LOCALISING A CORTICAL STROKE

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DEFINITION OF STROKE

- It is an abrupt onset of a focal neurological deficit which can be attributable to a vascular cause.
- Fever and hyperglycemia- 2 factors that worsen the prognosis of a stroke.
- If a neurological stroke clears within 1 hour (earlier guidelines mention 24 hour as the cut off), it is called a TIA (Transient ischemic attack).
- Stroke results in liquefactive necrosis in the brain.

Ischemic strokes (85%) are more common than Hemorrhagic strokes (15%).

Ischemic strokes can take genesis mostly from Anterior circulation (65%) and the posterior circulation (20%) as well.

With respect to etiology, embolic strokes are more common than thrombotic strokes.

- Causes of Thrombotic stroke:
- Atherosclerosis (MC)
- Takayasu arteritis

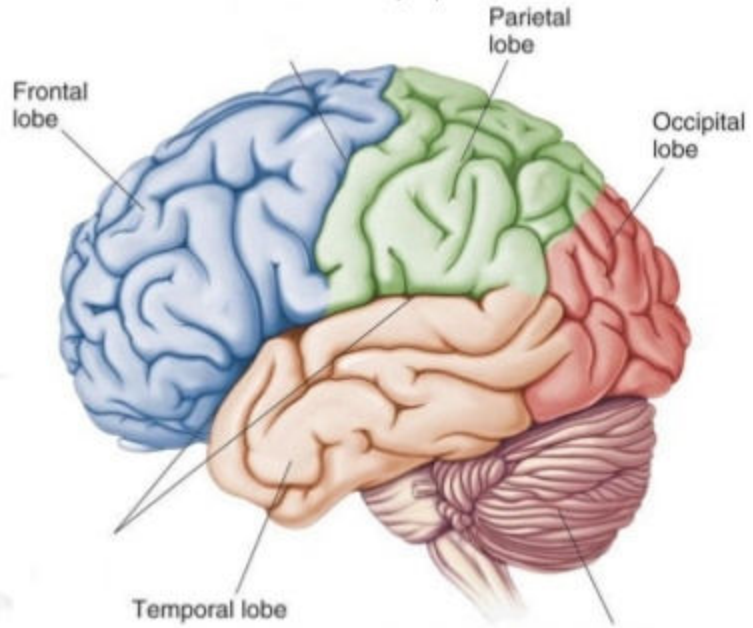
- Causes of Embolic stroke
- Non-rheumatic AF / rheumatic AF
- MS, AS
- Prosthetic valves
- Sick sinus syndrome
- Recent MI.

LOCALISING A STROKE/NEUROLOGICAL DEFICIT

Areas of the cortex.

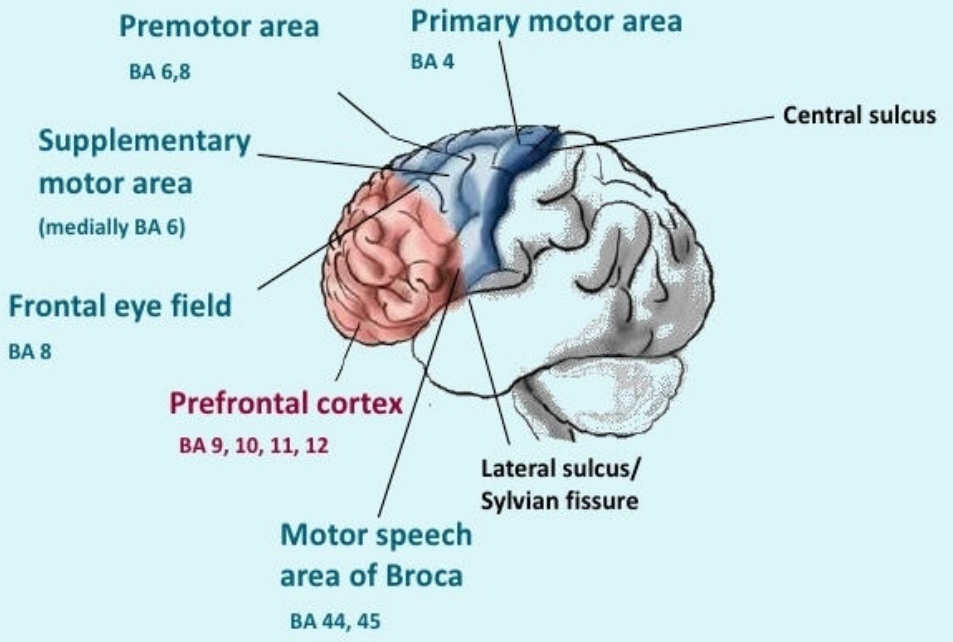
Lobes of the Brain (4)

- Frontal
- Parietal
- Occipital
- Temporal

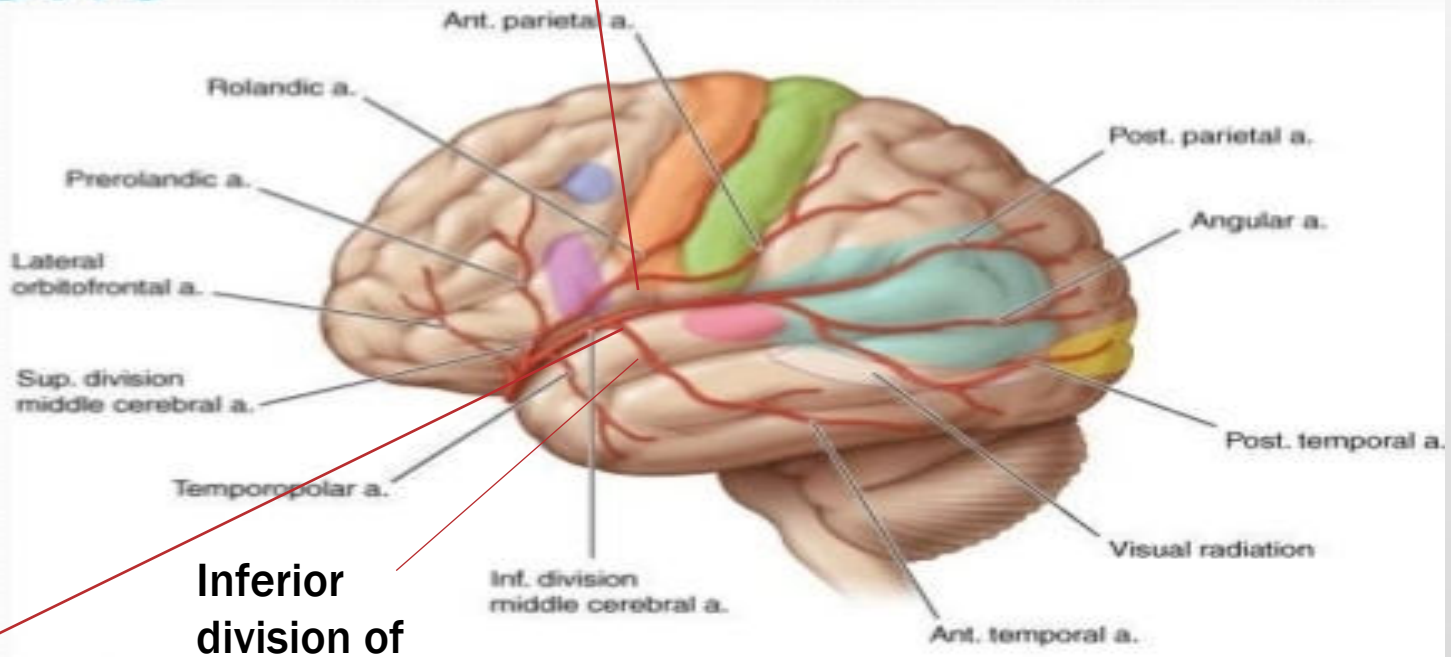


* Note: Occasionally, the Insula is considered the fifth lobe. It is located deep to the Temporal Lobe.

Functional Frontal Lobe Anatomy



M2 segment



Superior division of MCA

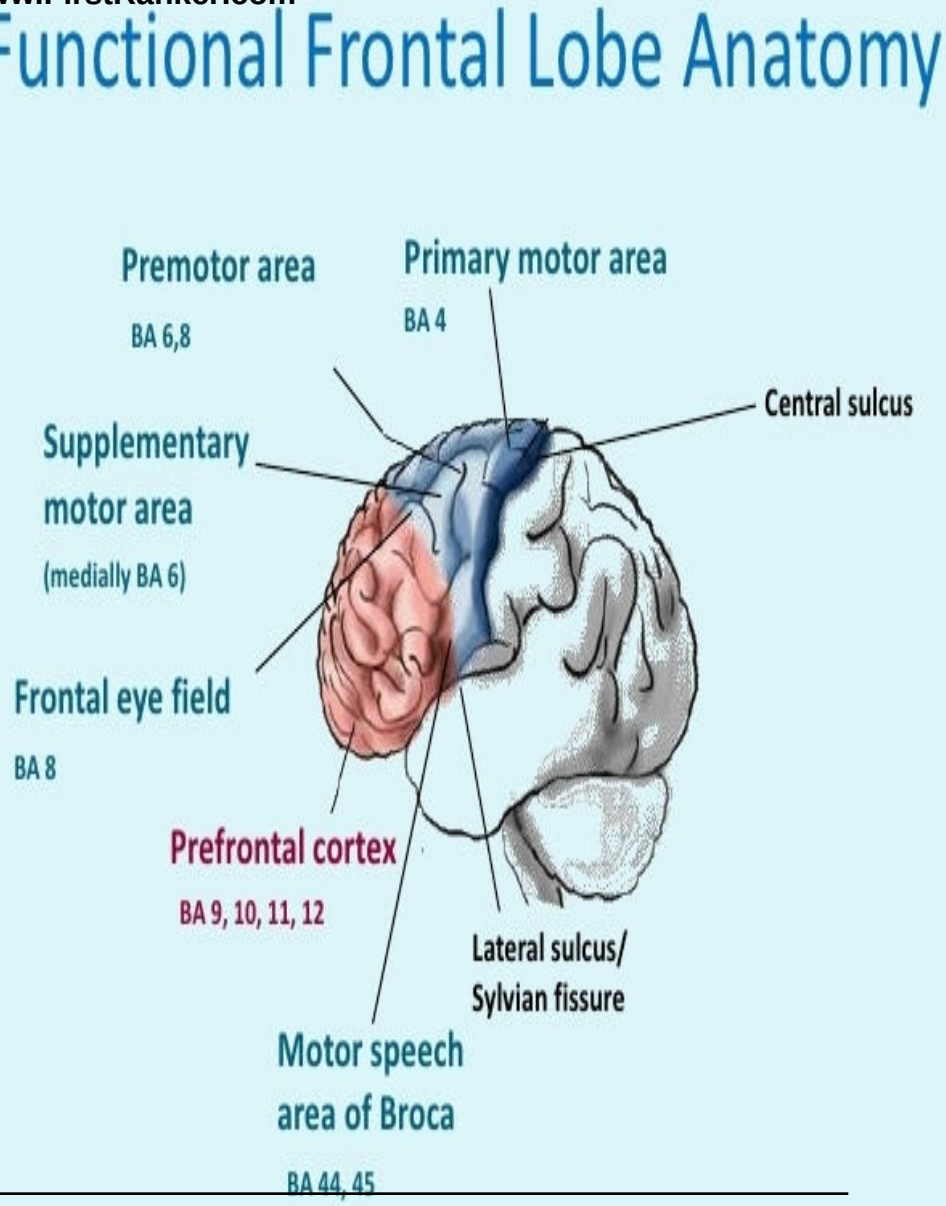
Inferior division of MCA

Sylvian fissure

- If there is occlusion to Superior division of MCA, because it supplies the important areas in frontal lobe, we can look for the following lesions:
- Weakness of arms and legs of the opposite side (hemiplegia)- 30 % of UMN fibres come from area 4 or motor area, rest 30 percent originate from pre and supplementary motor areas- area 6 mostly.
- Affliction to frontal eye field (area 8) results in loss of saccadic movements.
- In a frontal lobe lesion, patient looks towards side of lesion, the left frontal lobe helps you look to the right side and vice versa.

- Brocas areas-44 45- located in inferior frontal gyrus.
- “Dysarthric, effortful, telegraphic, sprase speech”
- Fluency, prosody are lost. Aggramatical speech.
- Repetition is also lost.
- Comprehension preserved.
- Reading and writing impaired.

Isolated lesion of pre motor cortex- rare, more spasticity is seen, primitive reflexes appear. Less paralysis.



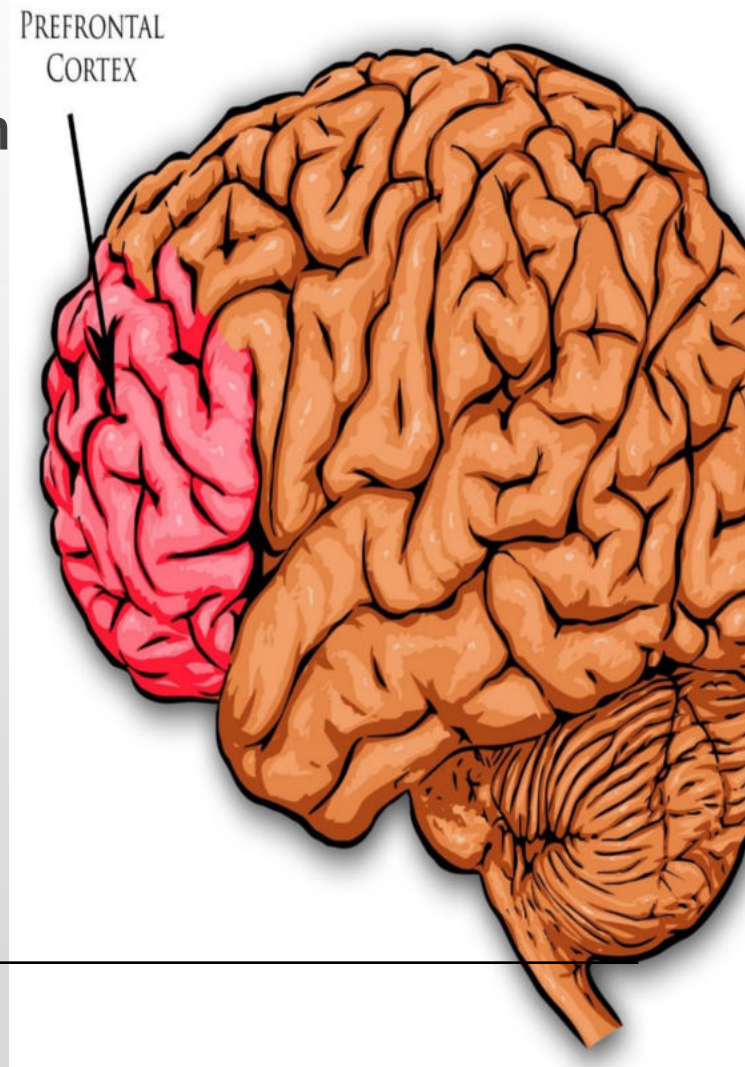
Pre frontal cortex- 9, 10, 11- “Makes you, you.”

Deals with our personality, creativity, insight, judgement, attention intelligence, motivation, social behaviour, language and problem solving capacity.

Also has a role in retrieval of memory (What did you have for breakfast?)

Lesions can thus affect all these areas, person may exhibit unacceptable behaviour, personality disorders.

PFC is also connected to caudate nucleus and putamen- hence extrapyramidal features can spring up.

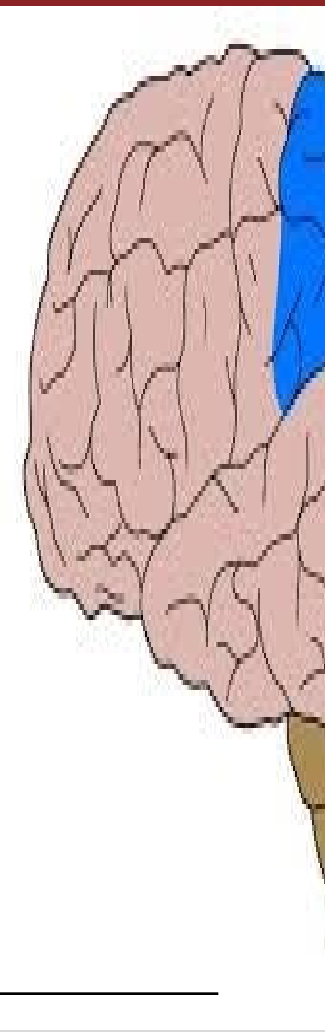


Medial surface of frontal cortex- Supplied by A2 branch of ACA

- Important areas: Paracentral lobule
- A lesion to this area- urinary incontinence
- Anterior Cingulate gyrus- above cingulate sulcus
- It has autonomic and limbic connections-lesions can result in emotional, autonomic and endocrine defects.
- B/L lesion- Abulia/ Akinetic mutism
- If there is a milder version of B/L frontal lobe lesion- gait apraxia or iginition foot.

PARIETAL LOBE- Superior division of M2 supplies it

- Post central gyrus- areas 312- somatosensory cortex- takes care of cortical sensations- Stereognosis, Tactile localisation, 2 point discrimination, Graphasthesia,
- Remember that pain and fine touch are carried by anterior spinothalamic tract, pressure and crude touch by lateral spinothalamic tract.
- A lesion here can be due to occlusion of the Superior segment of MCA
- Mild motor weakness can be seen here too. This is because 40 % of UMN fibres originate from area 312.
- Cortical sensory loss



- Homonymous inferior Quadrantanopia (Pie on the floor)
- Apraxia and loss of Optokinetic nystagmus.
- Supramarginal gyrus: lesion causes apraxia. Can be ideational or idiomotor.
- Left lobe is the dominant one.
- Gerstmann syndrome- seen in lesions to Angular gyrus:
- Loss of Rt and Lt discrimination
- Acalculia
- Agraphia with Alexia
- Finger agnosia

Dominant Lobe

- Lesions to Rt Parietal lobe:

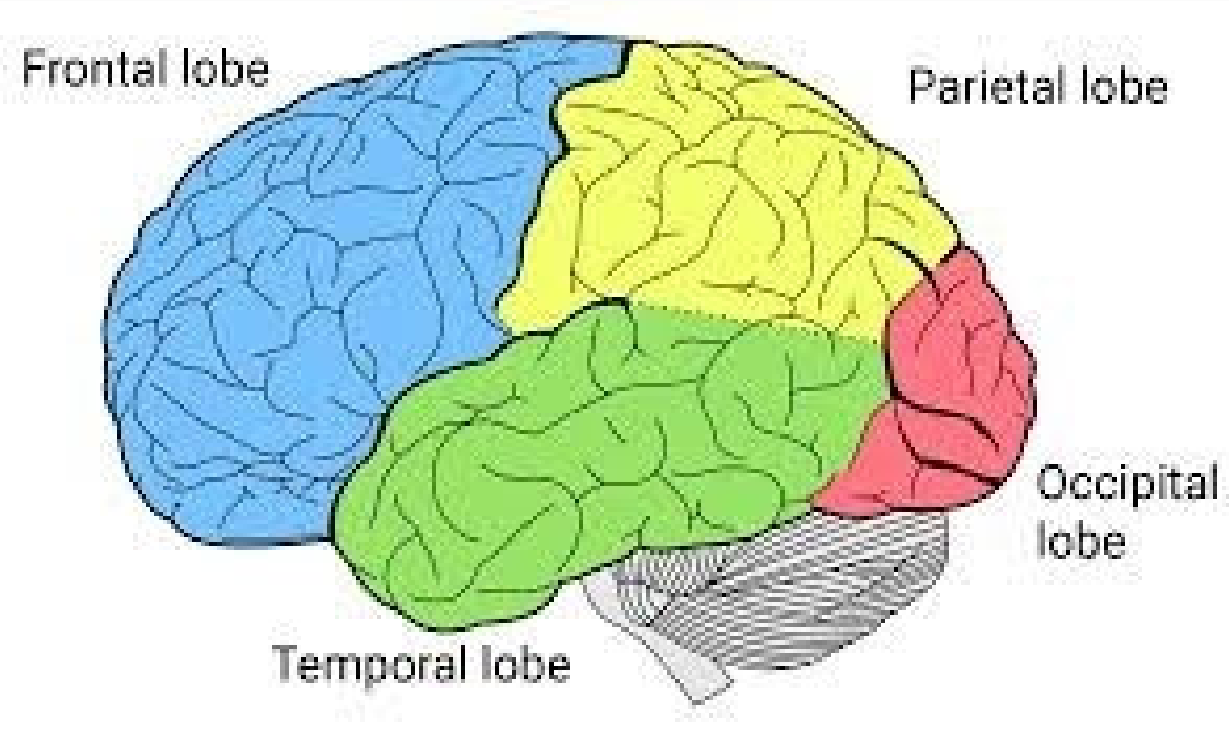
Visuospatial inattention

Hemispatial neglect or anosagnosia

Constructional apraxia

Dressing apraxia

TEMPORAL LOBE- Supplies by Inferior division of MCA



AREAS : WERNICKES AREA 22
PRIMARY AUDITORY AREAS 41 AND 42
SECONDARY AUDITORY AREA 22
VISUAL ASSOCIATION CORTEX- AREAS 21 AND 22

Hearing loss occurs only if there is B/L lesion.
Lesion affecting Wernickes area- Wernickes Aphasia- fluent sensory aphasia.
Patient manufactures his own words.
Cannot register new events in memory.

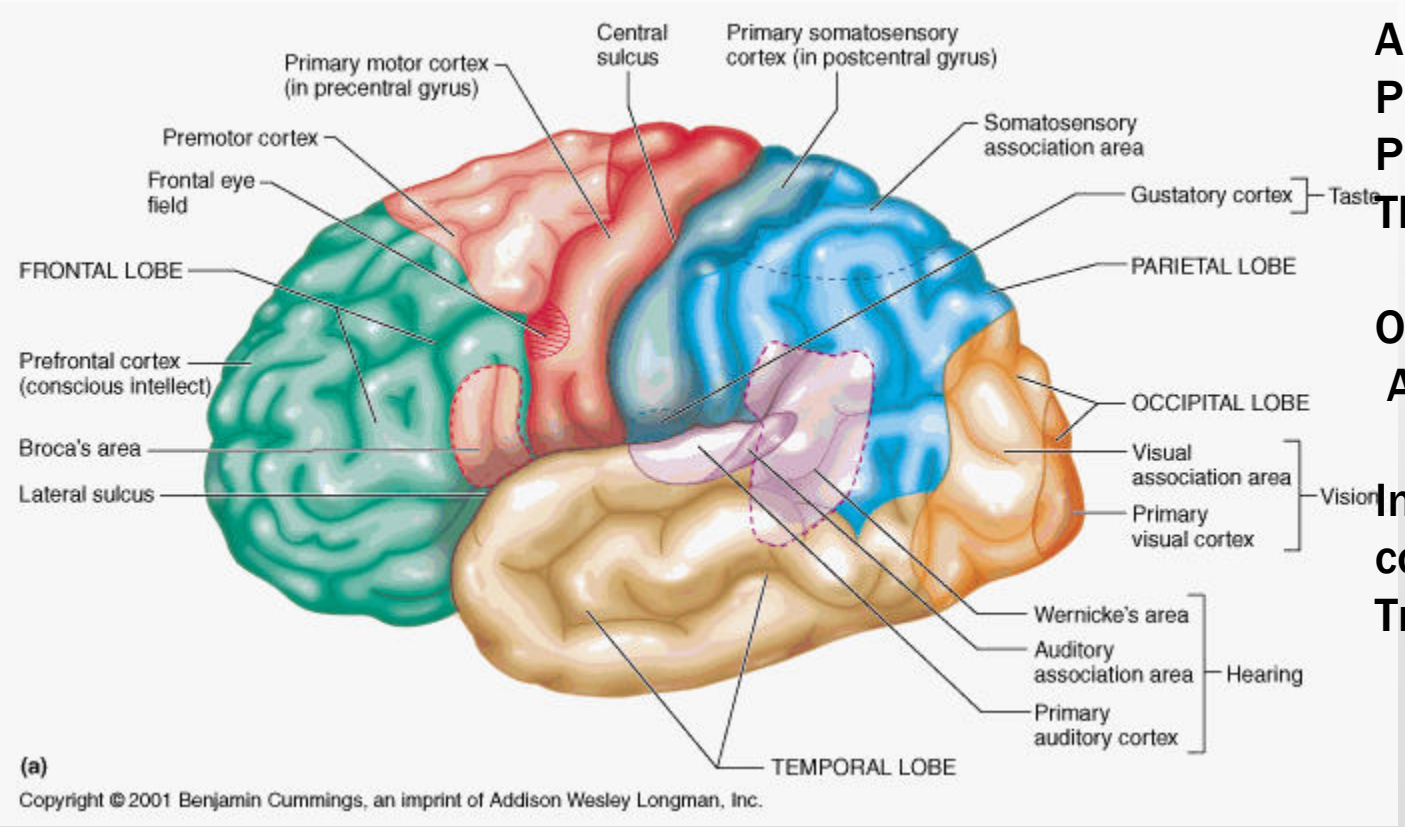
Lesion to Secondary auditory area: Auditory agnosia
Lesion to Visual association cortex: Visual agnosia

- Superior homonymous quadrantanopia. (Pie on the sky)
- Limbic cortex also forms a major part- mainly deals with our fear, aggression, feeding, sexual desire, goal directed behaviour.
- Consists of Amygdala and Hypothalamus.
- B/L Amygdala lesion- Kluver Bucy Syndrome.
- Remember that the temporal lobe does not have dominant or non dominant.
- So lesions can produce- Wernickes aphasia, audiotory and visual agnosia.
- Auditory, olfactory and gustatory hallucinations are also seen.

ALSO REMEMBER

- Medial temporal lobe is supplied by P2, a division of Posterior cerebral artery. P2 arises distal to PCOM.
- When Medial temporal lobe is affected: visual agnosia, memory loss (hippocampus is affected)
- P2 supplies occipital cortex as well.

OCCIPITAL LOBE Supplied by PCA- P2 division to be specific



AREAS
PRIMARY VISUAL CORTEX: 17
PERI AND PARASTRIATE CORTEX: 18. 19
They deal with ocular memory- fixation of size, shape and

Occipital cortex lesion can give rise to homonymous hemia
An optic tract lesion gives rise to the same.

In occipital cortex lesion, pupillary reflex is preserved. It is complete loss.
Tract lesion- reflex is affected, non congruous loss.

- Bilateral occipital cortex lesions-
- ANTON SYNDROME:
- SIMULTAGNOSIA (MISSING THE FOREST FOR TREES)
- OPTIC ATAXIA
- OCCULOMOTOR APRAXIA

BALINT SYNDROME

Cortical blindness, preserved light reflex, visual acuity impaired.

The patient denies blindness.

- Agnosias:
- Colour
- Prosopagnosia- inability to recognise faces.