

NeuroTrace Academy Study Guide

Category: Domain I - Neuroanatomy & EEG Localization

Topic: Brain Structures & Functions

Style: Anatomical, function-based, clinical correlation

1. Core Principle

Brain Structure = Function = Clinical Correlation

Understanding brain anatomy enables:

- Accurate EEG localization
- Clinical correlation with symptoms
- Recognition of lesion effects
- Proper interpretation of findings

2. External Structures: Cerebrum & Lobes

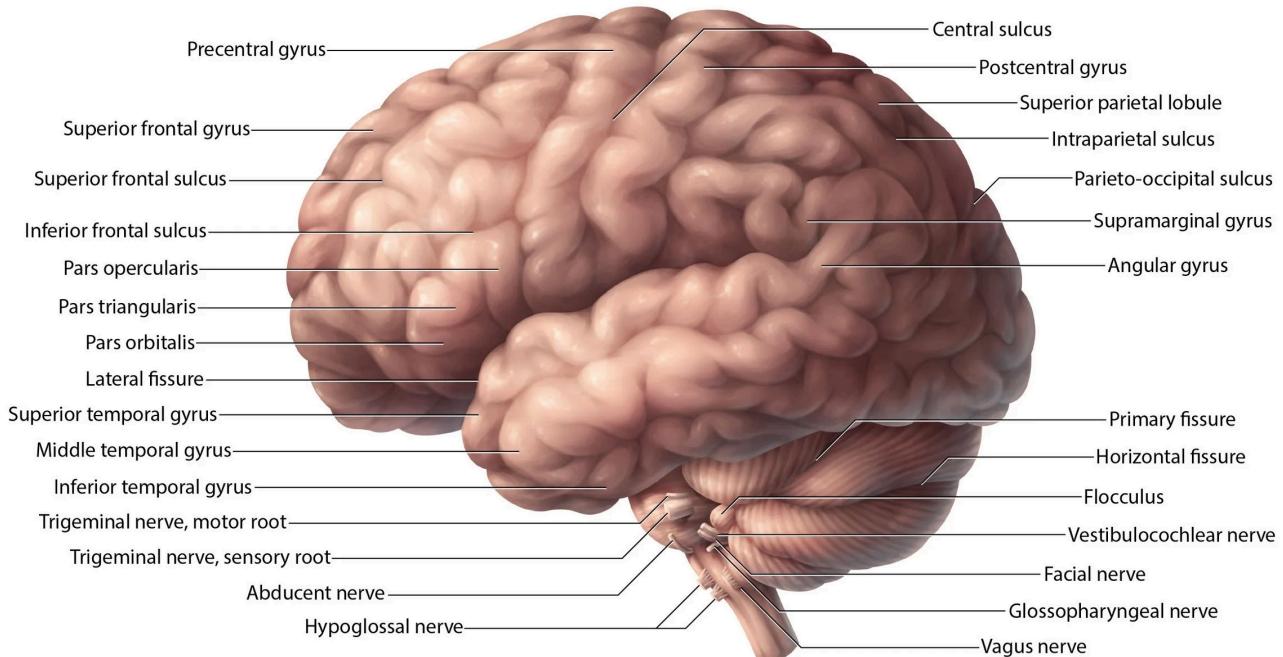


Figure 1: Lateral view of the brain.

Figure 1: Lateral view of the brain showing major gyri, sulci, lobes, and cranial nerves. Key landmarks include the precentral gyrus (motor cortex), postcentral gyrus (sensory cortex), central sulcus, lateral fissure (Sylvian fissure), and the four major lobes.

Cerebrum (Cerebral Cortex)

Location: Largest part of the brain, outermost layer

Function:

- Higher cognitive functions
- Voluntary movement
- Sensory processing
- Language and memory
- Consciousness

EEG Relevance:

- EEG primarily records cortical activity
 - Different lobes show different patterns
 - Lesions cause focal slowing or epileptiform discharges
-

An Introduction to Brain Structures

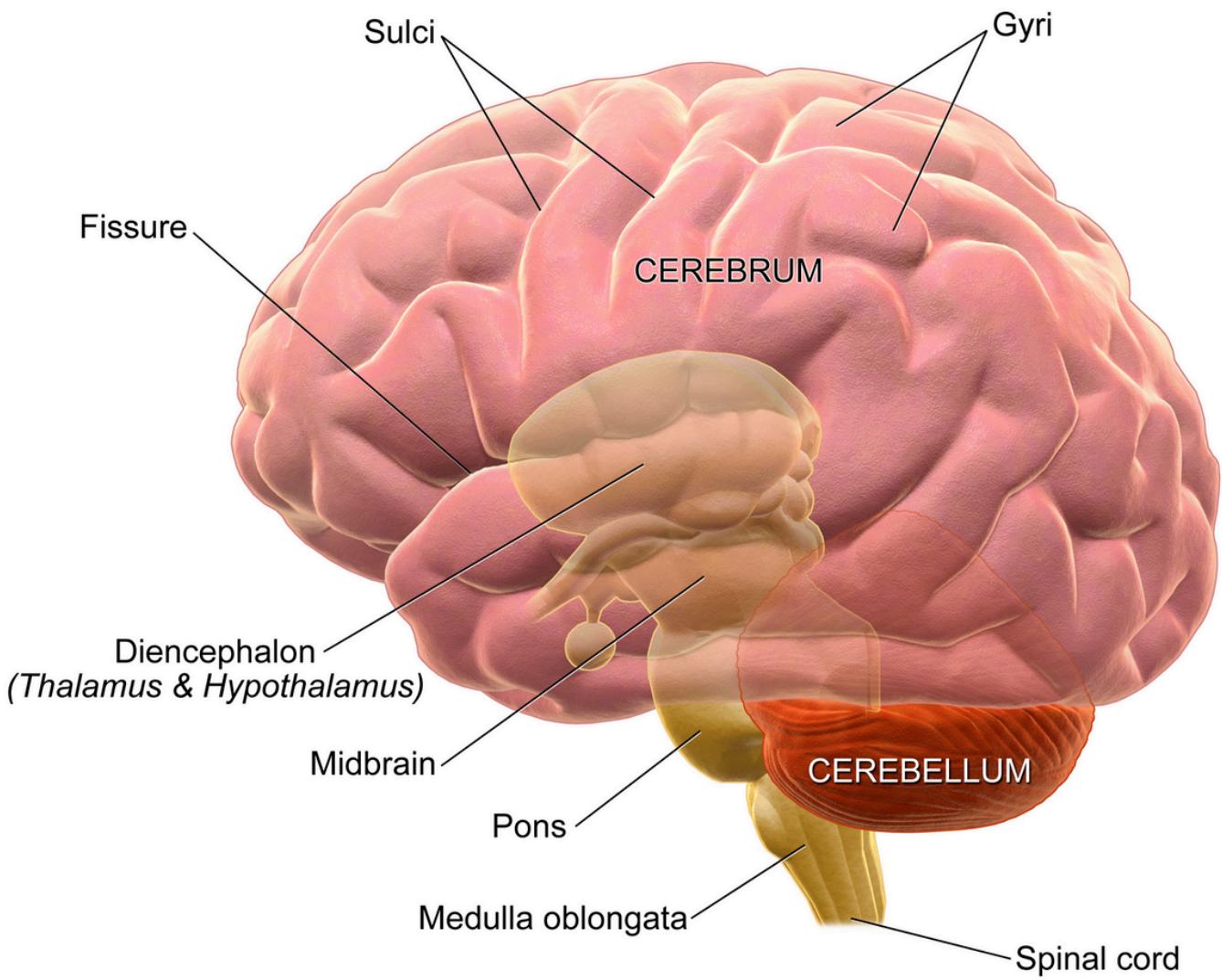


Figure 2: Lateral view of the brain with color-coded gyri. Red highlights the precentral gyrus (primary motor cortex), blue highlights the postcentral gyrus (primary sensory cortex). The central sulcus separates these two critical functional areas.

Frontal Lobe

Location: Front of the brain (anterior to central sulcus)

Key Structures:

- Precentral gyrus (primary motor cortex, Brodmann area 4) - See red area in Figure 2
- Broca area (areas 44, 45) - language production
- Prefrontal cortex - executive function

Functions:

- **Motor control:** Voluntary movement (contralateral body)
- **Language production:** Broca area (dominant hemisphere)

- **Executive function:** Planning, decision-making, judgment
- **Personality and behavior:** Social behavior, inhibition
- **Working memory**

Clinical Correlations:

- **Lesions cause:**
 - Contralateral weakness (motor cortex)
 - Broca aphasia (non-fluent speech, preserved comprehension)
 - Personality changes, disinhibition, poor judgment
 - Executive dysfunction
- **EEG:** Focal slowing or epileptiform discharges over frontal regions
- **Frontal lobe seizures:** Hypermotor behavior, brief, often nocturnal

EEG Localization:

- Fp1, Fp2, F3, F4, F7, F8 electrodes
 - Motor cortex: C3, C4 (precentral gyrus)
-

Parietal Lobe

Location: Behind frontal lobe (posterior to central sulcus, anterior to occipital lobe)

Key Structures:

- Postcentral gyrus (primary sensory cortex, Brodmann areas 3, 1, 2) - *See blue area in Figure 2*
- Superior parietal lobule
- Inferior parietal lobule

Functions:

- **Somatosensory processing:** Touch, pressure, temperature, proprioception
- **Spatial awareness:** Understanding spatial relationships
- **Sensory integration:** Combining different sensory inputs
- **Calculation and reading:** Angular gyrus (dominant hemisphere)

Clinical Correlations:

- **Lesions cause:**
 - Contralateral sensory loss (sensory cortex)
 - Hemispatial neglect (right parietal - left neglect)
 - Gerstmann syndrome (left parietal: acalculia, agraphia, finger agnosia, left-right confusion)
 - Apraxia (inability to perform learned movements)
- **EEG:** Focal slowing or epileptiform discharges over parietal regions

EEG Localization:

- P3, P4, Pz electrodes
 - Sensory cortex: C3, C4 (postcentral gyrus)
-

Temporal Lobe

Location: Below frontal and parietal lobes, on the sides of the brain

Key Structures:

- Superior temporal gyrus (auditory cortex, Brodmann area 41, 42)
- Wernicke area (area 22) - language comprehension

- Hippocampus (medial temporal) - memory formation
- Amygdala (medial temporal) - emotion

Functions:

- **Auditory processing:** Hearing and sound interpretation
- **Language comprehension:** Wernicke area (dominant hemisphere)
- **Memory formation:** Hippocampus (declarative memory)
- **Emotion:** Amygdala (fear, aggression)
- **Visual recognition:** Inferior temporal cortex

Clinical Correlations:

- **Lesions cause:**
 - Wernicke aphasia (fluent speech with poor comprehension)
 - Memory impairment (hippocampal damage)
 - Auditory processing deficits
 - Visual agnosia (inability to recognize objects)
- **EEG:** Temporal spikes/sharp waves (most common focal epilepsy location)
- **Temporal lobe seizures:** Aura (déjà vu, fear, epigastric rising), automatisms, postictal confusion

EEG Localization:

- T3, T4, T5, T6, T7, T8 electrodes
- Sphenoidal electrodes (for medial temporal foci)

Occipital Lobe

Location: Back of the brain (posterior)

Key Structures:

- Primary visual cortex (Brodmann area 17, calcarine sulcus)
- Visual association areas (areas 18, 19)

Functions:

- **Visual processing:** Receives and processes visual information
- **Visual recognition:** Object and face recognition
- **Color and motion perception**

Clinical Correlations:

- **Lesions cause:**
 - Contralateral homonymous hemianopia (visual field defect)
 - Cortical blindness (bilateral lesions)
 - Visual agnosia (inability to recognize objects despite normal vision)
- **EEG:** Focal slowing or epileptiform discharges over occipital regions
- **Occipital lobe seizures:** Visual hallucinations, visual field defects

EEG Localization:

- O1, O2, Oz electrodes
- Visual cortex: Calcarine sulcus (medial occipital)

Central Sulcus

Location: Separates frontal and parietal lobes

Function:

- Anatomical landmark
- Separates motor cortex (frontal) from sensory cortex (parietal)

Clinical Relevance:

- Lesions anterior to central sulcus → motor deficits
 - Lesions posterior to central sulcus → sensory deficits
-

3. Internal Structures: Deep Brain

Limbic Lobe

Location: Deep within the cerebrum, curved structure along inner surface

Components:

- Hippocampus
- Amygdala
- Cingulate gyrus
- Fornix
- Mammillary bodies

Functions:

- **Emotion:** Processing and regulation
- **Memory:** Formation and consolidation (hippocampus)
- **Learning:** Associative learning
- **Motivation:** Drive and reward

Clinical Correlations:

- **Lesions cause:**
 - Memory impairment (hippocampal damage)
 - Emotional dysregulation
 - Kluver-Bucy syndrome (bilateral temporal/limbic damage: placidity, hyperorality, hypersexuality)
 - **EEG:** Medial temporal spikes (temporal lobe epilepsy)
-

Corpus Callosum

Location: Large C-shaped band of nerve fibers connecting the two cerebral hemispheres

Function:

- **Interhemispheric communication:** Connects left and right hemispheres
- **Information transfer:** Allows coordination between hemispheres

Clinical Correlations:

- **Lesions cause:**
 - Callosal disconnection syndromes
 - Alien hand phenomenon (inability to name objects in left hand)
 - Split-brain syndrome (if completely severed)
 - **EEG:** May show asymmetric patterns if disconnected
-

Thalamus

Location: Large oval structure deep within the brain, below corpus callosum

Function:

- **Sensory relay:** Receives sensory information and relays to cortex
- **Motor relay:** Part of motor circuits
- **Consciousness:** Part of consciousness networks
- **Memory:** Part of memory circuits

Key Nuclei:

- **Ventral posterior (VP) nucleus:** Somatosensory relay (medial lemniscus, spinothalamic tract)
- **Lateral geniculate nucleus (LGN):** Visual relay
- **Medial geniculate nucleus (MGN):** Auditory relay
- **Ventral lateral (VL) nucleus:** Motor relay

Clinical Correlations:

- **Lesions cause:**
 - Contralateral sensory loss (VP nucleus)
 - Thalamic pain syndrome (Dejerine-Roussy) - severe, intractable pain
 - Memory impairment
 - Altered consciousness
- **EEG:** May show diffuse slowing with thalamic lesions

Hypothalamus

Location: Small structure directly below thalamus

Function:

- **Autonomic control:** Regulates body temperature, hunger, thirst
- **Endocrine control:** Controls pituitary gland
- **Circadian rhythms:** Sleep-wake cycle
- **Emotion:** Part of emotional processing

Clinical Correlations:

- **Lesions cause:**
 - Temperature dysregulation
 - Endocrine dysfunction
 - Sleep disorders
 - Behavioral changes
- **Hypothalamic hamartoma:** Precocious puberty, gelastic seizures

Pituitary Gland

Location: Small spherical gland hanging below hypothalamus

Function:

- **Endocrine control:** Master gland controlling other endocrine glands
- **Hormone production:** Growth hormone, ACTH, TSH, FSH, LH, prolactin

Clinical Correlations:

- **Tumors cause:**
 - Visual field defects (compression of optic chiasm)
 - Endocrine dysfunction
 - Headaches
-

Optic Chiasm

Location: Cross-shaped structure at bottom front of hypothalamus

Function:

- **Visual pathway:** Where optic nerves cross
- **Visual field processing:** Nasal fibers cross, temporal fibers don't

Clinical Correlations:

- **Lesions cause:**
 - Bitemporal hemianopia (pituitary tumors compressing chiasm)
 - Visual field defects
-

Mammillary Body

Location: Small spherical structure behind pituitary gland

Function:

- **Memory:** Part of memory circuits (Papez circuit)
- **Limbic system:** Connected to hippocampus via fornix

Clinical Correlations:

- **Lesions cause:**
 - Memory impairment (Wernicke-Korsakoff syndrome - thiamine deficiency)
 - **EEG:** May show memory-related changes
-

Pineal Gland

Location: Small pea-sized gland behind thalamus, above cerebellum

Function:

- **Melatonin production:** Regulates sleep-wake cycle
- **Circadian rhythms:** Responds to light-dark cycles

Clinical Correlations:

- **Tumors (pinealomas):**
 - May cause obstructive hydrocephalus
 - Parinaud syndrome (upward gaze palsy)
-

4. Brainstem Structures

Pons

Location: Large bulbous structure below thalamus, behind mammillary body

Function:

- **Relay center:** Connects cerebrum to cerebellum and spinal cord
- **Cranial nerves:** Houses CN V (trigeminal), CN VI (abducens), CN VII (facial), CN VIII (vestibulocochlear) nuclei
- **Sleep:** Part of sleep-wake regulation (REM sleep)
- **Respiration:** Part of respiratory control

Clinical Correlations:

- **Lesions cause:**
 - Millard-Gubler syndrome (ventral pons: CN VI/VII palsy + hemiparesis)
 - Locked-in syndrome (bilateral ventral pons: quadriplegia, preserved consciousness)
 - Respiratory dysfunction
- **EEG:** May show slowing or abnormal patterns with pontine lesions

Medulla Oblongata

Location: Elongated structure extending downward from pons, forms lower brainstem

Function:

- **Vital functions:** Controls breathing, heart rate, blood pressure
- **Cranial nerves:** Houses CN IX (glossopharyngeal), CN X (vagus), CN XI (accessory), CN XII (hypoglossal) nuclei
- **Reflexes:** Swallowing, vomiting, coughing

Clinical Correlations:

- **Lesions cause:**
 - Wallenberg syndrome (lateral medullary: PICA occlusion)
 - Respiratory failure
 - Cardiac arrhythmias
 - Swallowing difficulties
- **EEG:** May show severe abnormalities or isoelectric pattern with severe medullary dysfunction

5. Cerebellum

Location: Large highly convoluted structure at back and bottom of brain, beneath occipital lobe

Internal Structure:

- Arbor vitae (tree-like white matter pattern)

Functions:

- **Motor coordination:** Smooth, coordinated movements
- **Balance:** Posture and equilibrium
- **Motor learning:** Learning new motor skills
- **Cognitive functions:** Some role in cognition

Key Regions:

- **Vermis (midline):** Truncal ataxia, gait instability
- **Hemispheres:** Ipsilateral limb ataxia, intention tremor

Clinical Correlations:

- **Lesions cause:**
 - Ataxia (uncoordinated movements)
 - Dysmetria (past-pointing)

- Dysdiadochokinesia (impaired rapid alternating movements)
 - Intention tremor
 - Nystagmus
- **EEG:** Usually normal (cerebellum doesn't generate scalp EEG signals)

Important Note:

- Cerebellar lesions are **ipsilateral** (unlike cerebral lesions which are contralateral)
- Due to double decussation of pathways

6. Blood Supply of the Brain: Comprehensive Clinical Map

Master Summary: Anterior circulation strokes affect movement and language; posterior circulation strokes affect vision, balance, eye movements, and consciousness.

Circle of Willis

Anterior cerebral artery

Middle cerebral artery

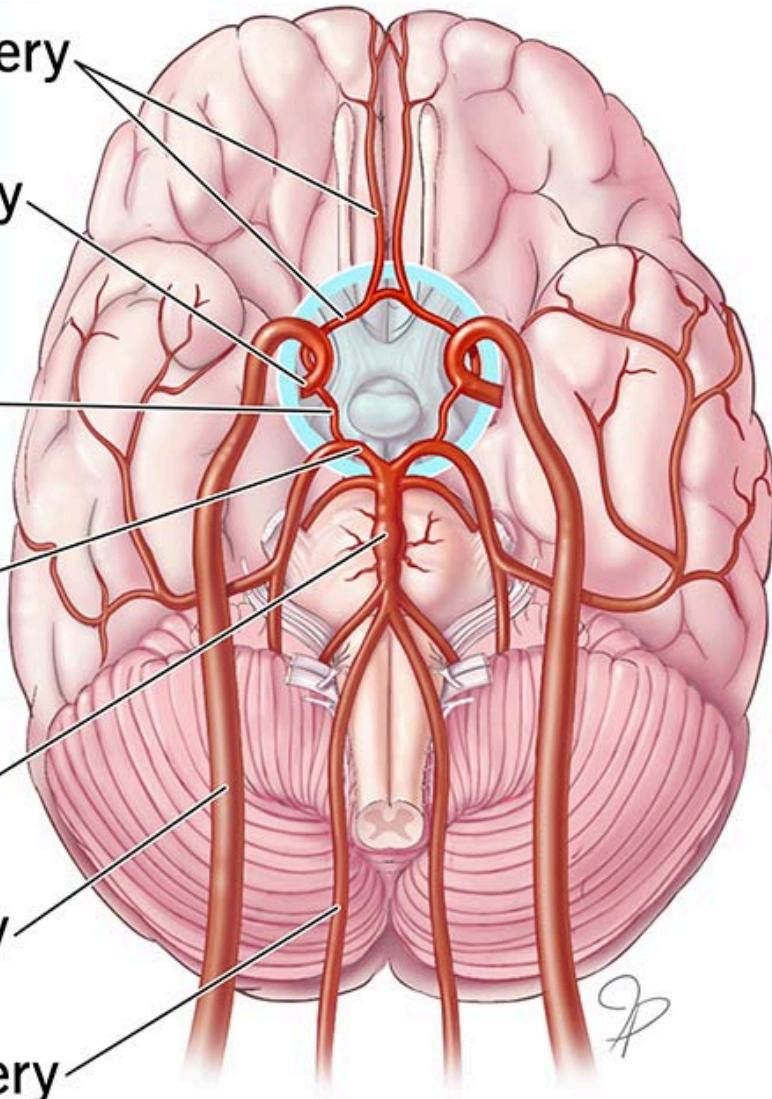
Posterior
communicating
artery

Posterior
cerebral artery

Basilar artery

Internal carotid artery

Vertebral artery



Bottom view of brain

Figure 3: Circle of Willis from inferior view showing the complete arterial anastomotic ring at the base of the brain. This critical network connects the anterior (carotid) and posterior (vertebrobasilar) circulations, providing collateral blood flow.

Circle of Willis

- Vessels comprising the circle of Willis include:

- **anterior circulation**

- left and right ICA
- horizontal (A1) segments of the left and right anterior cerebral arteries (ACA)
- single anterior communicating artery (ACOM)

- **posterior circulation**

- left and right posterior communicating arteries (PCOM)
- horizontal (P1) segments of left and right posterior cerebral arteries (PCA)
- single basilar artery (tip)

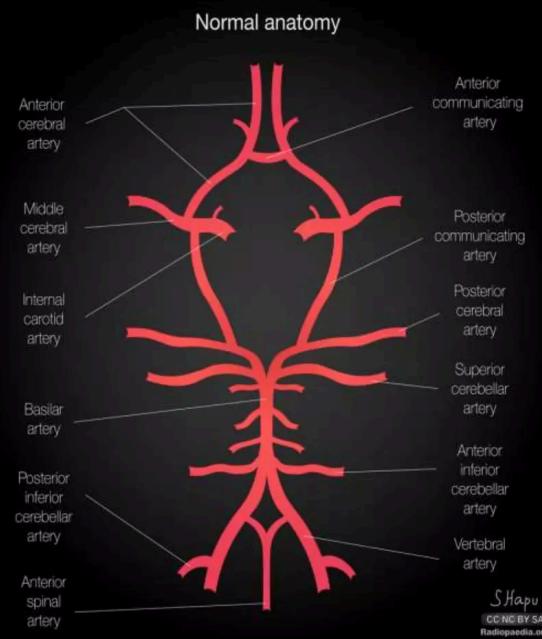
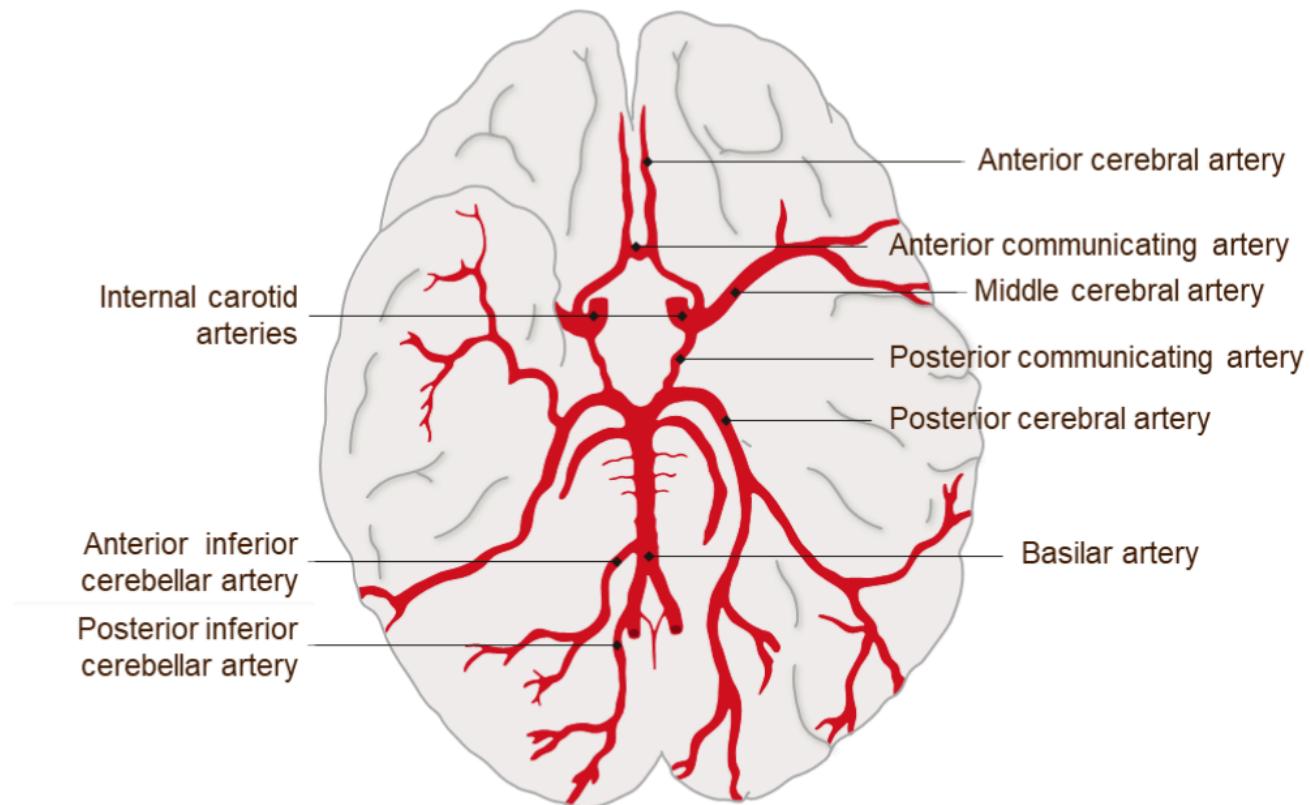


Figure 4: Educational diagram showing the components of the Circle of Willis. Anterior circulation (ICA, ACA, ACOM) and posterior circulation (PCOM, PCA, Basilar) are clearly labeled. The diagram also shows cerebellar arteries (PICA, AICA, SCA) and vertebral arteries.

Arteries of the brain



Konan et al. StatPearls Publishing; 2023. Available at: <https://www.ncbi.nlm.nih.gov/books/NBK532297/>; Moos & Møller. Basic Neuroanatomy. 2003



Figure 5: Inferior view of the brain showing the complete arterial network. The Circle of Willis is visible centrally, with major branches extending to supply different brain regions. Cerebellar arteries (AICA, PICA) are also shown.

Two Main Systems

1. **Anterior Circulation → Internal Carotid Arteries**
2. **Posterior Circulation → Vertebral → Basilar Arteries**

They connect via the **Circle of Willis**, providing collateral flow.

● **Anterior Circulation (Internal Carotid System)**

Circle of Willis – Why It Matters

Location: Base of the brain, surrounding the optic chiasm and pituitary gland

Function: Anastomotic arterial ring connecting anterior and posterior circulations

Components:

Anterior Circulation:

- **Internal Carotid Arteries (ICA):** Left and right, enter cranial cavity and branch into:
 - **Anterior Cerebral Arteries (ACA):** A1 segments (horizontal/pre-communicating)
 - **Middle Cerebral Arteries (MCA):** Largest branches, supply lateral hemispheres
 - **Anterior Communicating Artery (ACOM):** Single artery connecting left and right ACAs

Posterior Circulation:

- **Vertebral Arteries:** Left and right, ascend through cervical spine, unite to form:
 - **Basilar Artery:** Single artery along ventral brainstem, gives rise to:
 - **Posterior Cerebral Arteries (PCA):** P1 segments (horizontal/pre-communicating)
- **Posterior Communicating Arteries (PCOM):** Left and right, connect ICA to PCA

Clinical Implications:

- Provides **collateral flow** if one artery is occluded
- Common site for **berry aneurysms**
- Rupture → **subarachnoid hemorrhage**

Classic Aneurysm Effects:

- **ACom aneurysm:** Frontal lobe symptoms, behavioral changes
- **PCom aneurysm:** CN III palsy (blown pupil, ptosis, "down and out" eye)

1 **Anterior Cerebral Artery (ACA)**

Course and Segments:

- **A1 (Pre-communicating):** Horizontal segment from ICA to ACOM
- **A2 (Post-communicating):** Ascends superiorly after ACOM
- **A3 (Precallosal):** Curves around genu of corpus callosum
- **A4 (Supracallosal):** Runs along superior surface of corpus callosum
- **A5 (Postcallosal):** Continues posteriorly along corpus callosum

Branches:

- **Callosomarginal artery:** Major branch from A2
- **Supracallosal branches:** Supply medial frontal and parietal cortex

Supplies:

- Medial frontal lobe
- Medial parietal lobe
- Leg/foot motor & sensory cortex
- Corpus callosum (anterior portion)
- Anterior cingulate cortex

Clinical Pathology (ACA Stroke):

- Contralateral **leg weakness** (leg > arm > face)
- Contralateral sensory loss (leg > arm)
- **Urinary incontinence** (medial frontal micturition center)
- **Abulia / personality change** (apathy, disinhibition, poor judgment)
- Gait disturbance
- Grasp reflex
- Transcortical motor aphasia (if dominant hemisphere, less common than MCA)

★ **EEG:** Focal slowing over medial frontal regions (Fp1, Fp2, Fz)

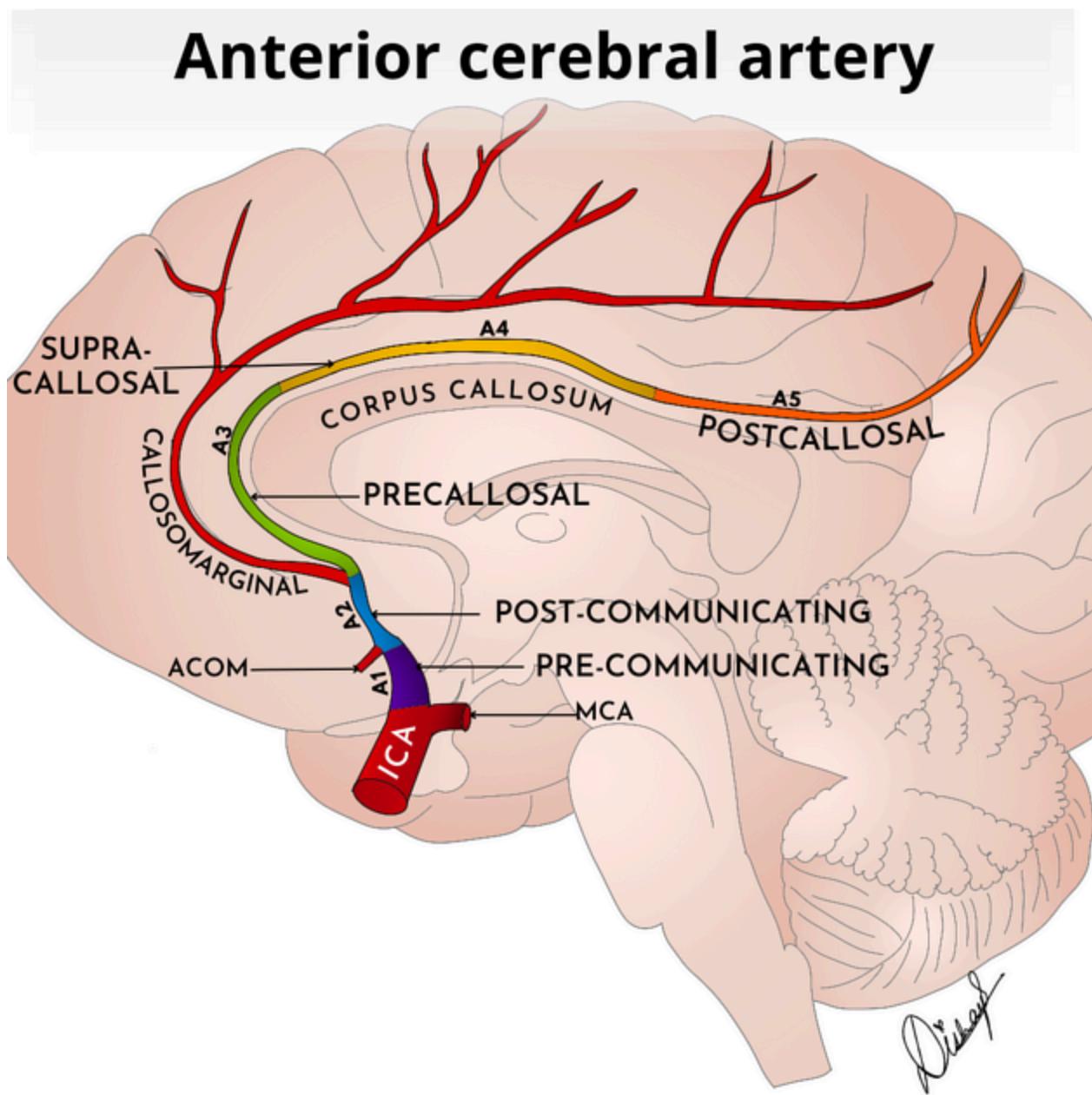


Figure 6:
Medial view of the brain showing the Anterior Cerebral Artery (ACA) and its segments (A1-A5). The ACA courses around the corpus callosum, with the corpus callosum shown in color-coded sections (precallosal in green, main body in yellow, postcallosal in orange). The callosom marginal artery and supracallosal branches are also visible.

2 Middle Cerebral Artery (MCA) — MOST COMMON STROKE

Course:

- Largest branch of ICA
- Extends laterally into lateral (Sylvian) fissure
- Branches over lateral surface of hemisphere

Supplies:

- Lateral frontal lobe (motor cortex for face/arm)
- Lateral parietal lobe (sensory cortex for face/arm)
- Lateral temporal lobe (Wernicke area, auditory cortex)
- Language cortex (dominant hemisphere)
- Insula
- Basal ganglia (lenticulostriate branches)

Clinical Pathology (MCA Stroke):

- Contralateral **face & arm weakness** (face/arm > leg)
- **Aphasia** (if left/dominant hemisphere):
 - **Broca aphasia** (if anterior MCA) - non-fluent speech, preserved comprehension
 - **Wernicke aphasia** (if posterior MCA) - fluent speech, poor comprehension
 - **Global aphasia** (if entire MCA) - both production and comprehension impaired
- **Hemispatial neglect** (if right/non-dominant hemisphere)
- **Gaze deviation toward lesion**
- **Homonymous hemianopia** (if posterior MCA affects optic radiations)
- **Sensory loss** (face/arm > leg) - contralateral

↗ **EEG:** Focal slowing over temporal-parietal regions (F3, F4, T3, T4, P3, P4)

3 Internal Carotid Artery (ICA)

Clinical Pathology:

- Combined ACA + MCA deficits
- **Amaurosis fugax** (retinal ischemia - transient monocular blindness)
- Large territorial infarction
- Watershed infarcts (border zone between ACA/MCA/PCA territories)

● Posterior Circulation (Vertebralbasilar System)

4 Posterior Cerebral Artery (PCA)

Course:

- Arises from basilar artery bifurcation (P1 segment)
- Receives PCOM connection (completes Circle of Willis)
- Curves around midbrain
- Supplies posterior brain

Supplies:

- Occipital lobe (primary visual cortex)
- Inferior temporal lobe

- Visual cortex
- Thalamus (via thalamoperforating branches)
- Midbrain (via perforating branches)
- Posterior corpus callosum

Clinical Pathology (PCA Stroke):

- Contralateral **homonymous hemianopia** (most common)
- **Visual hallucinations** (if affects visual association areas)
- **Memory impairment** (if affects hippocampus/thalamus)
- **Cortical blindness** (if bilateral)
- **Thalamic syndromes** (sensory loss, thalamic pain)
- **Visual agnosia** (if affects visual association areas)

↗ **EEG:** Posterior slowing, loss of PDR (posterior dominant rhythm)

5 Basilar Artery

Course:

- Formed by union of vertebral arteries
- Runs along ventral brainstem
- Bifurcates into PCAs

Supplies:

- Pons
- Brainstem nuclei
- Cerebellum (via AICA, SCA)
- Midbrain

Clinical Pathology (Basilar Stroke):

- **Diplopia** (double vision - cranial nerve palsies)
- **Dysarthria** (slurred speech)
- **Vertigo** (dizziness)
- **Ataxia** (uncoordinated movements)
- ↓ **Consciousness**
- Severe → **locked-in syndrome** (quadriplegia, preserved consciousness, only vertical eye movements)

↗ **EEG:** Diffuse slowing, reduced reactivity

6 Vertebral Arteries

Course:

- Left and right vertebral arteries
- Ascend through cervical spine
- Unite to form basilar artery

Clinical Pathology:

- **Lateral medullary (Wallenberg) syndrome** (PICA occlusion):
 - Vertigo, nystagmus
 - Dysphagia, hoarseness (CN IX, X)
 - Ipsilateral facial pain loss, contralateral body pain/temperature loss
 - Ipsilateral Horner syndrome

- Ataxia
-

Cerebellar Arteries

Posterior Inferior Cerebellar Artery (PICA):

- Branch of vertebral artery
- Supplies: Posterior and inferior cerebellum, medulla
- Occlusion → Wallenberg syndrome

Anterior Inferior Cerebellar Artery (AICA):

- Branch of basilar artery
- Supplies: Anterior and inferior cerebellum, pons
- Occlusion → Pontine signs + cerebellar ataxia

Superior Cerebellar Artery (SCA):

- Branch of basilar artery (near PCA origin)
 - Supplies: Superior cerebellum, midbrain
 - Occlusion → Midbrain signs + superior cerebellar ataxia
-

High-Yield Clinical Correlation Table

Artery	Key Deficit	Hallmark Sign	EEG Finding
ACA	Leg weakness	Incontinence	Medial frontal slowing (Fp1, Fp2, Fz)
MCA	Face/arm weakness	Aphasia	Lateral temporal-parietal slowing (F3, F4, T3, T4, P3, P4)
PCA	Visual loss	Hemianopia	Posterior slowing, loss of PDR (O1, O2, Oz)
Basilar	Brainstem signs	Diplopia	Diffuse slowing, reduced reactivity
Vertebral	Medullary signs	Vertigo	May show severe abnormalities

EEG-Specific Takeaway

Focal Slowing Patterns:

- **Focal slowing** → arterial territory ischemia
- **Posterior circulation strokes** → loss of PDR (posterior dominant rhythm)
- **Basilar compromise** → diffuse suppression / poor reactivity
- **Large MCA strokes** → focal attenuation or slowing

Acute vs Chronic:

- **Acute strokes:** Focal polymorphic delta slowing
- **Chronic strokes:** Focal slowing, may have epileptiform discharges
- **Watershed infarcts:** Bilateral slowing at border zones

Stroke vs Hemorrhage:

- **Ischemic stroke:** Focal slowing, may have periodic patterns
- **Hemorrhagic stroke:** More severe focal slowing, may have suppression

- **Subarachnoid hemorrhage:** Diffuse slowing, may have periodic lateralized epileptiform discharges (PLEDs)
-

7. EEG Electrode Localization

Standard 10-20 System Correlations

Frontal:

- Fp1, Fp2: Prefrontal cortex
- F3, F4: Dorsolateral frontal cortex
- F7, F8: Inferior frontal cortex (Broca area)

Central:

- C3, C4: Motor/sensory cortex (precentral/postcentral gyri)

Parietal:

- P3, P4, Pz: Parietal association cortex

Temporal:

- T3, T4: Middle temporal cortex
- T5, T6: Posterior temporal cortex (Wernicke area)
- T7, T8: Anterior temporal cortex

Occipital:

- O1, O2, Oz: Visual cortex (calcarine sulcus)
-

8. Clinical Pearls

Localization Principles

1. **Motor deficits:** Frontal lobe (precentral gyrus)
2. **Sensory deficits:** Parietal lobe (postcentral gyrus)
3. **Language deficits:** Frontal (Broca) or temporal (Wernicke) - dominant hemisphere
4. **Visual deficits:** Occipital lobe
5. **Memory deficits:** Temporal lobe (hippocampus)
6. **Balance/coordination:** Cerebellum
7. **Cranial nerve deficits:** Brainstem (nuclei locations)

EEG Correlation

- **Focal slowing:** Indicates cortical dysfunction in that region
 - **Focal spikes:** Indicates epileptogenic cortex
 - **Diffuse slowing:** Indicates diffuse cortical dysfunction or subcortical involvement
 - **Asymmetry:** May indicate structural lesion or dysfunction
-

9. Exam High-Yield Facts

1. **Broca area (left frontal):** Non-fluent aphasia
2. **Wernicke area (left temporal):** Fluent aphasia with poor comprehension
3. **Precentral gyrus:** Motor cortex (contralateral weakness)
4. **Postcentral gyrus:** Sensory cortex (contralateral sensory loss)
5. **Occipital lobe:** Visual cortex (contralateral hemianopia)

6. **Cerebellum:** Ipsilateral ataxia (unlike cerebrum which is contralateral)

7. **Thalamus:** Sensory relay, thalamic pain syndrome

8. **Brainstem:** Cranial nerve nuclei, vital functions

10. Summary Table

Structure	Location	Primary Function	Clinical Lesion Effect
Frontal Lobe	Anterior	Motor, language (Broca), executive function	Contralateral weakness, Broca aphasia, personality changes
Parietal Lobe	Superior	Sensory, spatial awareness	Contralateral sensory loss, hemispatial neglect
Temporal Lobe	Lateral	Auditory, language (Wernicke), memory	Wernicke aphasia, memory impairment, temporal lobe epilepsy
Occipital Lobe	Posterior	Visual processing	Contralateral hemianopia, cortical blindness
Thalamus	Deep	Sensory relay, consciousness	Thalamic pain syndrome, sensory loss, altered consciousness
Cerebellum	Posterior	Motor coordination, balance	Ipsilateral ataxia, dysmetria, nystagmus
Brainstem	Inferior	Cranial nerves, vital functions	Cranial nerve palsies, respiratory/cardiac dysfunction

Key Takeaway: Understanding brain anatomy and function is essential for accurate EEG interpretation and clinical correlation. Each structure has specific functions that, when lesioned, produce characteristic clinical and EEG findings.