PRESSURE - unless indicated (heart or renal failure,hypertensive encephalopathy or aortic dissection) it should not be lowered for the fear of expansion of infarct.

  **Ischaemic stroke - maintain 180/110 mm Hg**

  **Haemorrhagic stroke – keep MAP <115 mm Hg**
- BLOOD GLUCOSE - INSULIN to treat hyperglycaemia(can increase infarct size)

  - maintain < 200mg%
- TEMPERATURE - early use of antipyretics
- PRESSURE AREAS – To prevent occurrence of decubitus ulcers
- INCONTINENCE

# EARLY MANAGEMENT



# ACT FAST at the First Sign of STROKE


www.strokemn.org

Minnesota Stroke Association
Chapter of the National Stroke Association

# ISCHAEMIC STROKE

- THROMBOLYTICS and REVASCULARISATION -
  - tPA (alteplase)-0.9mg/kg(max 90mg)
    10% of dose – initial IV bolus
    remainder infused over one hour
  - to be used < 3 hrs of onset of symptoms (for maximum efficacy)
  - haemorrhage to be ruled out

- NEUROPROTECTIVE AGENTS.

# ANTI PLATELET THERAPY

- Asprin, Clopidogrel

  - act by inhibiting platelet aggregation and adhesion.

  - aspirin 300mg single dose to be given immediately following diagnosis.

  - if alteplase given it can be with held for 24 hrs.

  - later aspirin at a dose of 75 mg in combination with clopidogrel 75 mg daily for about one year duration.

# ANTI COAGULANTS

- HEPARINS , WARFARIN

  -**heparins** act by accelerating the inhibition of factor II and factor X of coagulation cascade

  -**warfarin** antagonises vitamin K to prevent activation of clotting factors

  -decrease risk of recurrence and venous thromboembolism

  -intra cranial haemorrhage to be excluded before therapy

  -more useful if stroke is evolving

- HYPEROSMOLAR AGENTS
  - reduce cerebral oedema
  - 20% mannitol IV – 100ml TID
  - oral glycerol if swallow is normal
- Concurrent medical problems such as atrial fibrillations to be tackled
- OTHERS:

- PENTOXYPHYLLINE to be used within 12 hrs

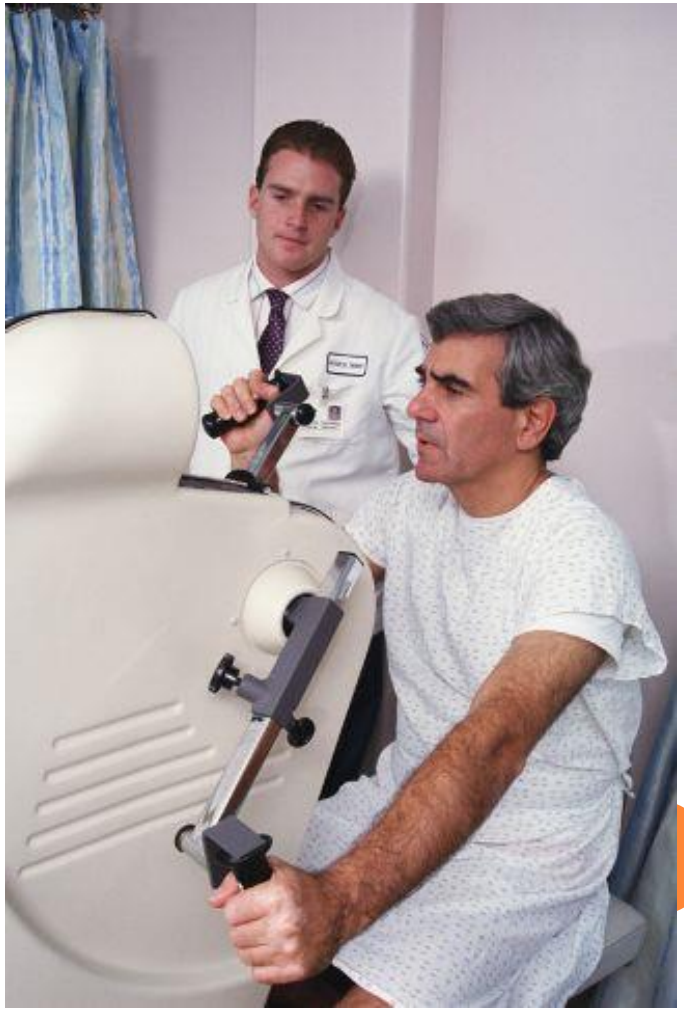
-NEUROPROTECTIVE AGENTS

# HAEMORRHAGIC STROKE

- Control of hypertension
- Control coagulation abnormalities (esp due to oral anticoagulants)
- Surgical decompression
- Surgery for aneurysms and arterio-venous malformations
- Anti platelet and anti coagulants are contraindicated

# REHABILITATION

- Physiotherapy - as early as possible
- Occupational therapy
- Speech therapy
- Improve quality of life with motor aids -leg brace, toe spring , cane , walking stick



# SECONDARY PREVENTION

- Blood pressure control
- Diabetes Management
- Lipid Management
- Smoking Cessation
- Alcohol Moderation
- Weight Reduction/Physical Activity
- Carotid Artery Interventions
- Anti platelet agents / Anti coagulants
- Statins
- Diuretics +/- ACE inhibitors

## 27.8 Complications of acute stroke

| Complication | Prevention | Treatment |
|---|---|---|
| **Chest infection** | Nurse semi-erect<br>Avoid aspiration (nil by mouth, nasogastric tube, possible gastrostomy) | Antibiotics<br>Physiotherapy |
| **Epileptic seizures** | Maintain cerebral oxygenation<br>Avoid metabolic disturbance | Anticonvulsants |
| **Deep venous thrombosis/ pulmonary embolism** | Maintain hydration<br>Early mobilisation<br>Anti-embolism stockings<br>Heparin (for high-risk patients only) | Anticoagulation (exclude haemorrhagic stroke first) |
| **Painful shoulder** | Avoid traction injury<br>Shoulder/arm supports<br>Physiotherapy | Physiotherapy<br>Local corticosteroid injections |
| **Pressure sores** | Frequent turning<br>Monitor pressure areas<br>Avoid urinary damage to skin | Nursing care<br>Pressure-relieving mattress |
| **Urinary infection** | Avoid catheterisation if possible<br>Use penile sheath | Antibiotics |
| **Constipation** | Appropriate aperients and diet | Appropriate aperients |
| **Depression and anxiety** | Maintain positive attitude and provide | Antidepressants |

# Poor prognostic factors in stroke

- Accompanying fever
- Hypotension
- Low oxygen saturation
- Hypoglyemia & hyperglycemia
- Pontine haemmorhage
- Low gcs score
- Heart failure
- Severity of hemiparesis

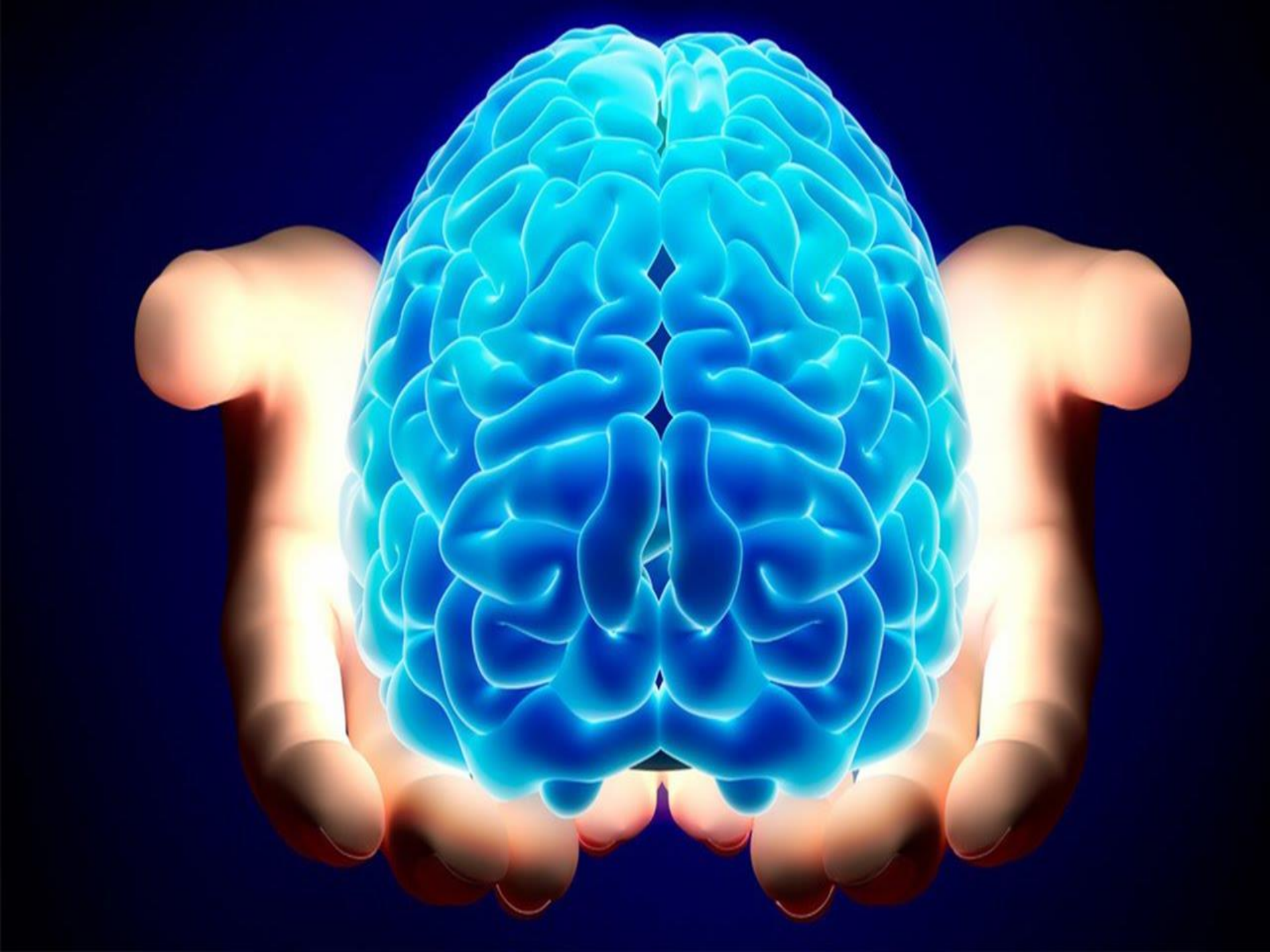
# PROGNOSIS

**ISCHAEMIC STROKE**

- Mortality rate in first 30 days is 8-12%
- Can vary depending upon size, location, symptoms of stroke
- Time that elapses from the event to medical intervention
- First 3 hrs after stroke - **GOLDEN PERIOD**

**INTRACEREBRAL HAEMORRHAGE**

- Mortality rate in first 30 days is almost 50%
- Site and extent of hematoma also plays a role in determining the prognosis
- **Hamorrhagic strokes have a poor prognosis compared to ischaemic type .**



- Thank You