

THE NERVOUS SYSTEM

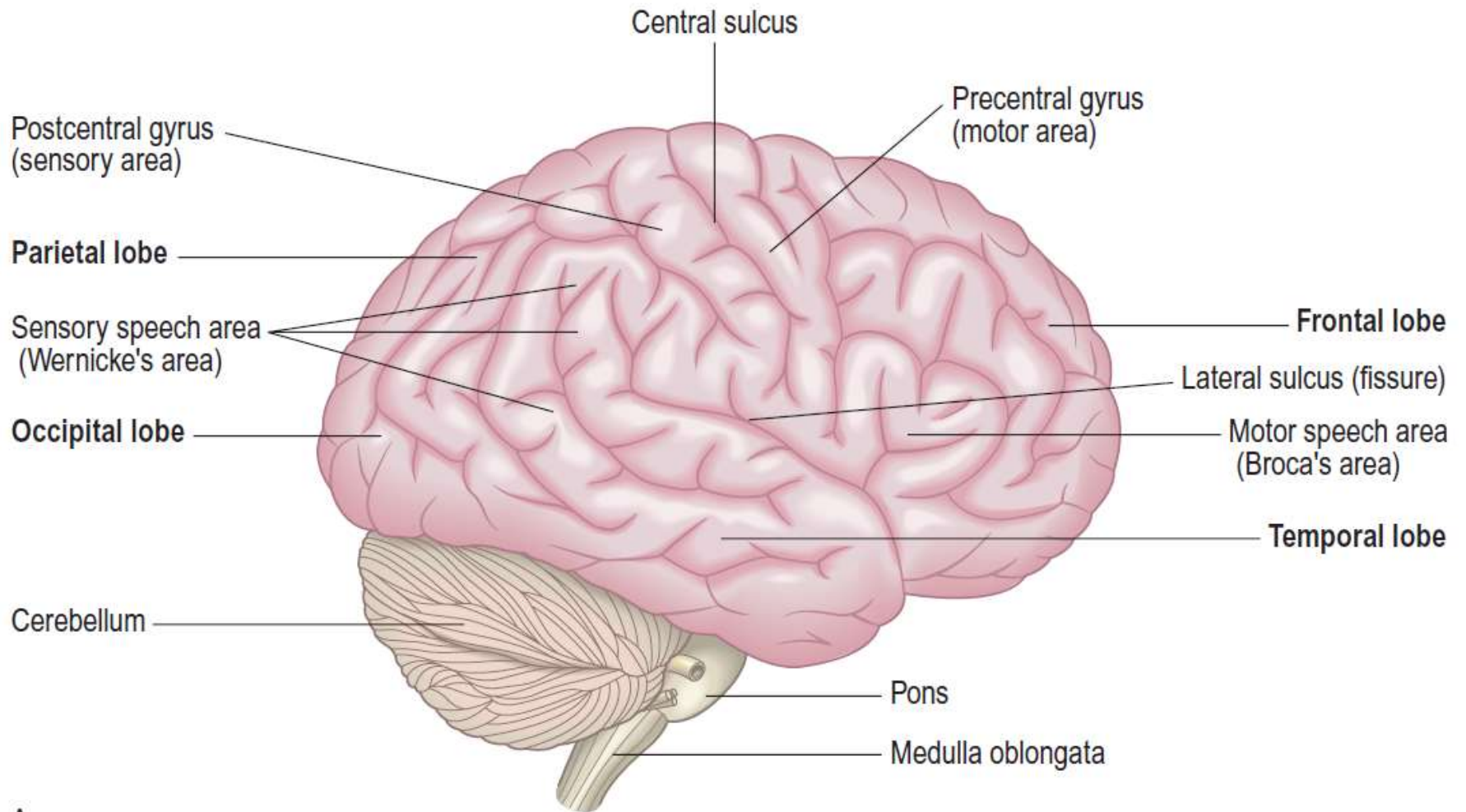
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ANATOMY AND PHYSIOLOGY

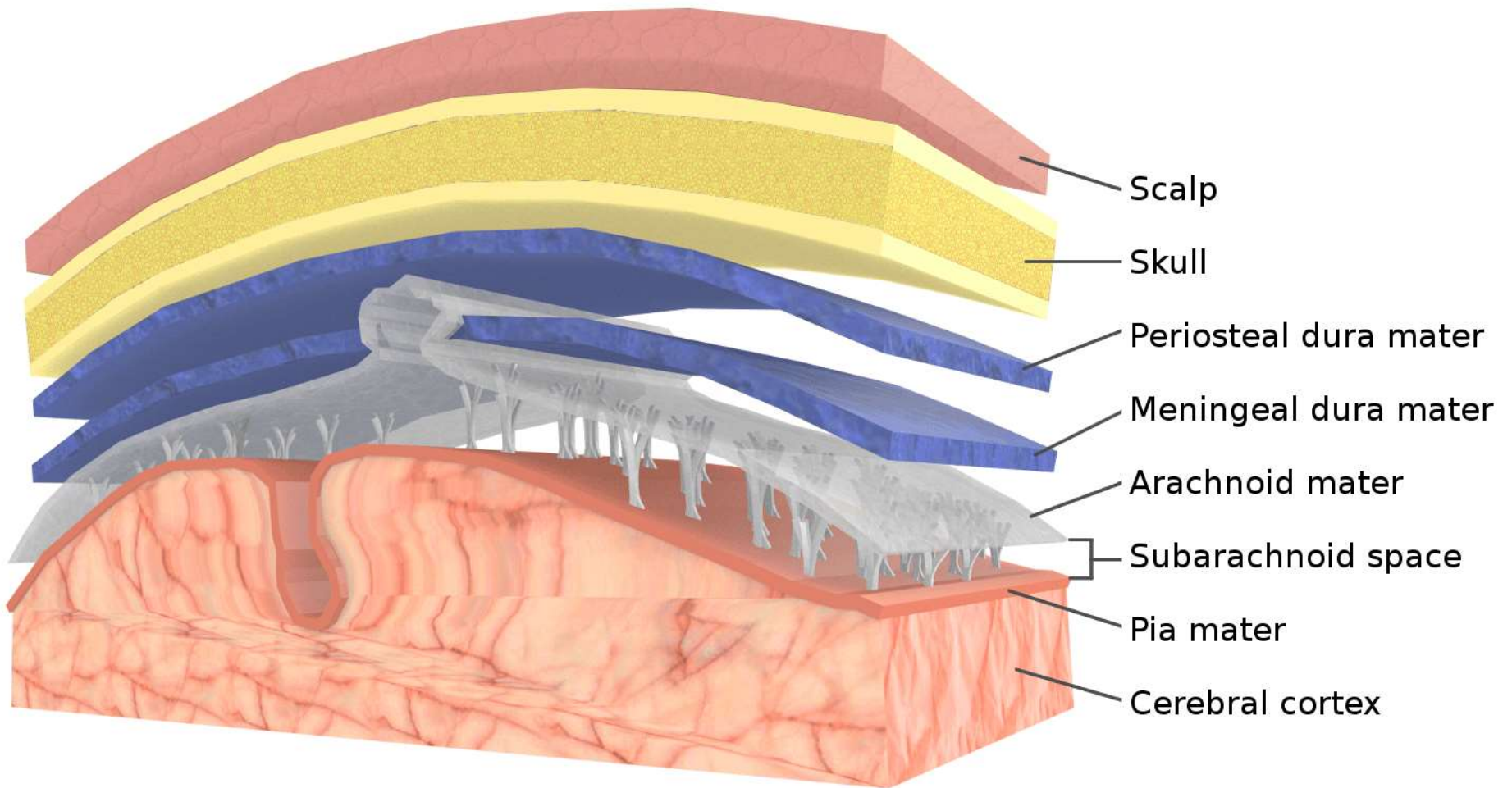
- CNS (Brain, Spinal Cord)
- PNS (Sensory Nerves, Motor Nerves, Autonomic Nerves)
- Functional unit is a Neuron (Dendrites, Cell body, Axon, Terminals)
- Supportive cells:
 - CNS: Oligodendrocytes, Microglia, Astrocyte, Ependymal cells.
 - PNS: Schwann cells, Satellite cells





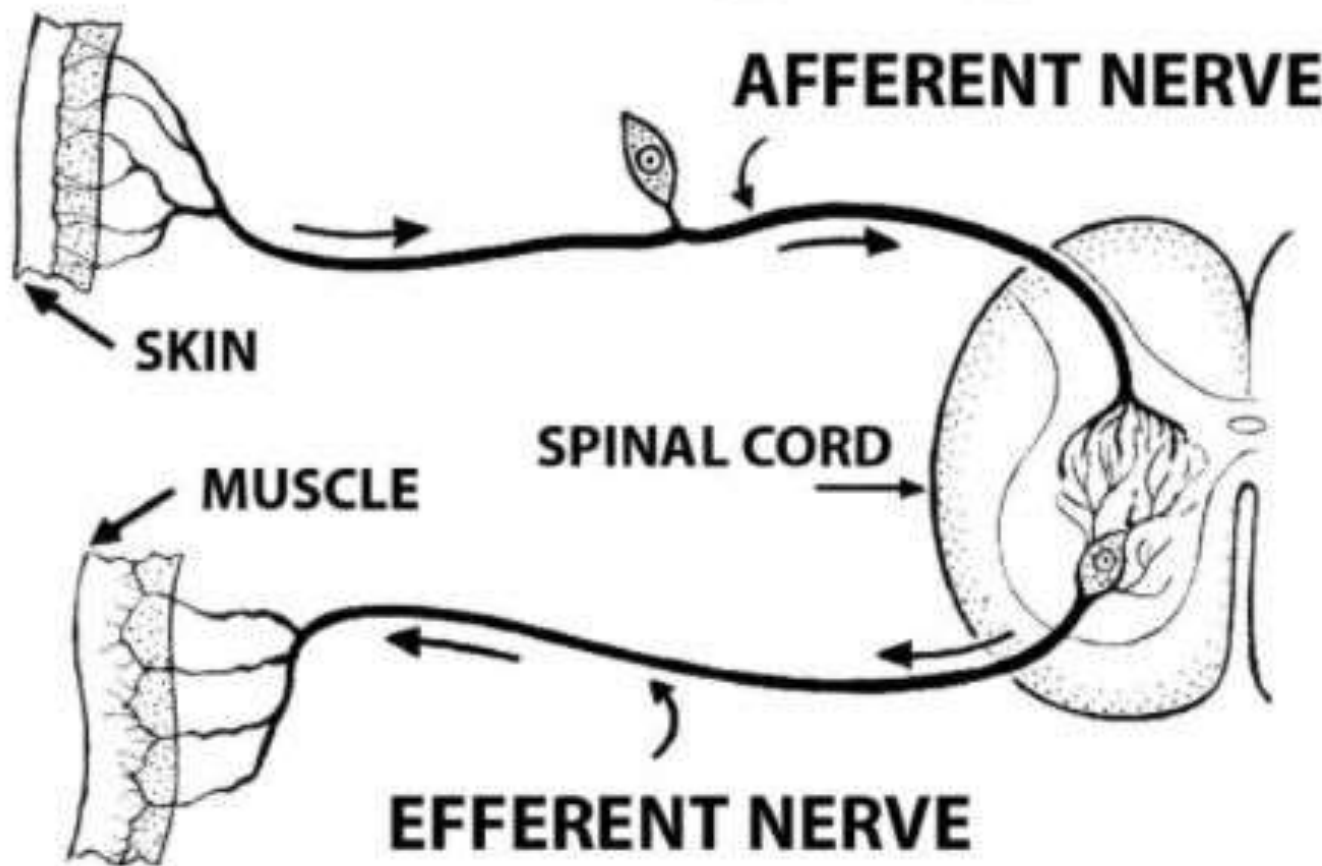
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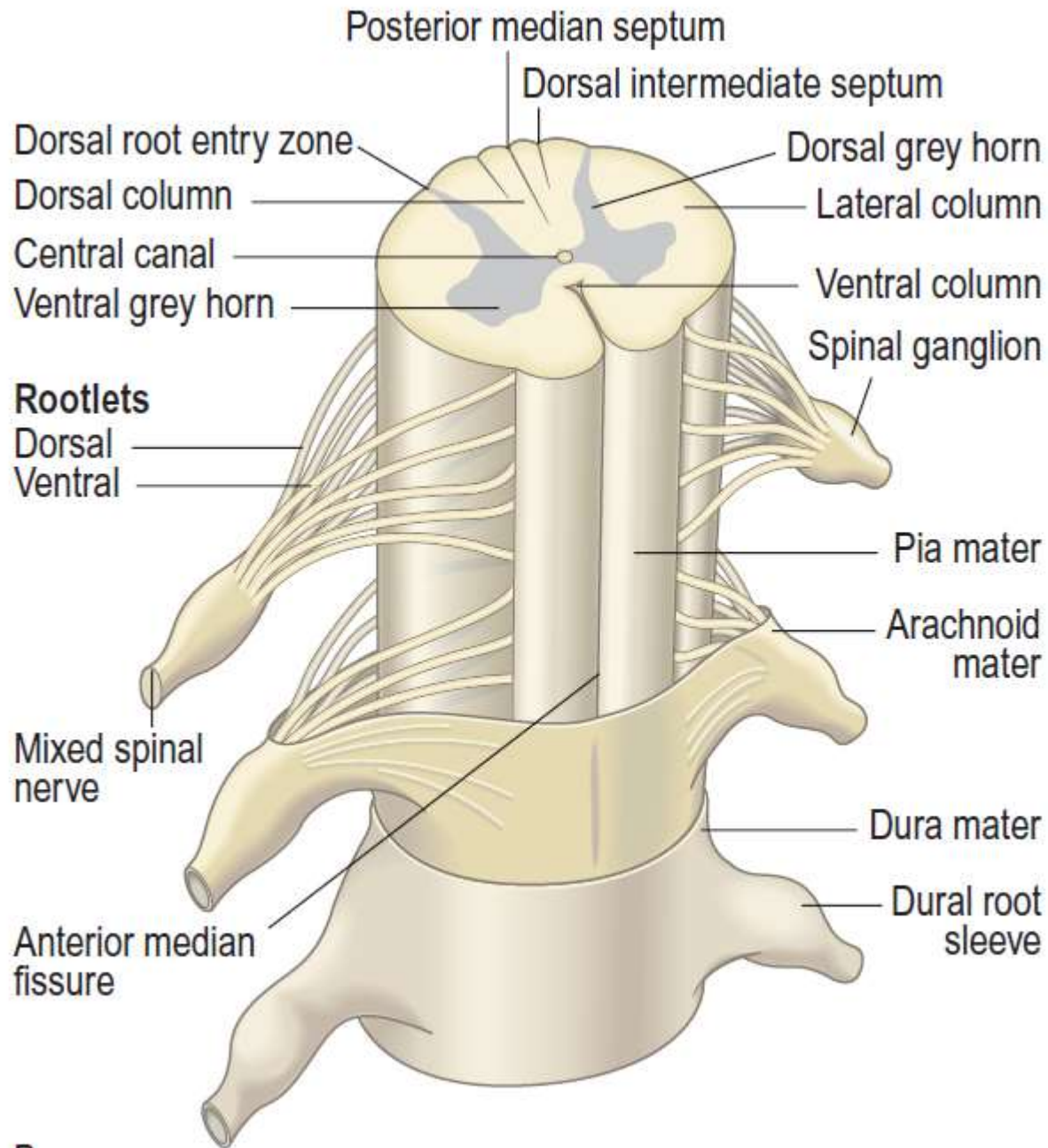


Afferent & efferent nerve pathways

- Afferents carry CNS inputs
- Afferents Arrive

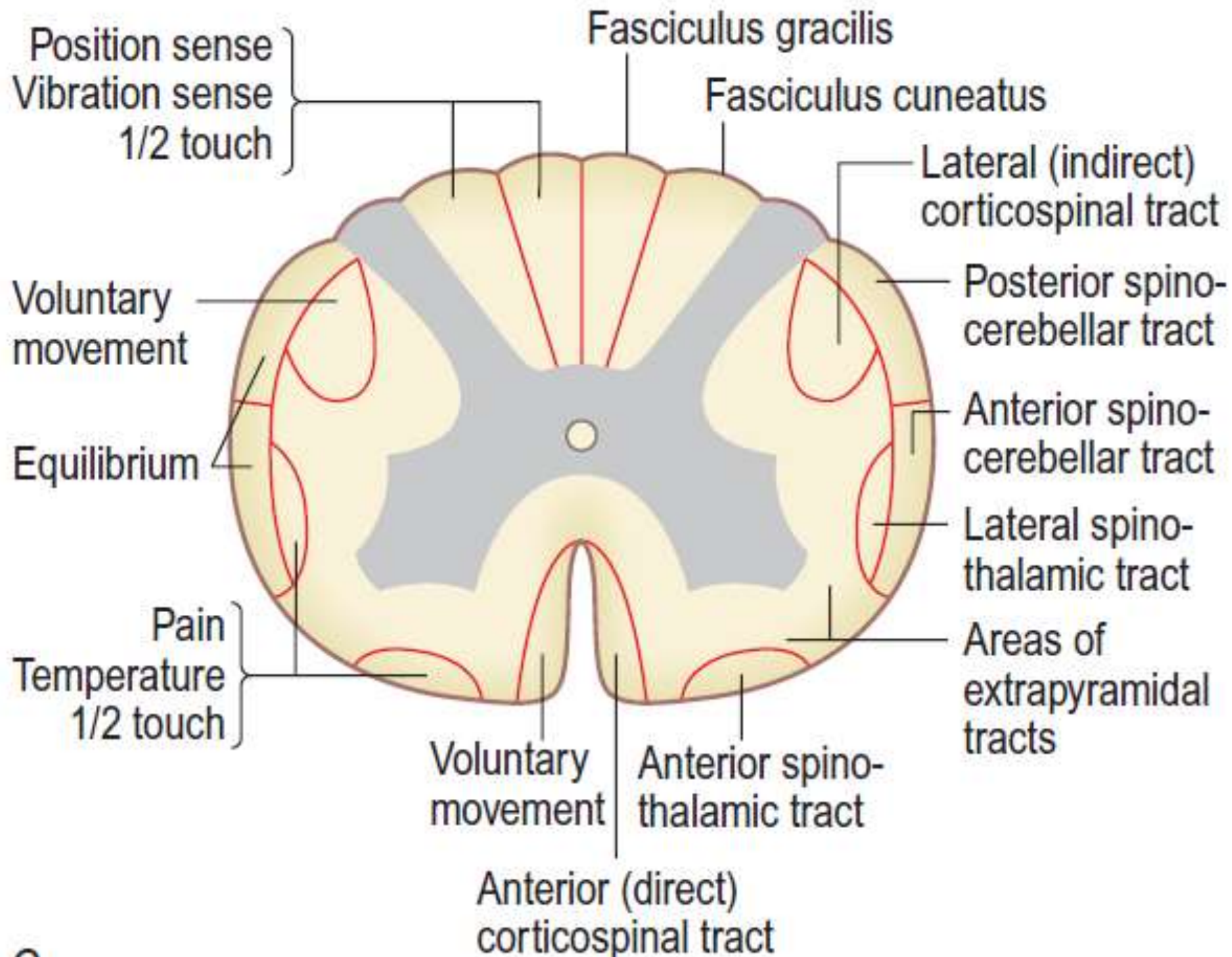


- Efferents carry CNS outputs
- Efferents Exit



B





Fasciculus gracilis

Fasciculus cuneatus

Lateral (indirect) corticospinal tract

Posterior spino-cerebellar tract

Anterior spino-cerebellar tract

Lateral spino-thalamic tract

Areas of extrapyramidal tracts

Voluntary movement

Anterior spino-thalamic tract

Anterior (direct) corticospinal tract

Position sense
Vibration sense
1/2 touch

Voluntary movement

Equilibrium

Pain
Temperature
1/2 touch

SYMPTOMS

- Headache (causes can be primary or secondary)

- Disturbances of consciousness
 1. Syncope
 2. Seizures
 3. Dizziness
 4. Vertigo

Look for: Timing, Precipitating, Exacerbating, Relieving factors, Associated symptoms.



7.1 Clinical characteristics of headache syndromes

	Onset	Duration/periodicity	Pain location	Associated features
Primary syndromes				
Migraine	Evolves over 30–120 mins	Usually last < 24 h, recurrent with weeks/months symptom-free	Classically unilateral but may be anywhere including face/neck	Aura (usually visual), nausea/vomiting, photophobia and phonophobia
Cluster headache	Rapid onset, often waking patient from sleep	30–120 mins, 1–4 attacks within 24 h, clusters usually last weeks to months, with months to years of remission	Orbital/retro-orbital; always same side during cluster, may switch sides between clusters	Autonomic features, including conjunctival injection, tearing, nasal stuffiness, ptosis, miosis, agitation
Stabbing headache	Abrupt, rarely from sleep	Very brief, seconds or less	Anywhere over head	Common in migraineurs
Secondary syndromes				
Meningitis	Usually evolves over a day or two, can be abrupt	Depends on cause and treatment, usually days to weeks	Global, including neck stiffness	Fever, meningism, rash, false localising signs, signs of raised intracranial pressure
Subarachnoid haemorrhage	Abrupt, immediately maximal, rare from sleep	May be fatal at onset, usually days to weeks	Anywhere, poor localising value	20% isolated headache only; nausea/vomiting, reduced consciousness, false localising signs, III nerve palsies
Temporal arteritis	Gradual onset of temple pain and scalp tenderness	Continuous	Temple and scalp	Usually in those > 55 years; unwell, jaw pain on chewing, visual symptoms, tender temporal arteries, elevated erythrocyte sedimentation rate and C-reactive protein



ONSET AND COURSE OF HEADACHES

Acute single episode

Subarachnoid haemorrhage
Acute meningitis

Vasodilator drugs
Angle-closure glaucoma

Acute recurrent

Migraine
Sinusitis
Neuralgias (e.g., trigeminal and post-herpetic)

Angle-closure glaucoma
Cluster headache

Subacute single episode

Infections (e.g., tuberculous meningitis, cerebral abscess)
Raised intracranial pressure (e.g., tumour, hydrocephalus)
Benign intracranial hypertension
Temporal arteritis

ONSET AND COURSE OF HEADACHES

Chronic

Chronic daily headache syndrome

Depression

Cervical spondylosis

Drugs (e.g., nitrates, overuse of analgesics)

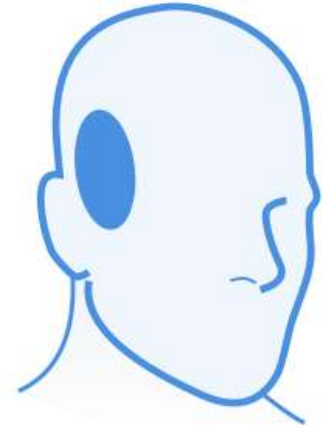




TENSION



CLUSTER



GIANT CELL ARTERITIS



SINUS



MIGRAINE



DISTURBANCES OF CONSCIOUSNESS

Syncope

- Alteration (or loss) of consciousness resulting from inadequate cerebral blood flow.
- Mechanism:
 - Reduced cardiac output (Cardiac syncope)
 - Peripheral vasodilatation (Vasovagal syncope)



CARDIAC SYNCOPE

- Provoked by exertion (severe aortic stenosis, HOCM) or sudden (arrhythmias).
- Recovery is usually rapid.



VASOVAGAL SYNCOPE

- Usually occurs while standing.
- Stimulation of the parasympathetic system e.g., pain, emotional upset.
- More common in warm environments.
- Often preceded by light-headedness, vision darkening, tinnitus, and nausea.
- It causes skin pallor.
- If kept flat, recovery is rapid.



EPILEPTIC SEIZURES

- Definition: paroxysmal electrical discharges from either the whole brain (generalized) or part of the brain (Focal)



Generalized tonic-clonic seizures pattern

Prodromal phase

Change of mood or 'odd' feeling (aura)

Tonic phase

Loss of consciousness

Spasm of all muscles

Cyanosis

Fall

Clonic phase

Jerking of limbs and trunk

Tongue biting

Incontinence of urine

Post-Ictal phase

Flaccidity

Confusion

Headache

Amnesia



- **Focal seizure:**

Simple focal

Complex focal

Features of complex partial seizures

- **Dream-like states**
- **Disturbances of memory (déjà-vu, jamais vu)**
- **Hallucinations of smell, taste or auditory**
- **Emotional disturbance**
- **Abnormal behavior**



7.2 Features that help discriminate vasovagal syncope from epileptic seizure

Feature	Vasovagal syncope	Seizure
Triggers	Typically pain, illness, emotion	Often none (sleep deprivation, alcohol, drugs)
Prodrome	Feeling faint/lightheaded, nausea, tinnitus, vision dimming	Focal onset (not always present)
Duration of unconsciousness	<60 s	1–2 mins
Convulsion	May occur but usually brief myoclonic jerks	Usual, tonic–clonic 1–2 mins
Colour	Pale/grey	Flushed/cyanosed, may be pale
Injuries	Uncommon, sometimes biting of tip of tongue	Lateral tongue biting, headache, generalised myalgia, back pain (sometimes vertebral compression fractures), shoulder fracture/dislocation (rare)
Recovery	Rapid, no confusion	Gradual, over 30 mins; patient is often confused, sometimes agitated/aggressive, amnesic



Item	Epileptic seizures	PNES
Eyes	Opened	Closed
Head	Fixed/unilateral	Side-to-side movements
Limbs	In phase/same direction	Out of phase
Body (axis)	Straight	Opisthotonus
Body (movement)	No rotation	Intense rotation in bed
Evolution of seizure	Continuous	Fluctuating

PNES, psychogenic non-epileptic seizures; ES, epileptic seizures.



DIZZINESS

May be due to:

1. Postural hypotension
2. Cerebrovascular disease/vertebrobasilar insufficiency
3. Arrhythmia
4. Hyperventilation



VERTIGO (THE ILLUSION OF MOVEMENT)

Causes of vertigo

Central

- Migraine
- Brainstem ischemia or infarction
- Multiple sclerosis

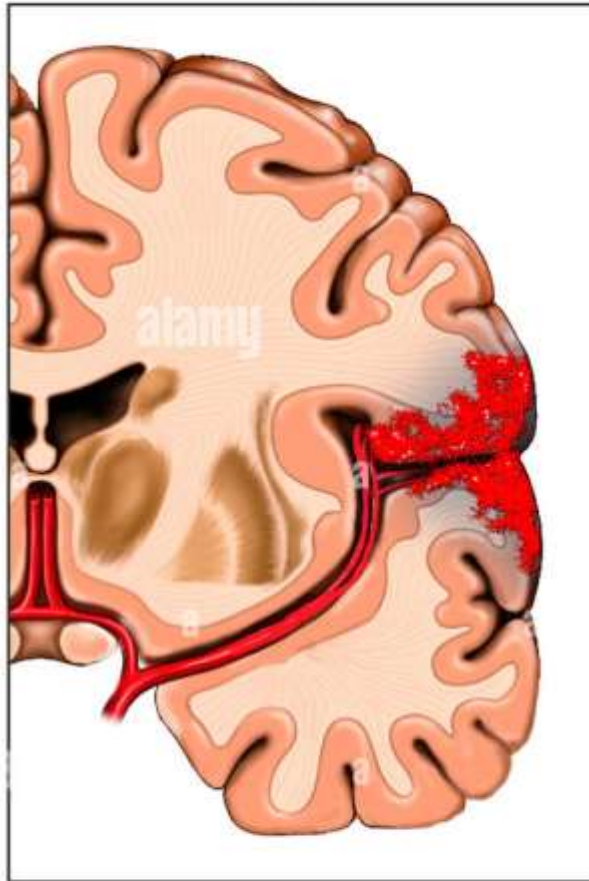
Peripheral

- Ménière's disease (hearing loss , tinnitus , nausea and vomiting)
- Benign paroxysmal positional vertigo
- Vestibular neuritis
- Trauma
- Drugs, e.g. gentamicin, anticonvulsants



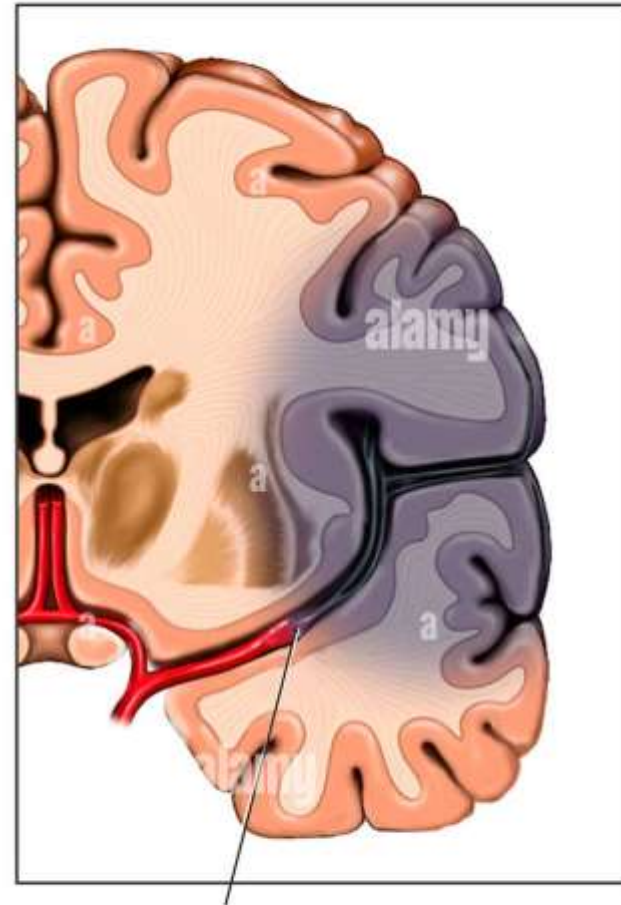
STROKE

Hemorrhagic Stroke



Hemorrhage/blood leaks into brain tissue

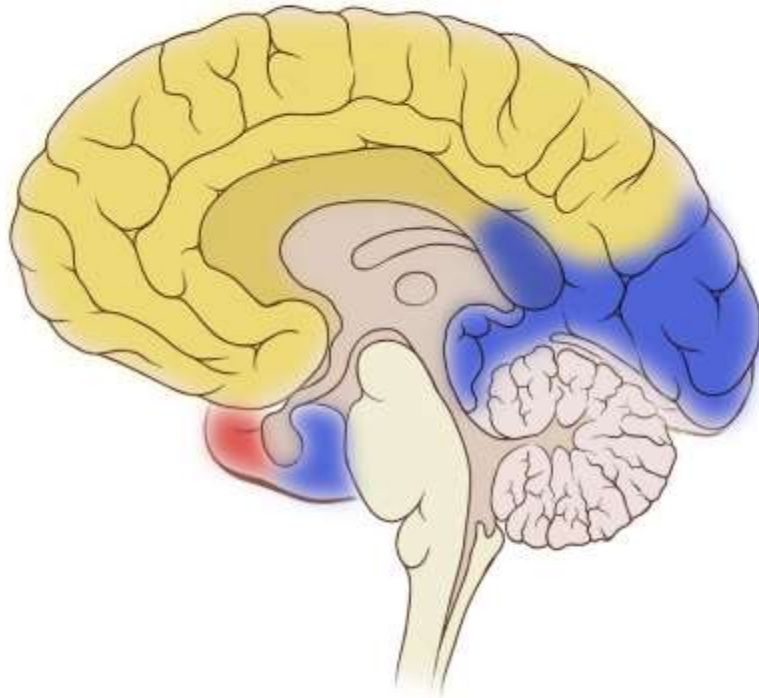
Ischemic Stroke



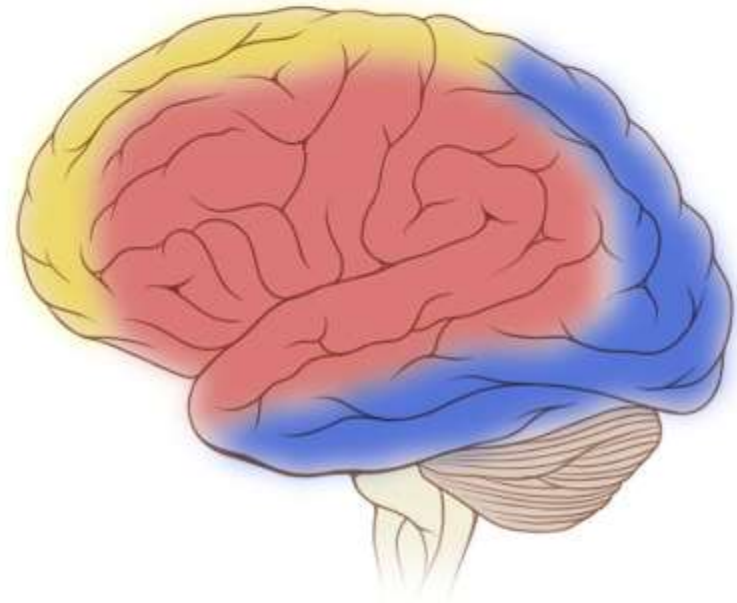
Clot stops blood supply to an area of the brain




TERRITORY OF THE CEREBRAL VESSELS

Medial view



Lateral view



-  Posterior cerebral artery
-  Middle cerebral artery
-  Anterior cerebral artery



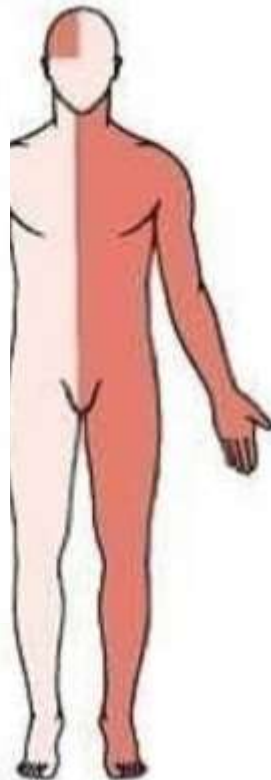
STROKE LEFT BRAIN

STROKE RIGHT BRAIN

Right-brain damage

(stroke on right side of the brain)

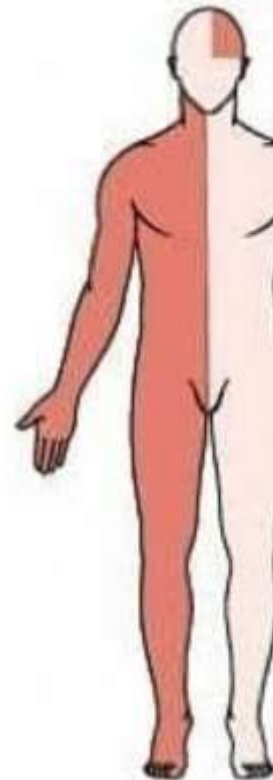
- Paralyzed left side: hemiplegia
- Left-sided neglect
- Spatial-perceptual deficits
- Tends to deny or minimize problems
- Rapid performance, short attention span
- Impulsive; safety problems
- Impaired judgement



Left-brain damage

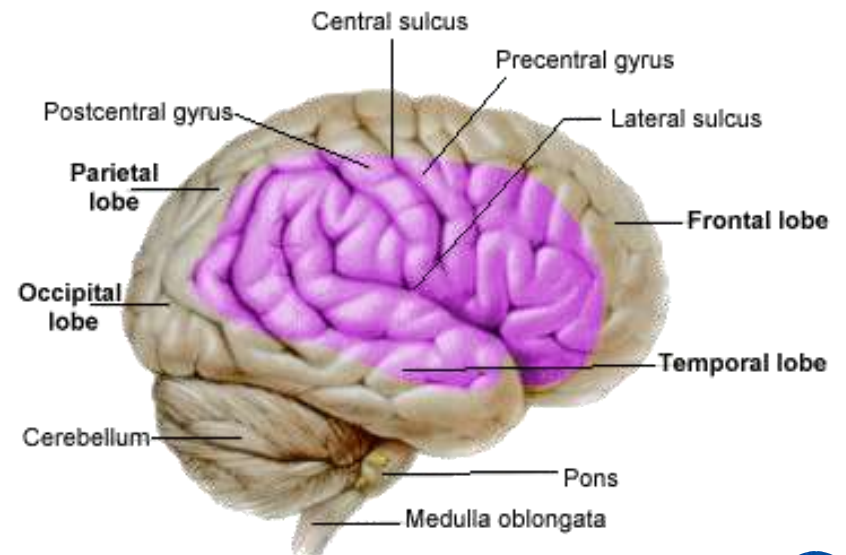
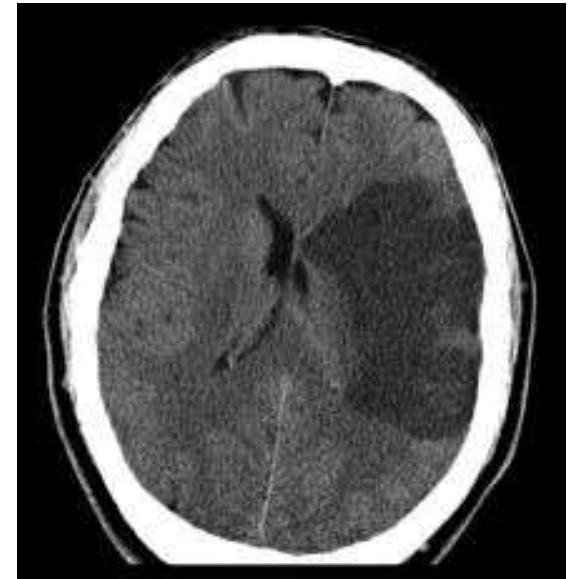
(stroke on left side of the brain)

- Paralyzed right side: hemiplegia
- Impaired speech-language (aphasias)
- Impaired right-left discrimination
- Slow performance, cautious
- Aware of deficits: depression, anxiety
- Impaired comprehension related to language, math



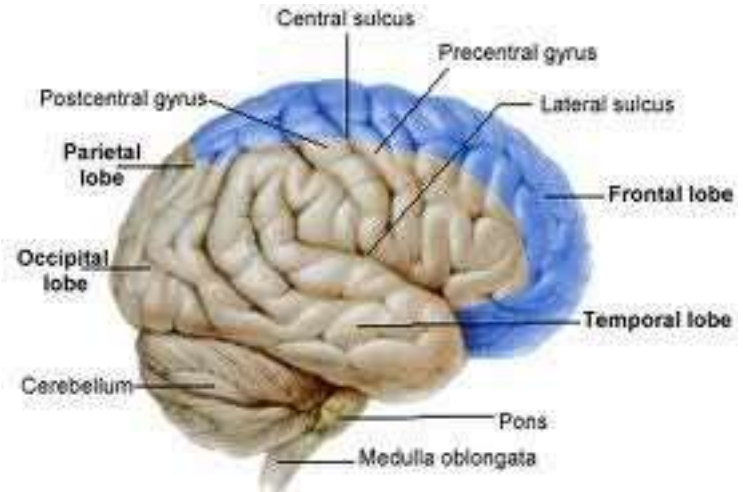
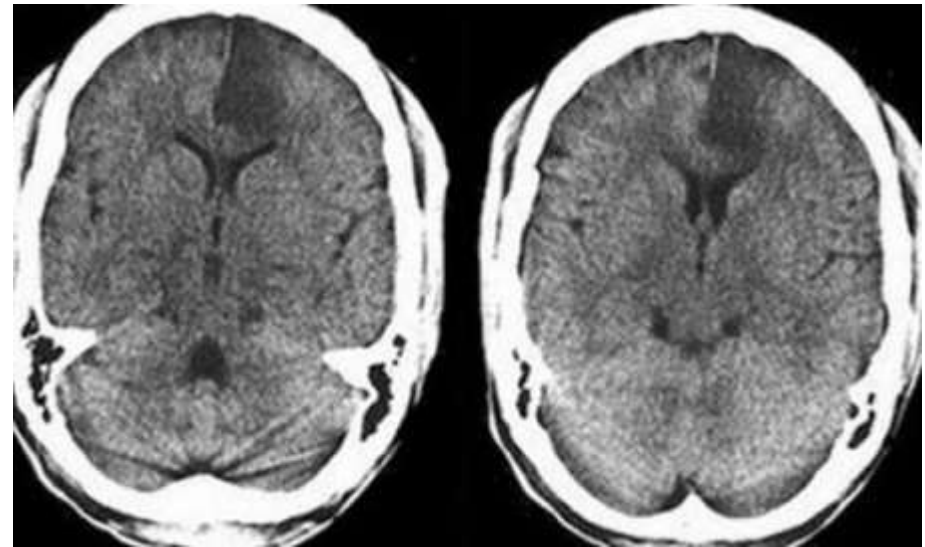
MIDDLE CEREBRAL ARTERY (MCA) OCCLUSION

- Contralateral lower face weakness
- Contralateral hemiplegia
- Contralateral hemianesthesia
- Ataxia
- Speech impairments (usually the left brain)
- Perceptual deficits (usually the right brain)
- Visual deficits



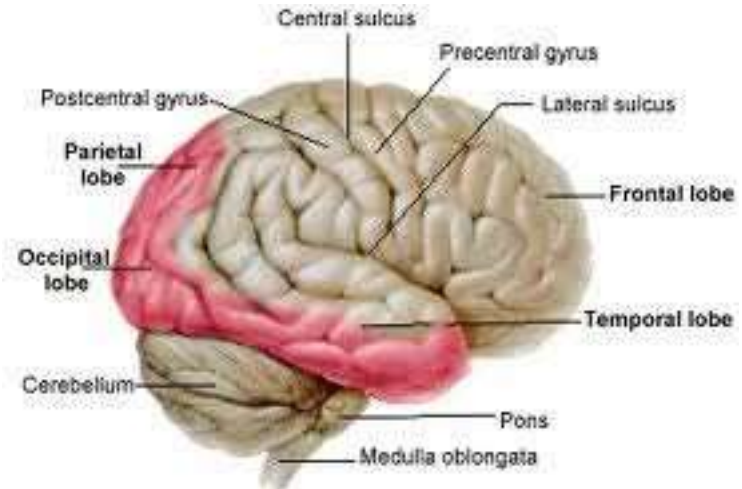
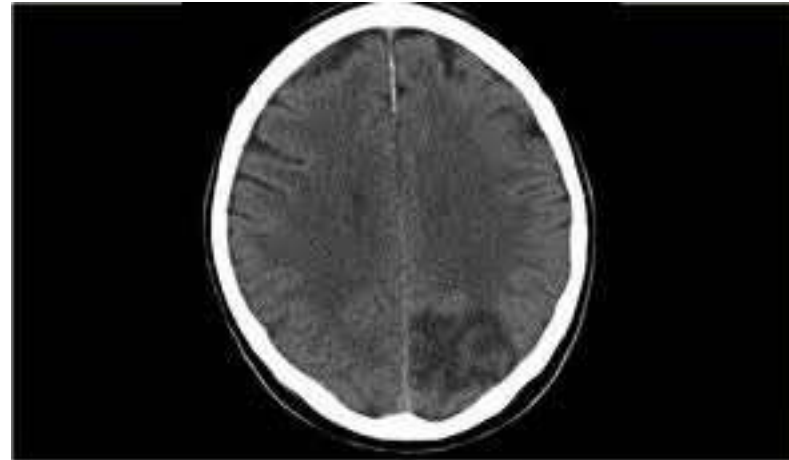
ANTERIOR CEREBRAL ARTERY (ACA) OCCLUSION

- Weakness of foot and leg
- Sensory loss of foot and leg
- Ataxia
- Incontinence



POSTERIOR CEREBRAL ARTERY (PCA) OCCLUSION

- Midbrain syndrome (Weber's Syndrome) occlusion of the paramedian branches of the posterior cerebral artery
 - Ipsilateral Third nerve palsy
 - Contralateral hemiplegia
- Visual field deficits (macular sparing)
- Visual hallucinations
- Memory problems



7.3 Clinical classification of stroke

Total anterior circulation syndrome (TACS)

- Hemiparesis, hemianopia and higher cortical deficit (e.g. dysphasia or visuospatial loss)

Partial anterior circulation syndrome (PACS)

- Two of the three components of a TACS
- OR isolated higher cortical deficit
- OR motor/sensory deficit more restricted than LACS (see below)

Posterior circulation syndrome (POCS)

- Ipsilateral cranial nerve palsy with contralateral motor and/or sensory deficit
- OR bilateral motor and/or sensory deficit
- OR disorder of conjugate eye movement
- OR cerebellar dysfunction without ipsilateral long-tract deficits
- OR isolated homonymous visual field defect

Lacunar syndrome (LACS)

- Pure motor > 2 out of 3 of face, arm, leg
- OR pure sensory > 2 out of 3 of face, arm, leg
- OR pure sensorimotor > 2 out of 3 of face, arm, leg
- OR ataxic hemiparesis



FUNCTIONAL NEUROLOGICAL SYMPTOMS

- Neurological symptoms not due to a neurological disease
- Symptoms that aren't compatible with a disease



THE HISTORY

- Past medical history:
 - Neurological diseases and risk factors
- Drug history
 - Prescriptions, OTC, Recreational, Neurotoxic.
- Family history
 - First degree relatives, Consanguinity (AR).
- Social history
 - Alcohol, Smoking, Vit Deficiencies, Sexual history, Travel history.
- Occupational history
 - Exposure (lead, manganese...), adverse effects on occupation.



GENERAL APPROACH TO NEUROLOGICAL EXAMINATION

- General look of patient
- Vital signs
- Level of consciousness
- High cognitive functions
- Stance and gait
- Cranial nerves 1-12
- Motor system
- Sensory system
- Coordination and cerebellum



GENERAL LOOK OF PATIENT

Begins with your first contact with the patient and continues during history taking

- Facial expression
- General demeanour
- Posture
- Gait
- Speech
- Involuntary movements



ASSESSMENT OF CONSCIOUS LEVEL

two main components: state and content.

- **The state of consciousness:**
 - dependent on the integrity of RAS(reticular activity system, extend from brainstem to thalamus)
 - describes how awake a person is

- **The content of consciousness:**
 - depends on the cerebral cortex, thalamus and their connections.
 - describes how aware the person is





19.14 Glasgow Coma Scale



Eye opening

Spontaneous	4
To speech	3
To pain	2
No response	1

Verbal response

Orientated	5
Confused: talks in sentences but disorientated	4
Verbalises: words, not sentences	3
Vocalises: sounds (groans or grunts), not words	2
No vocalisation	1

Motor response

Obeys commands	6
Localises to pain, e.g. brings hand up beyond chin to supraorbital pain	5
Flexion withdrawal to pain: no localisation to supraorbital pain but flexes elbow to nail bed pressure	4
Abnormal flexion to pain	3
Extension to pain: extends elbow to nail bed pressure	2
No response	1

Record the GCS as a total and its three separate components: e.g. GCS 9/15: E3, V2, M4



MENINGEAL IRRITATION SIGNS

- Neck stiffness
 - increased resistance to passive flexion of the neck

- Kernig's sign
 - increased resistance to passive extension of the leg

- Brudsiniski's sign
 - Flexion of the knees in response to neck flexion



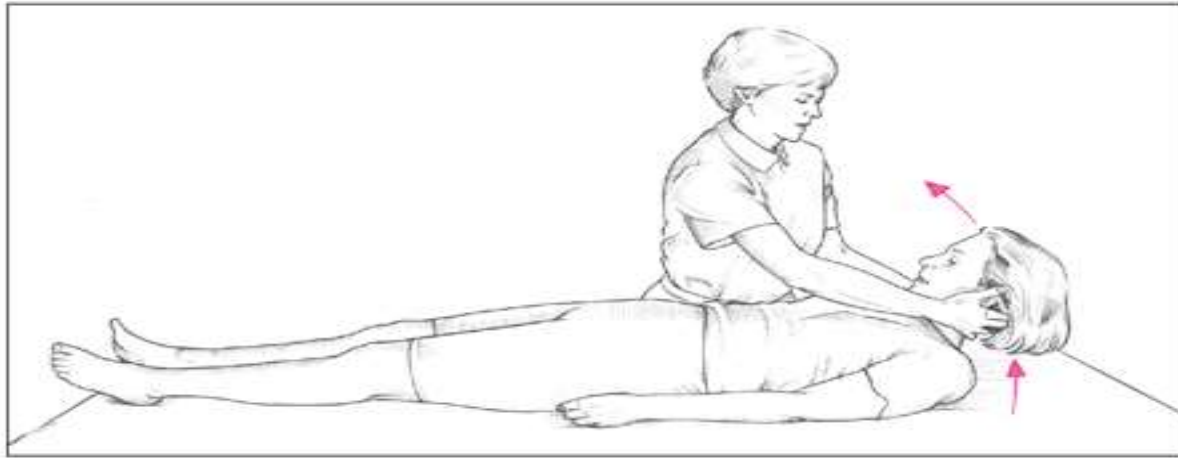
NECK STIFFNESS



Testing for Brudzinski's sign

Here's how to test for Brudzinski's sign when you suspect meningeal irritation:

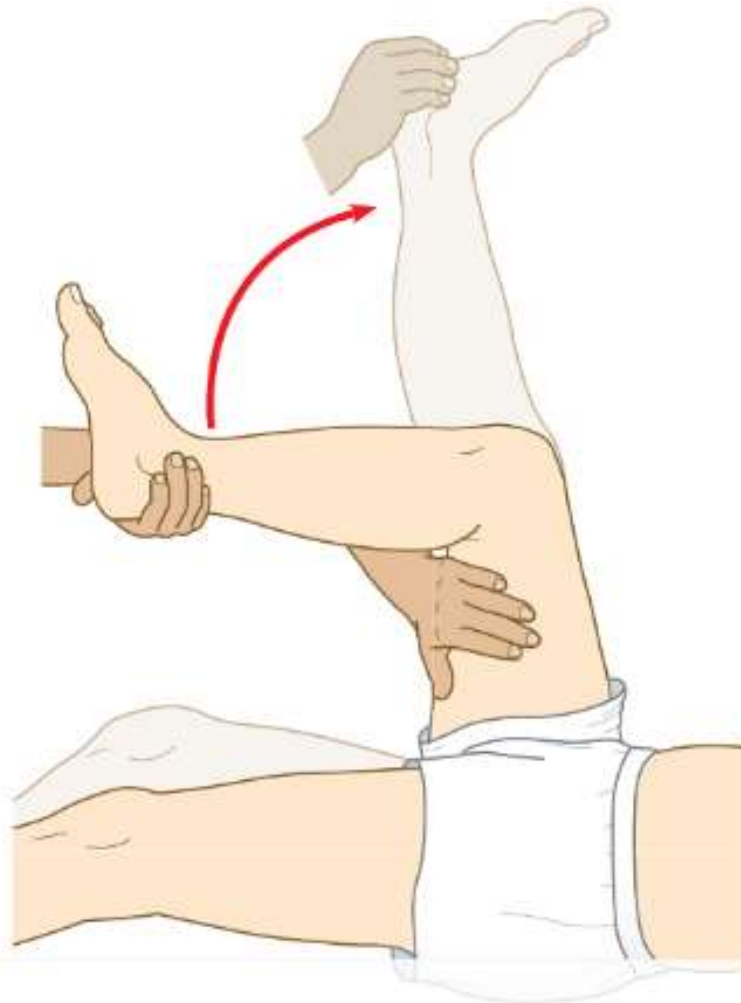
With the patient in a supine position, place your hands behind her neck and lift her head toward her chest.



If your patient has meningeal irritation, she'll flex her hips and knees in response to the passive neck flexion.



KERNIG'S SIGN



SPEECH EXAMINATION

- Listen to the patient's spontaneous speech, noting volume, rhythm and clarity.
- Ask the patient to repeat phrases such as 'yellow lorry' to test lingual (tongue) sounds and 'baby hippopotamus' for labial (lip) sounds, then a tongue twister, e.g. 'the Leith police dismisseth us'.
- Ask the patient to count steadily to 30 to assess fatigue.
- Ask the patient to cough and to say 'Ah'; observe the soft palate rising bilaterally.



SPEECH ABNORMALITIES

- **Dysarthria:**
 - slurred speech caused by articulation problems due to a motor deficit
- **Dysphonia:**
 - loss of volume caused by laryngeal disorders
- **Dysphasia:**
 - disturbance of language resulting in abnormalities of speech production and/or understanding
 - may also involve other language symptoms, e.g. writing and reading.



DYSARTHRIA

- **Pseudobulbar palsy:**
 - contracted, spastic tongue and difficulty pronouncing consonants
- **Bulbar palsy:**
 - Weakness of the tongue results in difficulty with lingual sounds, while palatal weakness gives a nasal quality to the speech.
- **Cerebellar dysarthria:**
 - slow and slurred, similar to alcohol intoxication.
- **Myasthenia gravis:**
 - fatiguing speech.
- **Parkinsonism:**
 - dysarthria and dysphonia, with a low-volume, monotonous voice



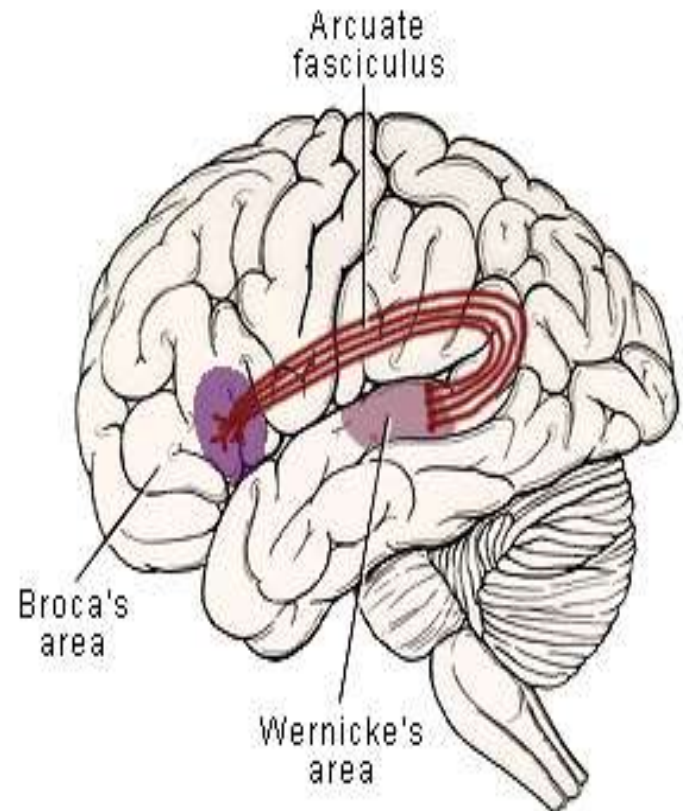
DYSPHASIA EXAMINATION

- Listen to the fluency and appropriateness of the content during speech.
- Ask the patient to name a common object
- Give a simple three-stage command
- Ask the patient to repeat a simple sentence
- Ask the patient to read a passage from a newspaper.
- Ask the patient to write a sentence; examine his handwriting.



DYSPHASIA

- *Expressive (motor) dysphasia*
- *Receptive (sensory) dysphasia*
- *Conduction dysphasia*
- *Global dysphasia*
- *Dyslexia*
- *Dyscalculia*
- *Dysgraphia*



Speech



**Frontal
Lobe**



**Temporal
Lobe**



Broca's Area:

- In the left frontal lobe
- Controls production of speech sounds
- Lies close to motor areas



Wernicke's Area:

- Left temporal lobe
- Gets meaning from words and sentences
- Formulates ideas into speech

2 Parietal lobe

Dominant side

FUNCTION

Calculation
Language
Planned movement
Appreciation of size, shape, weight and texture

LESIONS

Dyscalculia
Dysphasia
Dyslexia
Apraxia
Agnosia
Homonymous hemianopia

Non-dominant side

FUNCTION

Spatial orientation
Constructional skills

LESIONS

Neglect of non-dominant side
Spatial disorientation
Constructional apraxia
Dressing apraxia
Homonymous hemianopia

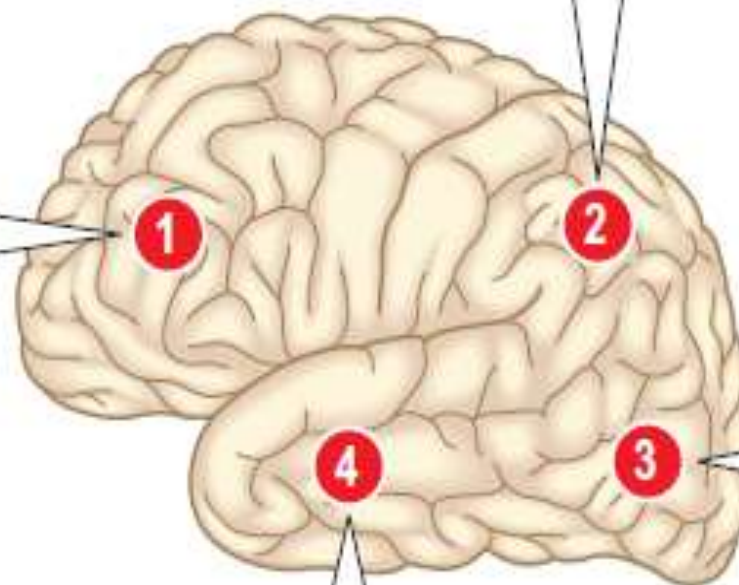
1 Frontal lobe

FUNCTION

Personality
Emotional response
Social behaviour

LESIONS

Disinhibition
Lack of initiative
Antisocial behaviour
Impaired memory
Incontinence
Grasp reflexes
Anosmia



3 Occipital lobe

FUNCTION

Analysis of vision

LESIONS

Homonymous hemianopia
Hemianopic scotomas
Visual agnosia
Impaired face recognition (prosopagnosia)
Visual hallucinations (lights, lines and zig-zags)

4 Temporal lobe

Dominant side

FUNCTION

Auditory perception
Speech, language
Verbal memory
Smell

LESIONS

Dysphasia
Dyslexia
Poor memory
Complex hallucinations (smell, sound, vision)
Homonymous hemianopia

Non-dominant side

FUNCTION

Auditory perception
Music, tone sequences
Non-verbal memory (faces, shapes, music)
Smell

LESIONS

Poor non-verbal memory
Loss of musical skills
Complex hallucinations
Homonymous hemianopia