La-Net

MED 408

NEUROLOGY I

Downoad on the App Store

GET IT ON Google Play

Course video is available on our app

lanetonline.com

Course code: MED 408

Course title: Neurology I

Course credit load: 2 Units

Series 1: Foundations of Neurology

- **Part 1.1: Overview of the Nervous System**
    - Sub Part 1.1.1: Organisation of the nervous system
    - Sub Part 1.1.2: Neurons and glial cells
    - Sub Part 1.1.3: The synapse and neurotransmitters
- **Part 1.2: The Central Nervous System**
    - Sub Part 1.2.1: The cerebral cortex and lobes
    - Sub Part 1.2.2: The brainstem and cerebellum
    - Sub Part 1.2.3: The spinal cord
- **Part 1.3: The Peripheral and Autonomic Nervous Systems**
    - Sub Part 1.3.1: The peripheral nervous system
    - Sub Part 1.3.2: The autonomic nervous system
    - Sub Part 1.3.3: The neuromuscular junction
- **Part 1.4: Neurological Localisation**
    - Sub Part 1.4.1: Principles of localisation
    - Sub Part 1.4.2: Upper and lower motor neuron lesions
    - Sub Part 1.4.3: Common localisation patterns

Introduction

Neurology is the branch of medicine concerned with disorders of the nervous system. A sound knowledge of neuroanatomy and neurophysiology is the essential starting point for clinical practice in neurology, because every deficit a patient presents with reflects damage to a specific, predictable part of the nervous system. This Series builds that foundation.

We shall commence by examining how the nervous system is organised and how its cells communicate. Next, we will explore the functional anatomy of the central nervous system. Moving on, we will consider the peripheral and autonomic nervous systems. Finally, we will apply all of this to neurological localisation, the clinical skill of identifying the site of a lesion from the pattern of the patient's deficits.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 1.1:** describe the organisation of the nervous system and the structure and function of neurons and glial cells,
- **SLO 1.2:** describe the structure and function of the synapse and the major neurotransmitters,
- **SLO 1.3:** describe the functional anatomy of the cerebral cortex, brainstem, cerebellum, and spinal cord,
- **SLO 1.4:** describe the organisation and function of the peripheral and autonomic nervous systems and the neuromuscular junction, and
- **SLO 1.5:** apply the principles of neurological localisation to distinguish upper from lower motor neuron lesions and identify common localisation patterns.

1.1 Overview of the Nervous System

1.1.1 Organisation of the nervous system

The nervous system is divided into two broad components. The central nervous system (CNS) consists of the brain and spinal cord, where information is integrated and processed. The peripheral nervous system (PNS) includes all neural tissue outside the CNS: cranial nerves, spinal nerves, ganglia, and plexuses.

Within the PNS, the somatic nervous system controls voluntary skeletal muscle movement and transmits sensory information from the body to the CNS. The autonomic nervous system (ANS) regulates involuntary visceral functions such as heart rate, blood pressure, and digestion, and is further divided into the sympathetic (fight-or-flight) and parasympathetic (rest-and-digest) divisions.

Fill in the blank: The brain and spinal cord together form the _____ nervous system. The division controlling involuntary visceral functions is the _____ nervous system.

1.1.2 Neurons and glial cells

The neuron is the functional unit of the nervous system. It consists of dendrites (short processes that receive incoming signals), a cell body or soma (containing the nucleus), and an axon (a single long process that conducts action potentials away from the soma to the axon terminals). Many axons are wrapped in a myelin sheath, which insulates the fibre and enables rapid saltatory conduction by allowing the impulse to jump between gaps called nodes of Ranvier.

Glial cells support and protect neurons. Oligodendrocytes produce myelin in the CNS (one cell myelinating up to 50 axons), while Schwann cells produce myelin in the PNS (one cell per axon segment). Astrocytes maintain the blood-brain barrier and provide metabolic support. Microglia are the resident immune cells of the CNS. Ependymal cells line the ventricles and produce cerebrospinal fluid.

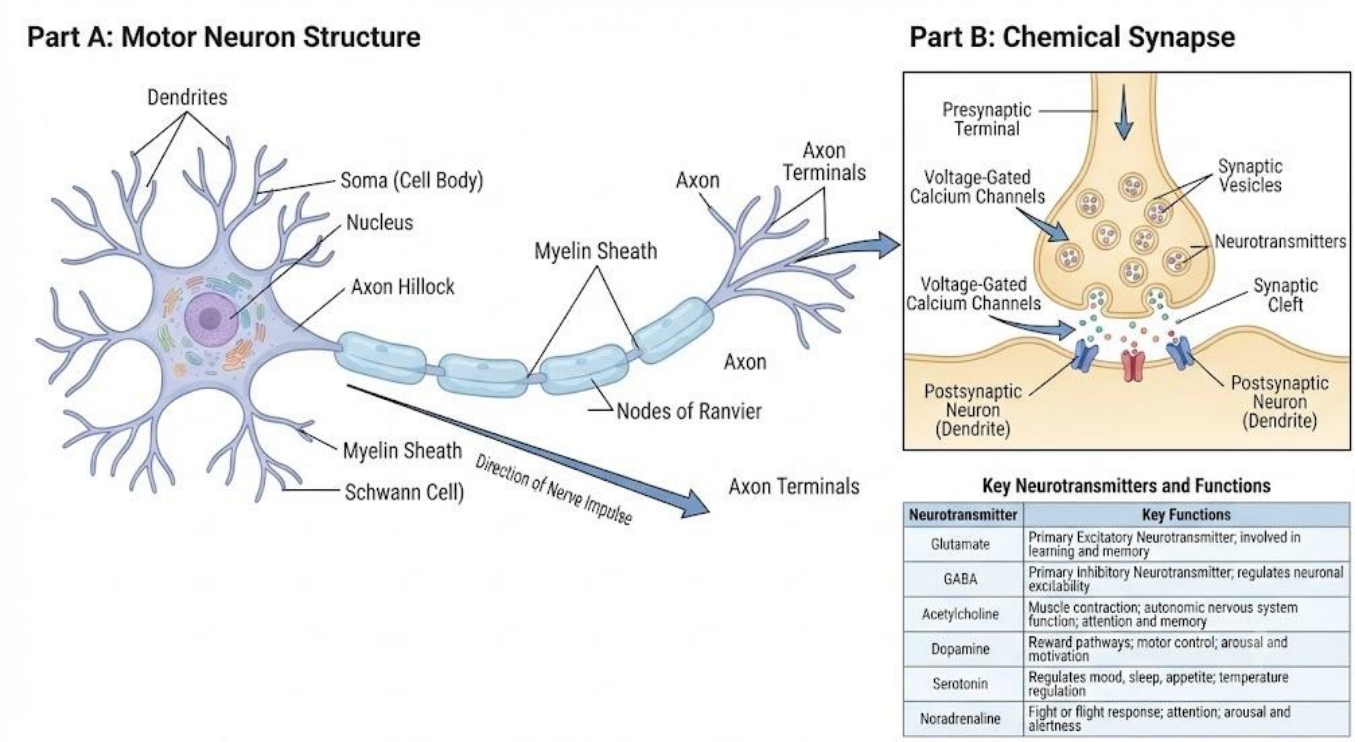
Fill in the blank: The process that conducts signals away from the neuron cell body is the _____. In the CNS, myelin is produced by _____, while in the PNS it is produced by Schwann cells.

1.1.3 The synapse and neurotransmitters

A synapse is the junction between two neurons or between a neuron and its target cell. When an action potential reaches the presynaptic terminal, calcium influx triggers release of neurotransmitter into the synaptic cleft. The neurotransmitter binds to receptors on the postsynaptic membrane, producing either excitation or inhibition. The signal ends when the neurotransmitter is broken down by enzymes or taken back up into the presynaptic terminal.

The major neurotransmitters are: glutamate (the main CNS excitatory neurotransmitter), GABA (the main CNS inhibitory neurotransmitter, enhanced by benzodiazepines), acetylcholine (at the neuromuscular junction and in the ANS), dopamine (movement and reward, depleted in Parkinson's disease), serotonin (mood and sleep, increased by SSRIs), and noradrenaline (arousal and sympathetic function).

Fill in the blank: The main inhibitory neurotransmitter in the CNS is _____, while the main excitatory neurotransmitter is glutamate. _____ is depleted in the substantia nigra in Parkinson's disease.



Key Neurotransmitters and Functions

Neurotransmitter	Key Functions
Glutamate	Primary Excitatory Neurotransmitter; involved in learning and memory
GABA	Primary Inhibitory Neurotransmitter; regulates neuronal excitability
Acetylcholine	Muscle contraction; autonomic nervous system function; attention and memory
Dopamine	Reward pathways; motor control; arousal and motivation
Serotonin	Regulates mood, sleep, appetite; temperature regulation
Noradrenaline	Fight or flight response; attention; arousal and alertness

Figure 1.1: Structure of a neuron and chemical synapse with neurotransmitter reference

Pilot questions 1.1

1. Distinguish the CNS from the PNS. (Linked to SLO 1.1)
2. Describe the structure of a neuron, naming each part and its function. (Linked to SLO 1.1)
3. Distinguish oligodendrocytes from Schwann cells. (Linked to SLO 1.1)
4. Describe the sequence of events at a chemical synapse. (Linked to SLO 1.2)
5. Name four major neurotransmitters and state the function of each. (Linked to SLO 1.2)

1.2 The Central Nervous System

1.2.1 The cerebral cortex and lobes

The cerebrum is divided into four lobes. The frontal lobe contains the primary motor cortex (voluntary movement), the prefrontal cortex (executive function and personality), and Broca's area in the dominant hemisphere (motor speech production). The parietal lobe contains the primary somatosensory cortex and Wernicke's area in the dominant hemisphere (language comprehension); the non-dominant parietal lobe mediates spatial attention. The temporal lobe

processes auditory information and houses the hippocampus (memory formation). The occipital lobe contains the primary visual cortex, processing the contralateral visual field.

All motor fibres from the cortex and all sensory fibres to the cortex pass through the internal capsule, a compact white matter tract deep within each hemisphere. A small lesion here causes a dense contralateral hemiplegia affecting the face, arm, and leg equally, because all fibres are packed into this small area.

Fill in the blank: Broca's area in the dominant hemisphere controls motor _____ production. Damage to Wernicke's area causes fluent but meaningless speech with impaired _____.

1.2.2 The brainstem and cerebellum

The brainstem (midbrain, pons, and medulla) contains the nuclei of cranial nerves III to XII, the reticular activating system (consciousness and arousal), and the respiratory and cardiovascular centres. All ascending sensory and descending motor tracts pass through it. A brainstem lesion produces the crossed deficit pattern: ipsilateral cranial nerve signs (from the cranial nerve nucleus at the level of the lesion) combined with contralateral limb weakness and sensory loss (from the long tracts).

The cerebellum coordinates movement and balance. Because of the double-crossing of its pathways, cerebellar lesions produce ipsilateral signs, remembered by the mnemonic DANISH: Dysdiadochokinesia, Ataxia, Nystagmus, Intention tremor, Scanning speech (dysarthria), and Hypotonia.

Fill in the blank: A brainstem lesion causes _____ cranial nerve signs combined with _____ limb signs (the crossed deficit). Cerebellar signs occur on the _____ side as the lesion.

1.2.3 The spinal cord

The spinal cord extends from the foramen magnum to the conus medullaris at L1 to L2. On cross-section, grey matter (H-shaped) contains the anterior horn lower motor neurons and the posterior horn sensory relay neurons, surrounded by white matter tracts. Three tracts are essential knowledge: the dorsal columns (fine touch, vibration, proprioception: cross at the medulla), the spinothalamic tract (pain and temperature: cross within 1 to 2 segments of entry), and the corticospinal tract (voluntary motor: crosses at the pyramidal decussation in the medulla).

A cord hemisection produces the Brown-Sequard syndrome: ipsilateral motor loss and dorsal column loss (fine touch, vibration, proprioception) combined with contralateral pain and temperature loss, all below the level of the lesion. This dissociated sensory loss is unique to spinal cord lesions.

Fill in the blank: The dorsal columns cross at the _____, while the spinothalamic tract crosses within _____ to two segments of entering the cord. A cord hemisection produces the _____ syndrome.

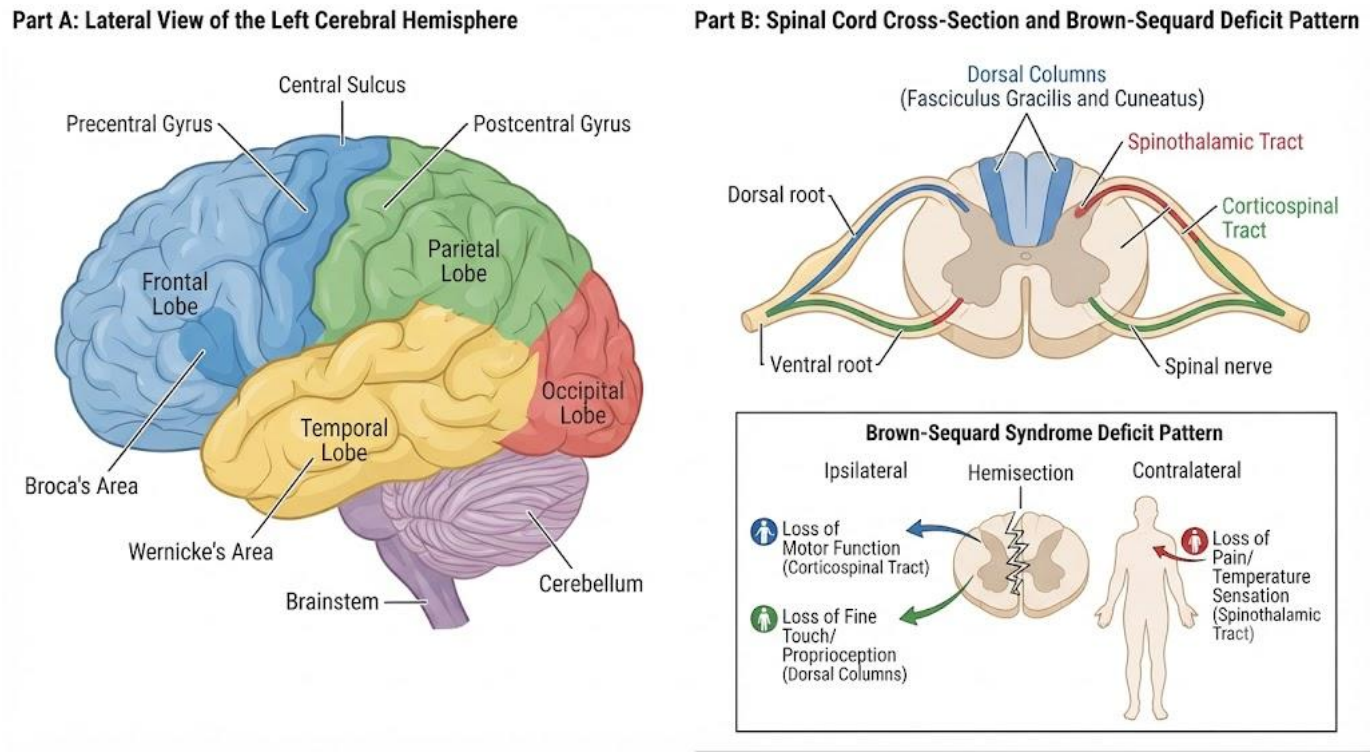


Figure 1.2: Cerebral lobes with key functional areas and spinal cord cross-section with tracts

Pilot questions 1.2

1. Describe the functions of the four cerebral lobes. (Linked to SLO 1.3)
2. Explain the difference between Broca's and Wernicke's aphasia. (Linked to SLO 1.3)
3. Describe the crossed deficit pattern of a brainstem lesion. (Linked to SLO 1.3)
4. List the DANISH signs of cerebellar disease. (Linked to SLO 1.3)
5. Describe the three major spinal cord tracts and the Brown-Sequard syndrome. (Linked to SLO 1.3)

1.3 The Peripheral and Autonomic Nervous Systems

1.3.1 The peripheral nervous system

There are 31 pairs of spinal nerves, each formed by the union of a dorsal (posterior) root carrying sensory fibres from the dorsal root ganglion, and a ventral (anterior) root carrying motor fibres from the anterior horn cells. After leaving the intervertebral foramen, spinal nerves form plexuses (brachial and lumbosacral) that give rise to the named peripheral nerves.

A dermatome is the area of skin supplied by a single dorsal spinal root; sensory loss mapped against a dermatome chart localises nerve root lesions. A myotome is the muscle group supplied by a single ventral root; testing specific muscle groups against their known root supply localises motor deficits. For example, shoulder abduction is C5, wrist extension is C7, and ankle dorsiflexion is L4.

Fill in the blank: Each spinal nerve is formed by the union of the dorsal _____ root and the ventral _____ root. The area of skin supplied by a single dorsal root is called a _____.

1.3.2 The autonomic nervous system

The sympathetic division originates from the thoracolumbar spinal cord (T1 to L2). Pre-ganglionic fibres are short, synapsing in the paravertebral chain, and long post-ganglionic fibres reach the target organs. The principal post-ganglionic neurotransmitter is noradrenaline (except at sweat glands, where it is acetylcholine). Sympathetic effects include increased heart rate and blood pressure, pupil dilation, bronchodilation, and inhibited digestion.

The parasympathetic division has a craniosacral origin: cranial nerves III, VII, IX, and X, plus sacral roots S2 to S4. Pre-ganglionic fibres are long, synapsing in ganglia close to the target organ, and post-ganglionic fibres are short. The neurotransmitter is acetylcholine acting on muscarinic receptors. Parasympathetic effects include decreased heart rate, pupil constriction, bronchoconstriction, and stimulated digestion.

Fill in the blank: The sympathetic division originates from the _____ spinal cord and uses _____ as its main post-ganglionic neurotransmitter. The parasympathetic uses acetylcholine on _____ receptors.

1.3.3 The neuromuscular junction

The neuromuscular junction (NMJ) is the synapse between a lower motor neuron terminal and the motor end plate of a skeletal muscle fibre. An action potential triggers calcium influx at the nerve terminal, causing acetylcholine (ACh) release into the synaptic cleft. ACh binds to nicotinic receptors on the motor end plate, depolarising the muscle fibre and triggering contraction. Acetylcholinesterase in the cleft then degrades ACh, terminating the signal.

Myasthenia gravis is the most important NMJ disease: IgG antibodies target the post-synaptic nicotinic ACh receptors, reducing their number and causing fatigable weakness that worsens with activity and improves with rest. Ocular muscles are typically affected first (ptosis and diplopia). Treatment includes pyridostigmine (an acetylcholinesterase inhibitor) and immunosuppression. In contrast, Lambert-Eaton myasthenic syndrome targets pre-synaptic voltage-gated calcium channels and is associated with small cell lung cancer; weakness paradoxically improves briefly with repeated activity.

Fill in the blank: At the NMJ, acetylcholine binds to _____ receptors on the motor end plate. In myasthenia gravis, antibodies target _____ receptors, causing _____ weakness that worsens with activity.

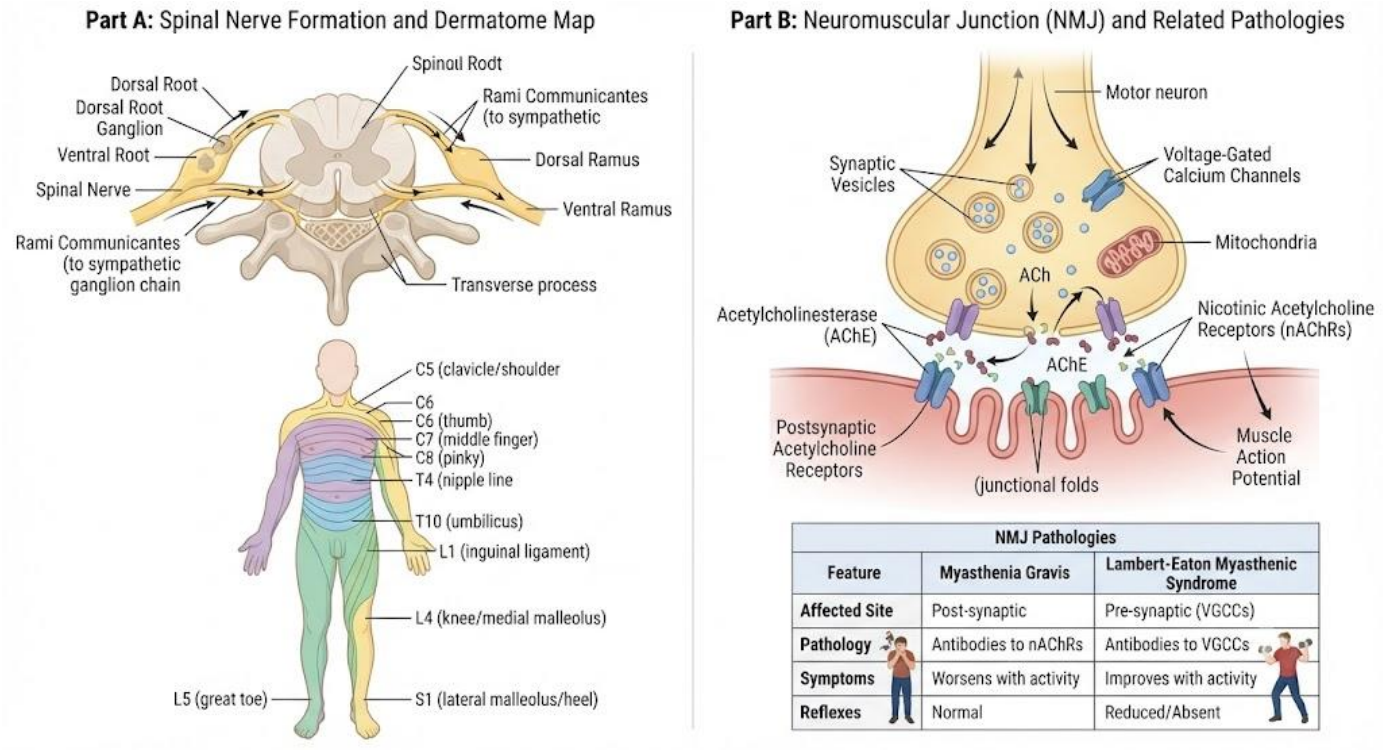


Figure 1.3: Spinal nerve formation with key dermatomes and the neuromuscular junction with NMJ disorders

Pilot questions 1.3

1. Describe the formation of a spinal nerve. (Linked to SLO 1.4)
2. Distinguish the sympathetic from the parasympathetic division. (Linked to SLO 1.4)
3. Describe the events at the NMJ leading to muscle contraction. (Linked to SLO 1.4)
4. Describe the pathophysiology and clinical features of myasthenia gravis. (Linked to SLO 1.4)
5. Distinguish myasthenia gravis from Lambert-Eaton syndrome. (Linked to SLO 1.4)

1.4 Neurological Localisation

1.4.1 Principles of localisation

Neurological localisation is the process of identifying the anatomical site of a lesion from the pattern of the patient's neurological deficits. It is the most fundamental clinical skill in neurology and must be performed before any investigation is ordered. The principle is simple: every part of

the nervous system serves specific, predictable functions, so damage at any given site produces a predictable pattern of deficits.

The approach is systematic: determine which modalities are affected (motor, sensory, autonomic, or higher functions); define the distribution of the deficit (unilateral or bilateral, which limbs, dermatomal or peripheral nerve territory); match the distribution to known anatomy to identify the lesion level; then consider the time course (sudden: vascular; progressive: structural or degenerative; relapsing-remitting: inflammatory or demyelinating).

Fill in the blank: Neurological localisation identifies the _____ site of the lesion from the pattern of deficits. A sudden onset deficit suggests a _____ cause, while a relapsing-remitting course suggests an inflammatory cause.

1.4.2 Upper and lower motor neuron lesions

Upper motor neurons (UMN) originate in the motor cortex and travel in the corticospinal tract to the anterior horn cells; they are entirely within the CNS. UMN lesions produce: spastic weakness (increased tone), hyperreflexia (brisk tendon reflexes), a positive Babinski sign (upgoing plantar), clonus, and absent muscle wasting in early disease.

Lower motor neurons (LMN) are the anterior horn cells and their axons forming the peripheral nerves: the final common pathway to muscle. LMN lesions produce: flaccid weakness (reduced tone), areflexia (absent reflexes), muscle wasting (denervation atrophy), and fasciculations (visible spontaneous twitching of motor units from denervated muscle). The plantar response is flexor or absent.

Fill in the blank: UMN lesions cause _____ weakness with hyperreflexia and a Babinski sign. LMN lesions cause _____ weakness with areflexia, wasting, and fasciculations.

1.4.3 Common localisation patterns

Key patterns from cortex to muscle: cortical lesion (contralateral hemiplegia or monoplegia; cortical signs such as dysphasia if dominant, neglect if non-dominant; seizures); internal capsule (dense contralateral hemiplegia affecting face, arm, and leg equally: the pure motor stroke); brainstem (crossed deficit: ipsilateral cranial nerve signs plus contralateral long-tract signs); spinal cord (Brown-Sequard pattern or bilateral paraplegia with a sensory level); nerve root (dermatomal sensory loss and myotomal weakness with radicular pain).

At the peripheral level: a peripheral nerve lesion (mononeuropathy) causes weakness and sensory loss confined to the territory of that named nerve; a polyneuropathy causes distal, symmetrical, glove-and-stocking weakness and sensory loss because the longest fibres are affected first; an NMJ lesion causes fatigable weakness without sensory loss; a myopathy causes proximal weakness without sensory loss or fasciculations.

Fill in the blank: An internal capsule lesion causes a dense contralateral hemiplegia affecting face, arm, and leg _____ (the pure motor stroke). A polyneuropathy affects the longest fibres first, producing a _____ and stocking distribution.

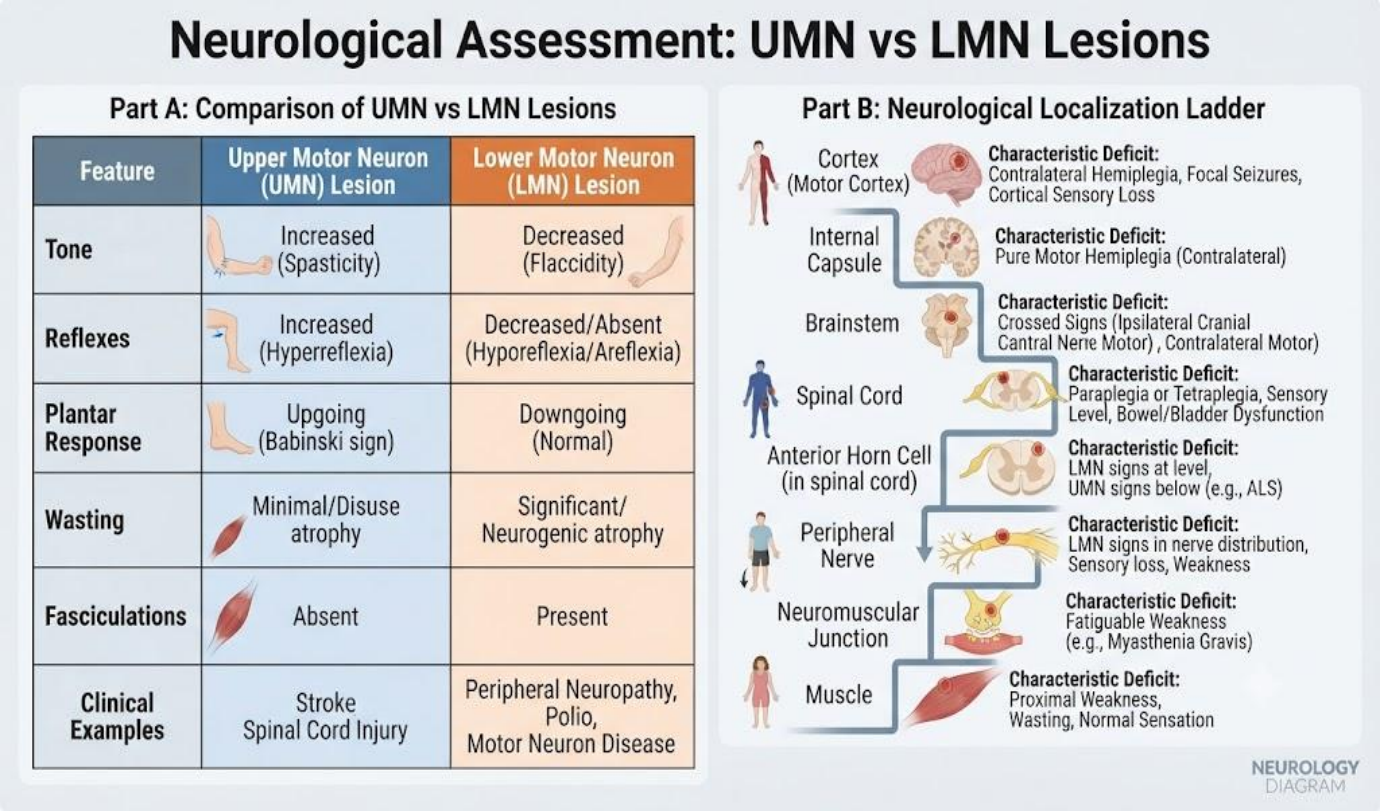


Figure 1.4: UMN versus LMN comparison and neurological localisation ladder

Pilot questions 1.4

1. Describe the systematic approach to neurological localisation. (Linked to SLO 1.5)
2. Distinguish UMN from LMN lesions across five clinical features. (Linked to SLO 1.5)
3. Describe the Brown-Sequard syndrome and its anatomical basis. (Linked to SLO 1.5)
4. Describe the deficits produced by a dominant hemisphere cortical lesion. (Linked to SLO 1.5)
5. Explain why a polyneuropathy produces a glove-and-stocking distribution. (Linked to SLO 1.5)

Series Summary

This Series established the neuroanatomical and physiological foundations of neurology. We began with nervous system organisation (CNS and PNS; somatic and autonomic; sympathetic and parasympathetic), neuron structure (dendrites, soma, axon, myelin, nodes of Ranvier), and glial cells (oligodendrocytes: CNS myelin; Schwann cells: PNS myelin; astrocytes: blood-brain barrier; microglia: immune defence). We described synaptic transmission and the major neurotransmitters: glutamate (excitatory), GABA (inhibitory, enhanced by benzodiazepines), acetylcholine (NMJ and ANS), dopamine (depleted in Parkinson's disease), serotonin (mood), and noradrenaline (arousal).

We then covered the CNS: four cerebral lobes and their functions; the crossed deficit pattern of brainstem lesions; DANISH cerebellar signs; and the three spinal cord tracts with their decussation levels and the Brown-Sequard pattern. We described spinal nerve formation, the sympathetic and parasympathetic divisions, and the NMJ with myasthenia gravis and Lambert-Eaton syndrome. Finally, we applied these foundations to neurological localisation, distinguishing UMN (spastic, hyperreflexic, Babinski) from LMN (flaccid, areflexic, wasting, fasciculations) lesions, and identifying the characteristic deficit patterns from cortex to muscle.

Glossary of Terms

- **Astrocyte:** a glial cell of the CNS that maintains the blood-brain barrier and provides metabolic support to neurons.
- **Babinski sign:** extension of the big toe on plantar stimulation; a pathological reflex indicating an upper motor neuron lesion.
- **Brown-Sequard syndrome:** spinal cord hemisection producing ipsilateral motor and dorsal column loss with contralateral pain and temperature loss below the lesion.
- **Corticospinal tract:** the main descending motor tract from the motor cortex to the anterior horn cells; crosses at the pyramidal decussation in the medulla.
- **Dermatome:** the area of skin supplied by a single dorsal spinal root; used to localise nerve root lesions.
- **Fasciculation:** a spontaneous visible twitch of a motor unit; a sign of lower motor neuron disease.
- **GABA:** gamma-aminobutyric acid; the main CNS inhibitory neurotransmitter; enhanced by benzodiazepines.
- **Myasthenia gravis:** autoimmune NMJ disorder: IgG antibodies against post-synaptic nicotinic ACh receptors causing fatigable weakness; treated with pyridostigmine and immunosuppression.

- **Neuromuscular junction:** the synapse between a motor neuron terminal and the motor end plate of a skeletal muscle fibre.
- **Oligodendrocyte:** a CNS glial cell that produces myelin around CNS axons; one cell myelinates up to 50 axons.
- **Saltatory conduction:** rapid action potential propagation along myelinated axons by jumping between nodes of Ranvier.
- **Spasticity:** velocity-dependent increased muscle tone from a UMN lesion, caused by loss of descending inhibitory control over spinal stretch reflexes.

Concept Check Questions [Series 1]

Objective questions

1. The brain and spinal cord together form the _____ nervous system. (Linked to SLO 1.1)
2. _____ produce myelin in the CNS; Schwann cells produce myelin in the PNS. (Linked to SLO 1.1)
3. The main CNS inhibitory neurotransmitter is _____, enhanced by benzodiazepines. (Linked to SLO 1.2)
4. A positive Babinski sign, increased tone, and hyperreflexia indicate a _____ motor neuron lesion. (Linked to SLO 1.5)
5. In myasthenia gravis, IgG antibodies target post-synaptic nicotinic ACh _____ at the NMJ. (Linked to SLO 1.4)

Short-answer questions

6. Describe the structure of a neuron, naming each part and its function. (Linked to SLO 1.1)
7. Describe the events at a chemical synapse. (Linked to SLO 1.2)
8. Describe the crossed deficit pattern of a brainstem lesion. (Linked to SLO 1.3)
9. Distinguish the sympathetic from the parasympathetic division. (Linked to SLO 1.4)
10. State the clinical features of a UMN lesion. (Linked to SLO 1.5)

Long-answer questions

11. Describe the functions of the four cerebral lobes and the deficits from Broca's and Wernicke's lesions. (Linked to SLO 1.3)
12. Describe the three spinal cord tracts and the Brown-Sequard syndrome. (Linked to SLO 1.3)
13. Compare UMN and LMN lesions across five clinical parameters. (Linked to SLO 1.5)
14. Describe the pathophysiology, features, and treatment of myasthenia gravis. (Linked to SLO 1.4)
15. Describe the localisation patterns from the cortex to the muscle. (Linked to SLO 1.5)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Central.
2. Oligodendrocytes.
3. GABA.
4. Upper.
5. Receptors.

Short-answer questions

6. Dendrites receive incoming signals; the soma contains the nucleus and integrates signals; the axon hillock is where action potentials are generated; the axon conducts action potentials away from the soma; the myelin sheath enables rapid saltatory conduction; nodes of Ranvier are gaps in myelin where the impulse is regenerated; axon terminals release neurotransmitter.
7. An action potential reaches the presynaptic terminal and opens voltage-gated calcium channels. Calcium influx triggers fusion of neurotransmitter-containing vesicles with the presynaptic membrane, releasing neurotransmitter into the synaptic cleft. The

neurotransmitter binds to postsynaptic receptors, producing excitation (EPSP) or inhibition (IPSP). The signal ends through enzymatic degradation or reuptake of the neurotransmitter.

8. A brainstem lesion damages the cranial nerve nucleus at the level of the lesion on the same side, producing ipsilateral cranial nerve signs. It also damages the long tracts at the same level, producing contralateral hemiplegia and hemisensory loss in the limbs. This crossed pattern (ipsilateral cranial nerve plus contralateral limb signs) is pathognomonic of a brainstem lesion.

9. Sympathetic: thoracolumbar origin (T1 to L2); short pre-ganglionic, long post-ganglionic fibres; noradrenaline at effectors (except sweat glands where acetylcholine); increases heart rate, dilates pupils. Parasympathetic: craniosacral origin (CN III, VII, IX, X, and S2 to S4); long pre-ganglionic, short post-ganglionic fibres; acetylcholine on muscarinic receptors; decreases heart rate, constricts pupils.

10. UMN lesion produces: spastic weakness (increased tone, clasp-knife rigidity), hyperreflexia (brisk tendon reflexes), positive Babinski sign (extensor plantar), clonus, and no significant muscle wasting in early disease. Fasciculations are absent.

Long-answer questions

11. **Basic response:** Frontal lobe: voluntary movement (primary motor cortex), executive function and personality (prefrontal cortex), and motor speech (Broca's area in dominant hemisphere: Broca's aphasia is non-fluent speech with intact comprehension). Parietal lobe: somatosensory cortex, language comprehension (Wernicke's area in dominant hemisphere: Wernicke's aphasia is fluent meaningless speech with impaired comprehension), and spatial attention (non-dominant: neglect if damaged). Temporal lobe: auditory processing and memory (hippocampus: anterograde amnesia if bilateral damage). Occipital lobe: primary visual cortex (contralateral homonymous hemianopia if damaged).

Value-added response: Broca's aphasia and Wernicke's aphasia can be distinguished at the bedside by assessing fluency and comprehension: non-fluent speech with intact comprehension points to Broca's; fluent meaningless speech with impaired comprehension points to Wernicke's. This distinction immediately localises the lesion to the frontal or temporal-parietal region.

12. **Basic response:** Dorsal columns: carry fine touch, vibration, and proprioception ipsilaterally to the medulla, where they cross. Spinothalamic tract: carries pain and temperature; fibres cross within 1 to 2 segments of entry through the anterior commissure, then ascend contralaterally. Corticospinal tract: crosses at the pyramidal

decussation in the medulla, then descends ipsilaterally in the lateral funiculus. Brown-Sequard syndrome from cord hemisection: ipsilateral motor loss and dorsal column loss (fine touch, vibration, proprioception) with contralateral pain and temperature loss below the lesion.

Value-added response: The dissociated sensory loss of Brown-Sequard syndrome (different sensory modalities lost on opposite sides) is unique to spinal cord lesions; no brain lesion produces this pattern, making it an immediate localising sign.

13. **Basic response:** UMN: increased tone (spasticity), hyperreflexia, positive Babinski sign, clonus present, no wasting in early disease, no fasciculations. LMN: decreased or absent tone (flaccidity), areflexia or hyporeflexia, flexor or absent plantar, no clonus, muscle wasting (denervation atrophy), fasciculations present.

Value-added response: Motor neuron disease is unique in producing simultaneous UMN and LMN signs: spasticity and hyperreflexia from corticospinal tract degeneration alongside wasting and fasciculations from anterior horn cell degeneration, all without sensory loss. This combination is the clinical hallmark of MND.

14. **Basic response:** Myasthenia gravis is an autoimmune NMJ disorder in which IgG antibodies target post-synaptic nicotinic ACh receptors, reducing their number through crosslinking, internalisation, and complement destruction. Clinical features: fatigable weakness worsening with activity and improving with rest; ocular muscles affected first (ptosis and diplopia); bulbar involvement (dysarthria and dysphagia); proximal limb weakness; respiratory crisis in severe disease. Treatment: pyridostigmine (acetylcholinesterase inhibitor), immunosuppression (prednisolone or azathioprine), thymectomy, plasma exchange or IVIg for crisis.

Value-added response: Myasthenic crisis (acute respiratory failure) is a medical emergency requiring ICU admission and ventilatory support. Precipitants include infection, surgery, and drugs impairing NMJ transmission such as aminoglycosides, fluoroquinolones, and beta-blockers.

15. **Basic response:** Cortex: contralateral hemiplegia or monoplegia; cortical signs (dysphasia if dominant, neglect if non-dominant); seizures. Internal capsule: dense contralateral hemiplegia affecting face, arm, and leg equally (pure motor stroke). Brainstem: crossed deficit (ipsilateral CN signs plus contralateral hemiplegia). Spinal cord: Brown-Sequard (hemisection) or bilateral paraplegia with sensory level (complete cord). Nerve root: dermatomal sensory loss, myotomal weakness, radicular pain. Peripheral nerve: mononeuropathy (named nerve territory) or polyneuropathy (distal symmetrical glove-and-stocking). NMJ: fatigable weakness, no sensory loss. Muscle: proximal weakness, no sensory loss, no fasciculations.

Value-added response: The internal capsule is clinically important because all motor fibres are compactly bundled here; a tiny lacunar infarct from small-vessel hypertensive disease can produce a dense hemiplegia equivalent to a large cortical stroke, highlighting why blood pressure control is the most important intervention for stroke prevention.

Answers to Pilot Questions [Series 1]

Part 1.1

1. The CNS consists of the brain and spinal cord (integration and processing centre). The PNS includes all neural tissue outside the CNS: cranial nerves, spinal nerves, ganglia, and plexuses. The PNS is subdivided into the somatic NS (voluntary motor and sensory) and the ANS (involuntary visceral regulation: sympathetic and parasympathetic divisions).
2. Dendrites (receive signals from other neurons). Cell body or soma (contains nucleus; integrates signals). Axon hillock (site of action potential generation). Axon (conducts action potentials away from soma). Myelin sheath (insulates axon; enables saltatory conduction). Nodes of Ranvier (gaps in myelin where action potential is regenerated). Axon terminals (release neurotransmitter into synaptic cleft).
3. Oligodendrocytes are CNS cells: one cell myelinates up to 50 different axons and does not support axon regeneration. Schwann cells are PNS cells: each cell myelinates a single internode of one axon and supports peripheral nerve regeneration after injury by providing a basement membrane tube for the regrowing axon.
4. An action potential reaches the presynaptic terminal, opening voltage-gated calcium channels. Calcium influx triggers fusion of neurotransmitter-containing vesicles with the presynaptic membrane, releasing neurotransmitter into the synaptic cleft. The neurotransmitter binds to postsynaptic receptors, producing excitation (EPSP) or inhibition (IPSP). The signal is terminated by enzymatic degradation or reuptake of the neurotransmitter.
5. Glutamate (main CNS excitatory neurotransmitter), GABA (main CNS inhibitory neurotransmitter; enhanced by benzodiazepines producing sedation and anticonvulsant effects), acetylcholine (NMJ and ANS; also in basal forebrain memory circuits), and dopamine (movement control via nigrostriatal pathway; reward via mesolimbic pathway; depleted in Parkinson's disease causing tremor, rigidity, and bradykinesia).

Part 1.2

1. Frontal lobe: voluntary movement (primary motor cortex), executive function (prefrontal cortex), motor speech production (Broca's area: dominant). Parietal lobe: somatosensory processing, language comprehension (Wernicke's area: dominant), spatial attention (non-dominant). Temporal lobe: auditory processing, memory (hippocampus). Occipital lobe: primary visual cortex (contralateral visual field). A lesion in each lobe causes deficits specific to its functions (hemiplegia, dysphasia, hemispatial neglect, anterograde amnesia, or homonymous hemianopia).
2. Broca's aphasia results from a lesion in Broca's area (dominant inferior frontal gyrus): speech is non-fluent and effortful with preserved comprehension; the patient understands but cannot produce fluent speech. Wernicke's aphasia results from a lesion in Wernicke's area (dominant posterior superior temporal gyrus): speech is fluent but meaningless with paraphasic errors; comprehension is severely impaired. The key distinction is fluency (non-fluent: Broca's; fluent: Wernicke's) and comprehension (intact: Broca's; impaired: Wernicke's).
3. A brainstem lesion simultaneously damages the cranial nerve nucleus at the level of the lesion (ipsilateral cranial nerve signs: for example ipsilateral facial palsy) and the long motor and sensory tracts passing through the same side of the brainstem (contralateral hemiplegia and hemisensory loss in the limbs). The anatomical basis: cranial nerve nuclei are at the same level as the lesion, while the long tracts have already crossed or will cross below it, producing deficits on opposite sides of the body.
4. DANISH: Dysdiadochokinesia (failure of rapid alternating movements), Ataxia (broad-based unsteady gait), Nystagmus (rhythmic involuntary eye movements, fast phase towards the lesion), Intention tremor (worsens as limb approaches target on finger-nose testing), Scanning speech or dysarthria (irregular explosive monotonous speech), Hypotonia (reduced muscle tone). All signs are ipsilateral to the cerebellar lesion.
5. Dorsal columns: carry fine touch, vibration, and proprioception ipsilaterally to the nucleus gracilis and cuneatus in the medulla, where they cross. Spinothalamic tract: carries pain and temperature; second-order fibres cross within 1 to 2 spinal segments through the anterior commissure then ascend contralaterally. Corticospinal tract: crosses at the pyramidal decussation in the medulla then descends ipsilaterally in the lateral funiculus. Brown-Sequard syndrome: ipsilateral motor and dorsal column loss with contralateral pain and temperature loss below the lesion.

Part 1.3

1. Each spinal nerve is formed by the union of the dorsal (posterior) root, which carries afferent sensory fibres from the dorsal root ganglion into the posterior horn, and the

ventral (anterior) root, which carries efferent motor fibres from the anterior horn cells to skeletal muscles. After leaving the intervertebral foramen, adjacent spinal nerves form plexuses (brachial, lumbosacral) that give rise to named peripheral nerves.

2. Sympathetic: thoracolumbar origin (T1 to L2); short pre-ganglionic and long post-ganglionic fibres; noradrenaline at effectors (acetylcholine at sweat glands); increases heart rate, dilates pupils, bronchodilates, inhibits digestion. Parasympathetic: craniosacral origin (CN III, VII, IX, X, and S2 to S4); long pre-ganglionic and short post-ganglionic fibres; acetylcholine on muscarinic receptors; decreases heart rate, constricts pupils, bronchoconstricts, promotes digestion.
3. An action potential reaches the motor nerve terminal and opens voltage-gated calcium channels. Calcium influx triggers fusion of ACh-containing vesicles with the presynaptic membrane. ACh is released into the synaptic cleft and binds to nicotinic receptors on the motor end plate, causing sodium influx and depolarisation. This generates a muscle action potential, triggering calcium release from the sarcoplasmic reticulum and muscle contraction. Acetylcholinesterase then degrades ACh, terminating the signal.
4. Pathophysiology: IgG antibodies target post-synaptic nicotinic ACh receptors at the NMJ, reducing their number through crosslinking, internalisation, and complement destruction. Clinical features: fatigable weakness worsening with activity and improving with rest; ocular muscles first (ptosis and diplopia); bulbar involvement (dysarthria and dysphagia); proximal limb weakness; respiratory crisis in severe cases. Treatment: pyridostigmine (acetylcholinesterase inhibitor), immunosuppression, thymectomy, plasma exchange or IVIg for crisis.
5. Myasthenia gravis: antibodies against post-synaptic nicotinic ACh receptors; fatigable weakness worsening with activity; ocular and bulbar involvement common; responds to pyridostigmine; thymic abnormality in 75 percent. Lambert-Eaton: antibodies against pre-synaptic voltage-gated calcium channels; proximal limb weakness with minimal ocular involvement; strength paradoxically improves briefly with repeated activity; strongly associated with small cell lung cancer; poor response to pyridostigmine.

Part 1.4

1. Systematic approach: (1) determine which modalities are affected (motor, sensory, autonomic, higher functions); (2) define the distribution (unilateral or bilateral, which limbs, dermatomal or named nerve or glove-and-stocking); (3) match distribution to anatomy to identify lesion level (cortex, internal capsule, brainstem, spinal cord, nerve root, peripheral nerve, NMJ, or muscle); (4) consider the time course (sudden: vascular;

progressive: structural or degenerative; relapsing-remitting: inflammatory or demyelinating).

2. UMN: increased tone (spasticity: clasp-knife rigidity), hyperreflexia (brisk tendon reflexes), positive Babinski sign (extensor plantar), clonus present, no significant muscle wasting in early disease, no fasciculations. LMN: reduced or absent tone (flaccidity), areflexia or hyporeflexia, flexor or absent plantar (normal), no clonus, muscle wasting (denervation atrophy), fasciculations present.
3. Brown-Sequard syndrome results from hemisection of the spinal cord. The corticospinal tract and dorsal columns have not yet crossed at the level of the lesion, so both are damaged on the same side, producing ipsilateral motor loss and dorsal column sensory loss (fine touch, vibration, proprioception) below the lesion. The spinothalamic fibres have already crossed within 1 to 2 segments of entry; the portion ascending on the same side as the lesion was carrying signals from the contralateral body, so contralateral pain and temperature is lost below the lesion.
4. A dominant hemisphere cortical lesion produces: contralateral hemiplegia or monoplegia (from motor cortex damage), contralateral hemisensory loss (from somatosensory cortex damage), Broca's aphasia (inferior frontal gyrus: non-fluent speech, intact comprehension), Wernicke's aphasia (posterior superior temporal gyrus: fluent meaningless speech, impaired comprehension), global aphasia (if both language areas are damaged), contralateral homonymous hemianopia (if the optic radiation is damaged), and focal seizures (from cortical irritation).
5. A polyneuropathy affects all peripheral nerves diffusely; the longest fibres (supplying the feet) have the greatest metabolic demands and the greatest distance for axoplasmic transport from the cell body, so they are most vulnerable. Deficits begin at the feet and spread proximally, producing distal, symmetrical glove-and-stocking sensory loss and weakness. A mononeuropathy, by contrast, affects one named peripheral nerve and produces weakness and sensory loss confined entirely to that nerve's territory.

References and Attributions [Series 1]

Textual References

- Kandel, E. R., Schwartz, J. H., Jessell, T. M., Siegelbaum, S. A. and Hudspeth, A. J. (2013). Principles of Neural Science (5th ed.). New York: McGraw-Hill.
- Fuller, G. and Manford, M. (2010). Neurology: An Illustrated Colour Text (4th ed.). Edinburgh: Churchill Livingstone.

- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- Longmore, M., Wilkinson, I., Baldwin, A. and Wallin, E. (2014). Oxford Handbook of Clinical Medicine (9th ed.). Oxford: Oxford University Press.

Figures, Plates, Tables, and Boxes

- Figure 1.1: Structure of a neuron and chemical synapse. AI-generated using the prompt: "Draw a labelled diagram with Part A showing a motor neuron and Part B showing a chemical synapse with a neurotransmitter reference table." Suggested OER search string: "neuron synapse labelled diagram neurotransmitters OER."
- Figure 1.2: Cerebral lobes and spinal cord cross-section with tracts. AI-generated using the prompt: "Two-part diagram: colour-coded lateral brain with four lobes and key functional areas; spinal cord cross-section with three colour-coded tracts and Brown-Sequard inset." Suggested OER search string: "cerebral lobes diagram spinal cord cross section tracts Brown-Sequard OER."
- Figure 1.3: Spinal nerve formation and the neuromuscular junction. AI-generated using the prompt: "Two-part diagram: spinal nerve formation with dermatome map; NMJ with ACh vesicles, calcium channels, nicotinic receptors, and myasthenia gravis versus Lambert-Eaton inset." Suggested OER search string: "spinal nerve dermatomes neuromuscular junction myasthenia gravis Lambert-Eaton OER."
- Figure 1.4: UMN versus LMN comparison and neurological localisation ladder. AI-generated using the prompt: "Two-part diagram: UMN versus LMN comparison table; vertical localisation ladder from cortex to muscle with characteristic deficit at each level." Suggested OER search string: "UMN LMN comparison neurological localisation patterns diagram OER."

Course code: MED 408

Course title: Neurology I

Course credit load: 2 Units

Series 2: Neurological Patient Evaluation

- **Part 2.1: History Taking in Neurology**
    - Sub Part 2.1.1: The neurological history
    - Sub Part 2.1.2: Common neurological complaints
    - Sub Part 2.1.3: Past history, family history, and social history
- **Part 2.2: The Neurological Examination**
    - Sub Part 2.2.1: General inspection and higher mental functions
    - Sub Part 2.2.2: The motor examination
    - Sub Part 2.2.3: The sensory and coordination examination
- **Part 2.3: Neurological Investigations**
    - Sub Part 2.3.1: Neuroimaging
    - Sub Part 2.3.2: Electrodiagnostic tests
    - Sub Part 2.3.3: Cerebrospinal fluid analysis
- **Part 2.4: The Glasgow Coma Scale and Consciousness**
    - Sub Part 2.4.1: Consciousness and its assessment
    - Sub Part 2.4.2: The Glasgow Coma Scale
    - Sub Part 2.4.3: The unconscious patient

Introduction

The ability to evaluate a neurological patient systematically is one of the most demanding yet rewarding skills in clinical medicine. A carefully taken history and a structured neurological examination provide the raw material from which the clinician constructs a differential diagnosis and identifies the site of the lesion, often before a single investigation has been ordered. In

Nigeria, where advanced investigations may not always be immediately available, these bedside skills are particularly vital.

In this Series, we shall begin with history taking, examining what makes a neurological history distinctive and how to extract the key information from each presenting complaint. We will then walk through the neurological examination systematically, from general inspection and higher mental functions through to the motor, sensory, and coordination examination. Moving on, we will survey the major neurological investigations available in Nigerian practice. Finally, we will address the assessment of consciousness, culminating in the Glasgow Coma Scale and the approach to the unconscious patient.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 2.1:** take a structured neurological history, identifying key features of common neurological complaints,
- **SLO 2.2:** perform a systematic neurological examination, including assessment of higher mental functions, the motor system, and the sensory system,
- **SLO 2.3:** select and interpret appropriate neurological investigations including neuroimaging, electrodiagnostic tests, and CSF analysis, and
- **SLO 2.4:** assess the level of consciousness using the Glasgow Coma Scale and describe the approach to the unconscious patient.

2.1 History Taking in Neurology

2.1.1 The neurological history

A neurological history follows the same broad structure as any medical history, but the detail required is greater and the framing of questions is more precise. You must establish the presenting complaint and then characterise it fully: the onset (sudden onset suggests a vascular cause such as stroke; gradual onset suggests a structural or degenerative process; episodic symptoms suggest epilepsy, migraine, or transient ischaemic attacks), the duration, the progression (improving, static, or worsening), and any precipitating, aggravating, or relieving factors. The time course alone often narrows the differential diagnosis considerably.

Always ask about associated symptoms, since neurological diseases rarely affect a single function in isolation. A patient presenting with weakness should be asked about sensory symptoms, visual changes, sphincter disturbance, and cognitive change, as the combination of

these additional symptoms will point to the level of the lesion. Also enquire about previous similar episodes, since a relapsing-remitting pattern is the hallmark of multiple sclerosis, while a stepwise deterioration is characteristic of vascular dementia.

Fill in the blank: A sudden onset neurological deficit suggests a _____ cause, while a relapsing-remitting course is the hallmark of _____. Always enquire about _____ symptoms, since neurological diseases rarely affect a single function in isolation.

2.1.2 Common neurological complaints

The most common neurological presenting complaints are headache, dizziness, weakness, sensory disturbance, visual symptoms, speech difficulty, loss of consciousness, and memory problems. Each must be characterised in detail. Headache requires assessment of its site, onset (thunderclap onset suggests subarachnoid haemorrhage), character, severity, duration, associated features (nausea, photophobia, neck stiffness, focal neurology), and triggers. Dizziness must be distinguished into vertigo (a false sense of rotational movement, suggesting vestibular disease) and presyncope (a feeling of impending faint, suggesting cardiovascular or metabolic causes).

Weakness should be characterised by its distribution (proximal versus distal, unilateral versus bilateral), onset, and progression. Sensory symptoms include numbness, tingling (paraesthesiae), and pain; their distribution in a dermatomal, peripheral nerve, or glove-and-stocking pattern will immediately point towards the level of the lesion. Speech difficulty should be distinguished into dysphasia (a language disorder from cortical damage) and dysarthria (a motor difficulty with speech articulation from brainstem, cerebellar, or peripheral nerve damage).

Fill in the blank: Thunderclap onset headache should raise suspicion of _____. Vertigo is a false sense of _____ movement and suggests vestibular disease, whereas presyncope suggests a _____ or metabolic cause.

2.1.3 Past history, family history, and social history

The past medical history in neurology should specifically enquire about previous episodes of neurological symptoms (even if minor and self-resolving), head injury, epilepsy, hypertension (the most important risk factor for stroke in Nigeria), diabetes mellitus (diabetic peripheral neuropathy is very common), HIV infection (HIV neurological complications include dementia, peripheral neuropathy, and opportunistic CNS infections), tuberculosis (TB meningitis is a major cause of neurological morbidity in Nigeria), sickle cell disease, and previous malaria.

The family history is important in conditions with a genetic basis, including hereditary neuropathies, familial epilepsy, Huntington's disease, and familial stroke syndromes. The drug history must cover all current medications (many drugs cause neurological side effects: isoniazid causes peripheral neuropathy; antiretrovirals can cause neuropathy; antiepileptics interact with each other) and traditional herbal remedies. The social history should document alcohol and substance use, occupation (exposure to neurotoxic substances), and functional capacity, including the ability to work, drive, and care for dependants.

Fill in the blank: The most important cardiovascular risk factor for stroke in Nigeria is _____. A family history of progressive chorea and dementia should raise suspicion of _____ disease.

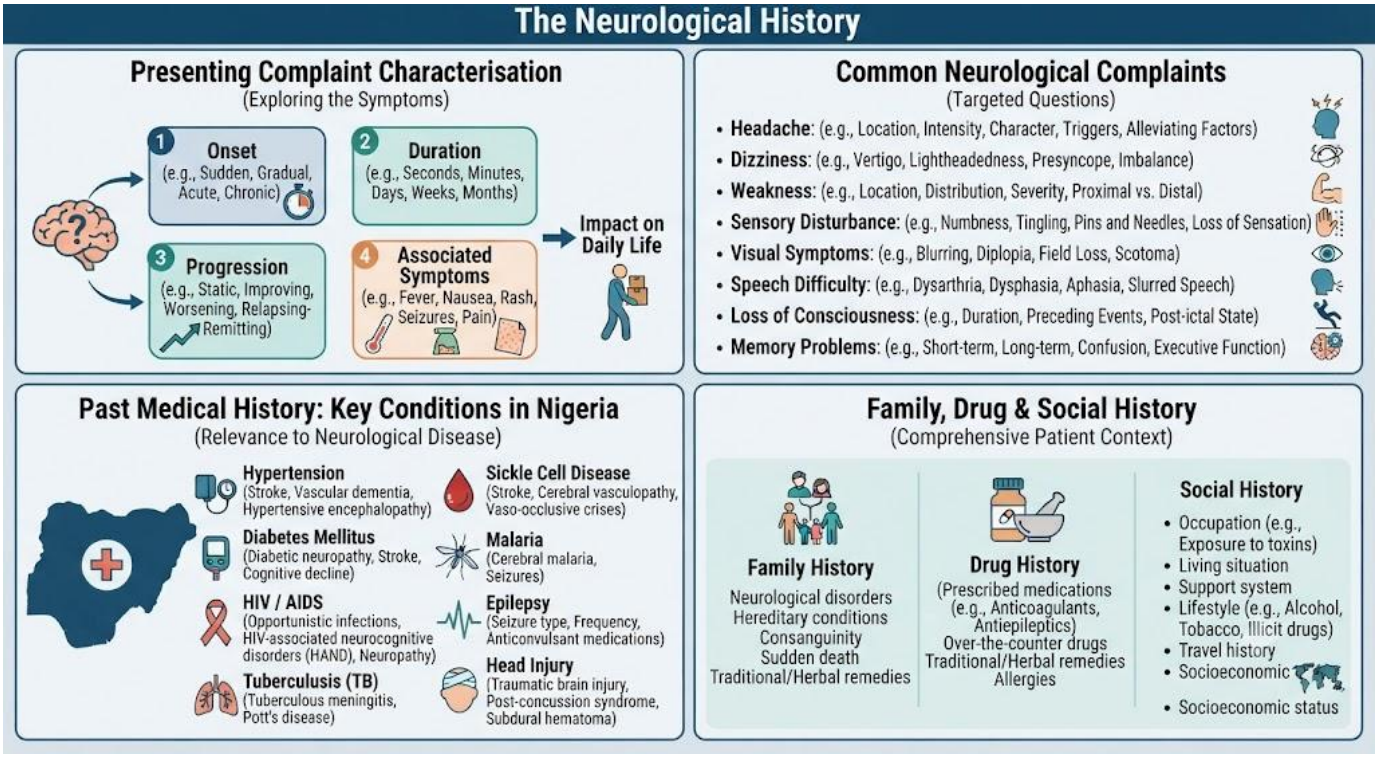


Figure 2.1: Structure of the neurological history

Pilot questions 2.1

1. Describe how the time course of a neurological symptom helps narrow the differential diagnosis. (Linked to SLO 2.1)
2. Distinguish vertigo from presyncope and state what each suggests diagnostically. (Linked to SLO 2.1)
3. Distinguish dysphasia from dysarthria and state the likely site of the lesion in each. (Linked to SLO 2.1)
4. State five past medical conditions that are particularly important in the neurological history in Nigeria. (Linked to SLO 2.1)
5. Explain why the drug history is especially important in Nigerian neurological patients. (Linked to SLO 2.1)

2.2 The Neurological Examination

2.2.1 General inspection and higher mental functions

The neurological examination begins before you even touch the patient. Observe the patient's general appearance, nutritional status, and any obvious asymmetry, abnormal posture, or involuntary movements. Note the level of consciousness and the patient's ability to engage with you. Assess speech immediately during history taking: is it fluent or non-fluent (Broca's aphasia)? Is comprehension intact? Is articulation normal or slurred (dysarthria)?

Higher mental function testing assesses orientation (to time, place, and person), memory (registration: repeat three words; short-term recall: repeat after five minutes; long-term: historical facts), attention and concentration (serial sevens: subtract 7 from 100 repeatedly; spelling WORLD backwards), language (naming, repetition, reading, and writing), visuospatial function (copy a clock face or a complex figure), and executive function (abstract reasoning). The Mini-Mental State Examination (MMSE, scored out of 30) and the Montreal Cognitive Assessment (MoCA) are standardised bedside tools; a score below 24 on the MMSE suggests cognitive impairment.

Fill in the blank: During the neurological examination, speech fluency and comprehension are assessed to distinguish _____ from dysarthria. An MMSE score below _____ out of 30 suggests cognitive impairment.

2.2.2 The motor examination

The motor examination follows a systematic sequence: inspection (wasting, fasciculations, abnormal posture, and involuntary movements), tone (increased in UMN lesions: spasticity; reduced in LMN and cerebellar lesions: flaccidity; rigidity in Parkinson's disease: uniform cogwheel or lead-pipe resistance throughout the range of movement), power (tested in all muscle groups and graded on the MRC scale: 0 to 5), and reflexes (tendon reflexes graded 0 to 4+, and the plantar response: upgoing in UMN lesions).

The MRC power scale is: 0 (no movement), 1 (flicker of contraction), 2 (movement with gravity eliminated), 3 (movement against gravity but not resistance), 4 (movement against some resistance), and 5 (normal power). Always test at least the key muscle groups in each limb: shoulder abduction (C5), elbow flexion (C5/C6), wrist extension (C7), finger extension (C7), finger abduction (T1) in the upper limb; and hip flexion (L2/L3), knee extension (L3/L4), ankle dorsiflexion (L4/L5), and plantar flexion (S1/S2) in the lower limb.

Fill in the blank: On the MRC power scale, a grade of _____ indicates movement against gravity but not resistance, and a grade of _____ indicates normal power. Cogwheel rigidity is characteristic of _____ disease.

2.2.3 The sensory and coordination examination

Test each sensory modality separately, since the dorsal columns and the spinothalamic tract may be selectively damaged. Light touch (cotton wool: tests dorsal columns and spinothalamic tract), pinprick (tests spinothalamic tract for pain and temperature), vibration sense (128 Hz tuning fork placed on a bony prominence: tests dorsal columns), proprioception or joint position sense (tests dorsal columns), and two-point discrimination and graphaesthesia (cortical sensory functions). Map out the boundaries of any sensory loss systematically, working from the area of loss towards normal, to define a dermatomal, peripheral nerve, or long-tract pattern.

Coordination is tested by finger-nose testing (ask the patient to alternately touch their nose and your finger: intention tremor and past-pointing indicate cerebellar disease), heel-shin testing (in the lower limb), rapid alternating movements (dysdiadochokinesia in cerebellar disease), and gait assessment (broad-based ataxic gait in cerebellar disease; high-stepping gait in foot drop from L4/L5 or common peroneal nerve lesion; shuffling festinant gait in Parkinson's disease; scissor gait in bilateral UMN lesions; sensory ataxia worsening with eyes closed: Romberg's sign positive in dorsal column disease).

Fill in the blank: Proprioception and vibration sense test the integrity of the _____ columns. A positive Romberg's sign (unsteadiness worsening with eyes closed) indicates _____ ataxia from dorsal column disease.

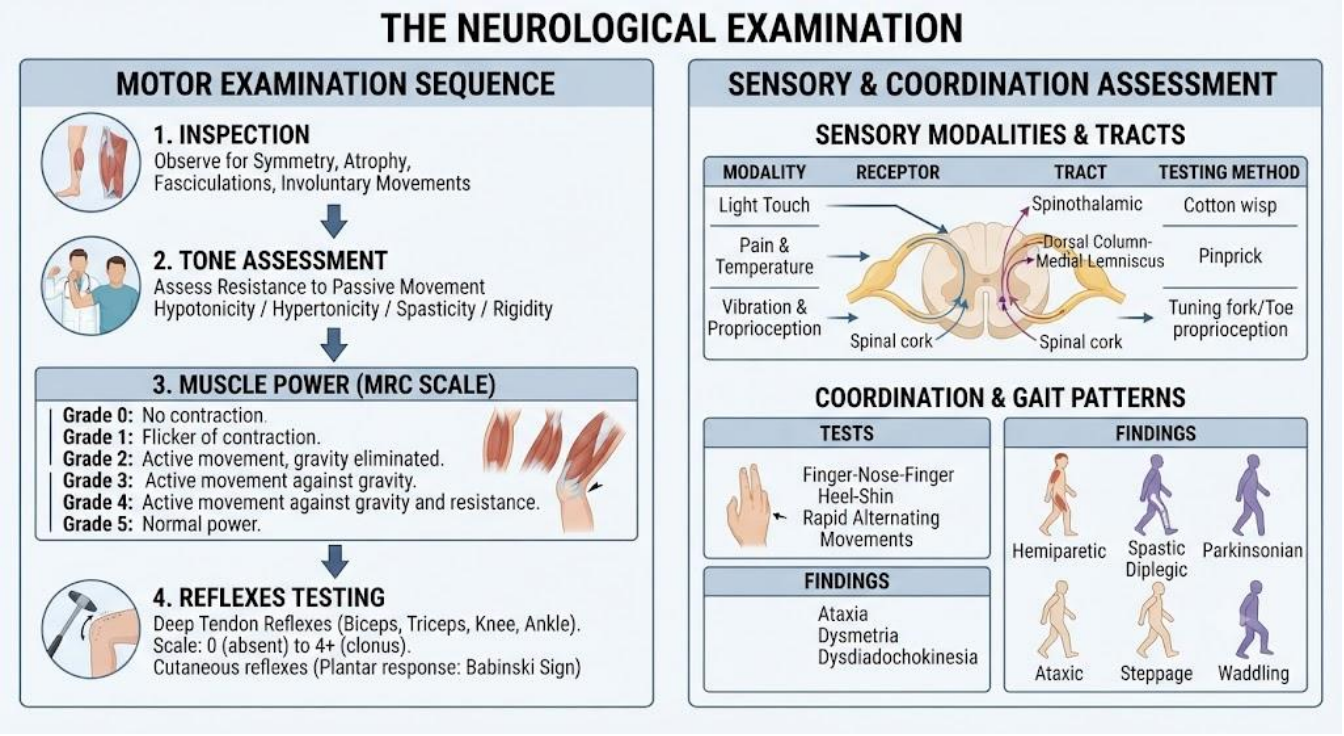


Figure 2.2: The motor and sensory-coordination neurological examination

Pilot questions 2.2

1. Describe what you observe during general inspection at the start of a neurological examination. (Linked to SLO 2.2)
2. Describe the MRC power scale and state what each grade means. (Linked to SLO 2.2)
3. Explain the difference between spasticity, flaccidity, and cogwheel rigidity and state the lesion responsible for each. (Linked to SLO 2.2)
4. Describe how you would test each major sensory modality and state which tract each tests. (Linked to SLO 2.2)
5. Describe the gait abnormality in cerebellar disease and in Parkinson's disease. (Linked to SLO 2.2)

2.3 Neurological Investigations

2.3.1 Neuroimaging

Computed tomography (CT) of the brain is the most widely available neuroimaging modality in Nigerian hospitals and is the first-line investigation in most acute neurological emergencies. A non-contrast CT scan immediately distinguishes haemorrhagic stroke (hyperdense blood: bright white) from ischaemic stroke (hypodense area: dark, but may be normal in the first few hours), identifies acute subarachnoid haemorrhage (blood in the subarachnoid space: hyperdense), shows cerebral oedema, mass lesions, hydrocephalus, and depressed skull fractures. CT is fast, widely available, and safe in patients with metallic implants.

Magnetic resonance imaging (MRI) of the brain and spine provides superior soft tissue resolution and is more sensitive than CT for most neurological conditions, including early ischaemic stroke, demyelinating plaques (multiple sclerosis), posterior fossa lesions, spinal cord pathology, and small cortical or subcortical lesions. Diffusion-weighted imaging (DWI) is the most sensitive sequence for acute ischaemic stroke (bright signal within minutes of onset). MRI is contraindicated in patients with certain metallic implants (some cardiac pacemakers, cochlear implants, and ferromagnetic foreign bodies) and requires patient cooperation due to the longer scan time.

Fill in the blank: A hyperdense area on non-contrast CT of the brain indicates _____, while a hypodense area suggests _____. _____ imaging (DWI) is the most sensitive MRI sequence for detecting acute ischaemic stroke.

2.3.2 Electrodiagnostic tests

The electroencephalogram (EEG) records the electrical activity of the cerebral cortex via scalp electrodes and is the most important investigation in epilepsy. It identifies epileptiform discharges (spikes and spike-and-wave complexes) and classifies seizure types (generalised versus focal). A normal EEG does not exclude epilepsy, since discharges may not occur during the recording period; sleep-deprived EEG and ambulatory (24-hour) EEG increase the diagnostic yield. EEG is also used to assess encephalopathy (diffuse slowing) and to confirm brain death (electrocerebral silence).

Nerve conduction studies (NCS) measure the conduction velocity and amplitude of electrical signals in peripheral nerves and are essential for diagnosing peripheral neuropathies and distinguishing axonal from demyelinating neuropathies: in demyelinating neuropathies (such as Guillain-Barre syndrome), conduction velocity is markedly slowed; in axonal neuropathies (such as diabetic neuropathy), amplitude is reduced with relatively preserved velocity. Electromyography (EMG) records the electrical activity of muscle fibres at rest and during voluntary contraction; it detects denervation (fibrillations and positive sharp waves at rest) and myopathic changes.

Fill in the blank: The EEG investigation of choice in epilepsy identifies epileptiform discharges of _____ and spike-and-wave complexes. In a _____ neuropathy such as Guillain-Barre syndrome, nerve conduction velocity is markedly slowed.

2.3.3 Cerebrospinal fluid analysis

Lumbar puncture (LP) is performed to obtain CSF for analysis. The patient is positioned in the lateral decubitus or sitting position, and a spinal needle is inserted at the L3/L4 or L4/L5 interspace (below the conus medullaris at L1/L2, so the cord is not at risk). The opening pressure is measured (normal: 6 to 20 cmH2O in the lateral decubitus position), and CSF is collected for: appearance (clear and colourless: normal; turbid: bacterial meningitis; xanthochromic: yellow-stained from bilirubin: subarachnoid haemorrhage), protein (normal: 0.15 to 0.45 g/L), glucose (normal: above two-thirds of plasma glucose), and cell count (normal: fewer than 5 white cells per microlitre, all lymphocytes).

Lumbar puncture must not be performed if there are signs of raised intracranial pressure (papilloedema, deteriorating consciousness, or focal neurological signs suggesting a mass lesion) without first performing CT of the brain to exclude a space-occupying lesion, as LP in this setting can cause fatal brainstem herniation. Additional CSF investigations include Gram stain and culture (bacterial meningitis), India ink stain and CrAg LFA (cryptococcal meningitis), acid-fast bacillus stain and GeneXpert (TB meningitis), viral PCR (viral encephalitis), and oligoclonal bands (multiple sclerosis).

Fill in the blank: Lumbar puncture is performed at the _____ or L4/L5 interspace, below the conus medullaris. _____ of the CSF (yellow staining) indicates previous subarachnoid haemorrhage.

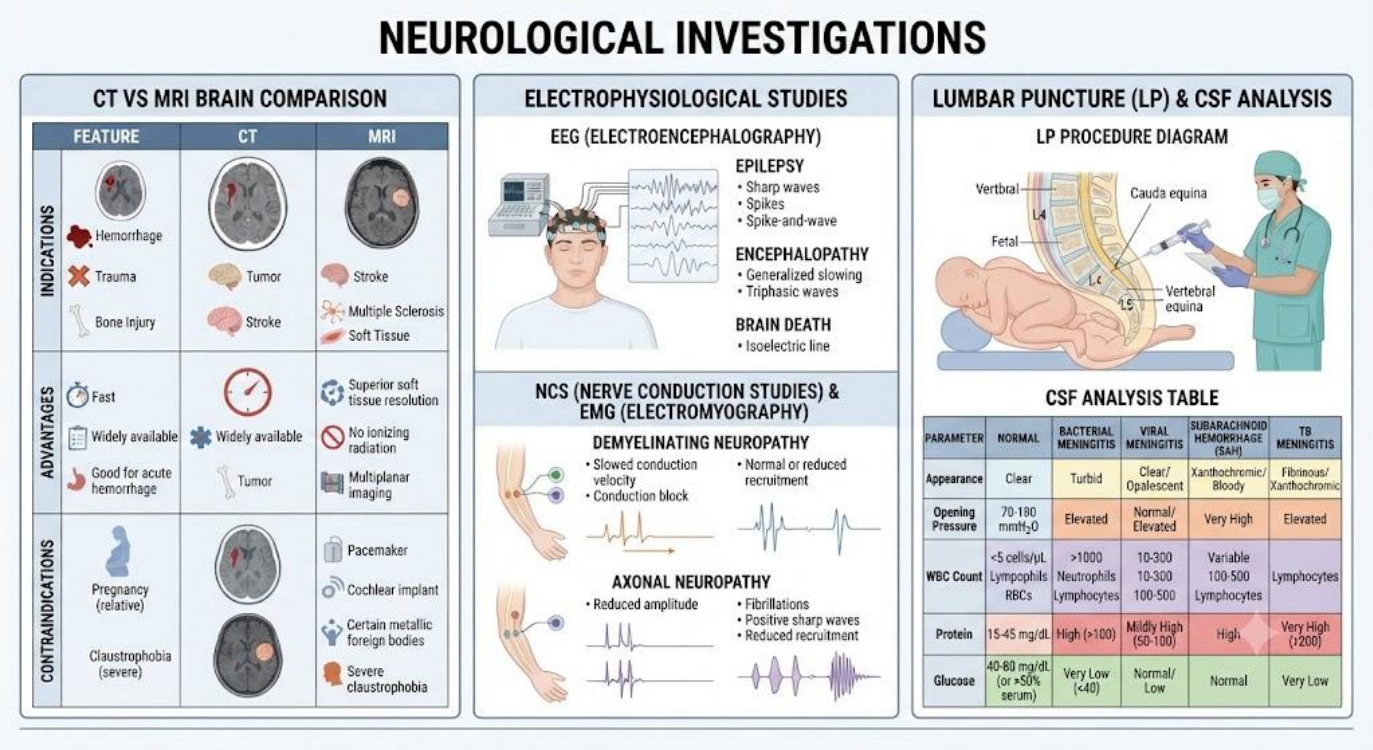


Figure 2.3: Neurological investigations: CT and MRI, electrodiagnostic tests, and CSF analysis

Pilot questions 2.3

1. Describe the findings on CT brain in haemorrhagic stroke and in subarachnoid haemorrhage. (Linked to SLO 2.3)
2. State the main indication for EEG and describe what it can show. (Linked to SLO 2.3)
3. Distinguish a demyelinating from an axonal peripheral neuropathy on nerve conduction studies. (Linked to SLO 2.3)
4. Describe the procedure for lumbar puncture and state the contraindication to performing it. (Linked to SLO 2.3)
5. Describe the CSF findings in bacterial meningitis and distinguish them from viral meningitis. (Linked to SLO 2.3)

2.4 The Glasgow Coma Scale and Consciousness

2.4.1 Consciousness and its assessment

Consciousness is the state of awareness of self and environment. It depends on two components: arousal (the level of wakefulness, maintained by the reticular activating system in the brainstem

and thalamus) and content (the cognitive functions subserved by the cerebral cortex). Disorders of consciousness range along a spectrum: full consciousness, confusion or delirium (arousal present but content disturbed), obtundation (reduced alertness requiring stimulation), stupor (arousal only with vigorous stimulation), and coma (no arousal even with painful stimulation). The causes of impaired consciousness are classified as structural (brainstem or bilateral hemisphere lesions: stroke, haemorrhage, tumour, hydrocephalus) or metabolic and toxic (hypoglycaemia, hepatic encephalopathy, hypoxia, uraemia, drug overdose, septic encephalopathy).

Bedside assessment of consciousness should document: the spontaneous level of arousal, the best verbal response, and the best motor response to stimulation. Always check the blood glucose immediately in any patient with impaired consciousness, since hypoglycaemia is a rapidly reversible cause that must not be missed. Pupillary responses (size, equality, and reactivity to light) give critical information: fixed dilated pupils indicate midbrain compression or herniation; pinpoint pupils indicate pontine lesion or opioid toxicity; asymmetric pupils (anisocoria) indicate third nerve compression from uncal herniation.

Fill in the blank: Consciousness depends on two components: _____ (maintained by the brainstem reticular activating system) and _____ (subserved by the cerebral cortex). Fixed dilated pupils in a comatose patient indicate _____ compression or herniation.

2.4.2 The Glasgow Coma Scale

The Glasgow Coma Scale (GCS) is the most widely used standardised tool for quantifying the level of consciousness. It scores three components independently: Eye opening (E: 1 to 4), Verbal response (V: 1 to 5), and Motor response (M: 1 to 6), giving a total score of 3 to 15. Eye opening: 4 (spontaneous), 3 (to voice), 2 (to pain), 1 (none). Verbal response: 5 (orientated), 4 (confused), 3 (inappropriate words), 2 (incomprehensible sounds), 1 (none). Motor response: 6 (obeys commands), 5 (localises pain), 4 (withdrawal from pain), 3 (abnormal flexion: decorticate), 2 (extension: decerebrate), 1 (none).

A GCS of 13 to 15 indicates mild impairment, 9 to 12 moderate impairment, and 8 or below severe impairment. A GCS of 8 or below is the threshold that indicates the need for airway protection (endotracheal intubation), since the patient can no longer protect their own airway. In clinical documentation, the GCS should always be recorded as the three component scores (for example E3 V4 M5 = 12) rather than just the total, since the motor component in particular carries the most prognostic information.

Fill in the blank: The three components of the GCS are _____ opening, verbal response, and _____ response. A GCS of _____ or below indicates the need for airway protection.

2.4.3 The unconscious patient

The management of the unconscious patient begins with the ABCDE approach. Airway: position the patient in the recovery position (lateral decubitus) to protect the airway; use airway adjuncts (oropharyngeal airway) or endotracheal intubation if GCS is 8 or below. Breathing: assess

respiratory rate, pattern (Cheyne-Stokes breathing indicates bilateral hemisphere or diencephalic dysfunction; central neurogenic hyperventilation indicates midbrain damage; ataxic breathing indicates medullary damage), and oxygen saturation; give high-flow oxygen. Circulation: obtain IV access, take blood for glucose, FBC, urea and electrolytes, liver function tests, blood cultures, and toxicology screen; give IV dextrose immediately if blood glucose is below 3 mmol/L.

Neurological assessment in the unconscious patient: document the GCS, pupillary responses (size and reactivity), eye position and movements (conjugate gaze deviation towards the lesion in a cortical stroke; deviation away from the lesion in a pontine stroke), and best motor response to pain (localisation: cortical; decorticate flexion: above the midbrain; decerebrate extension: midbrain or pontine level; no response: medulla or lower). Urgent CT brain must be performed once the patient is stabilised to identify the cause. In the Nigerian context, always exclude malaria (blood film and RDT) and bacterial meningitis (LP after CT if safe) as treatable reversible causes.

Fill in the blank: In an unconscious patient, a GCS of 8 or below requires _____ protection with intubation. Conjugate gaze deviation _____ the side of the lesion indicates a cortical stroke, while deviation _____ the lesion suggests a pontine lesion.

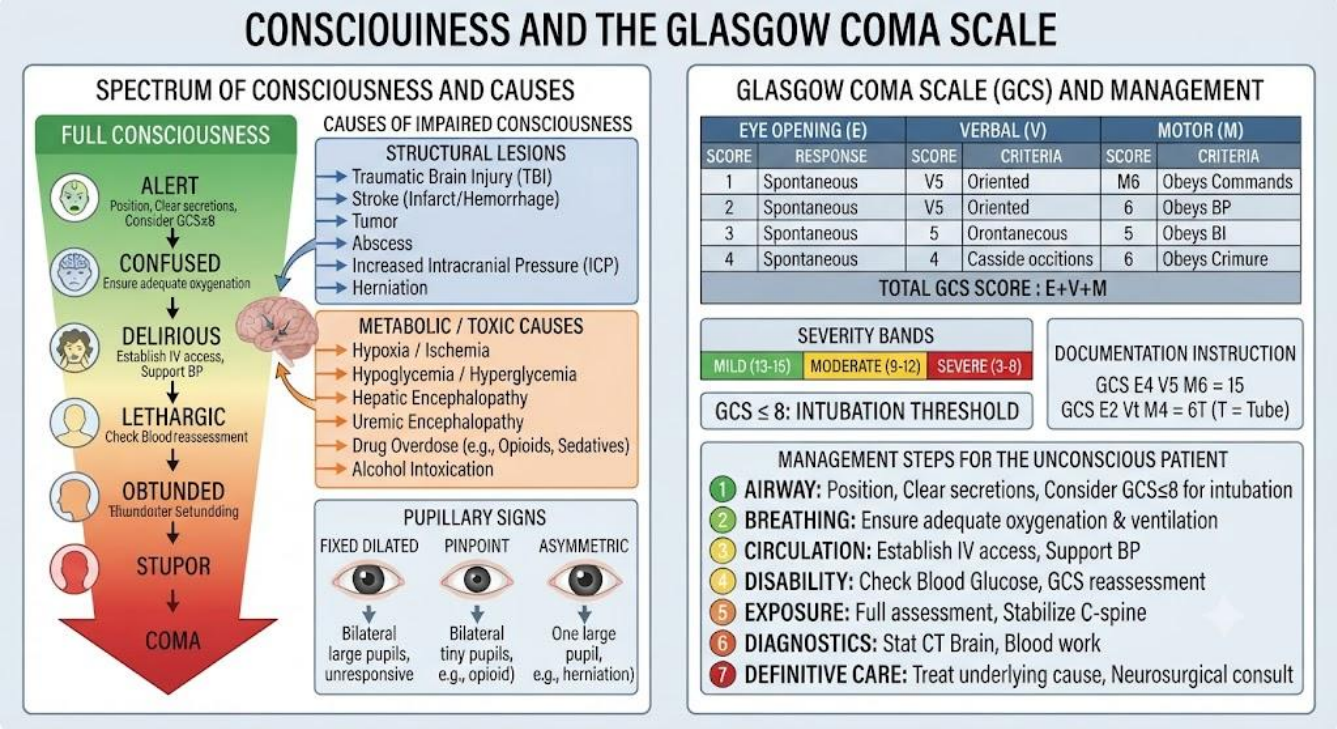


Figure 2.4: The spectrum of consciousness, Glasgow Coma Scale, and management of the unconscious patient

Pilot questions 2.4

1. Describe the two components of consciousness and the spectrum of impaired consciousness. (Linked to SLO 2.4)

2. Describe what pinpoint pupils and fixed dilated pupils indicate in an unconscious patient. (Linked to SLO 2.4)
3. Describe the three components of the GCS and state the score for each. (Linked to SLO 2.4)
4. State the GCS threshold for airway protection and explain why this threshold is used. (Linked to SLO 2.4)
5. Describe the initial management of an unconscious patient using the ABCDE approach. (Linked to SLO 2.4)

Series Summary

This Series developed the clinical skills needed to evaluate a neurological patient. We began with history taking: characterising the presenting complaint by onset (sudden: vascular; gradual: structural; episodic: epilepsy or migraine), distinguishing common complaints (vertigo versus presyncope; dysphasia versus dysarthria; proximal versus distal weakness), and identifying the key past medical and social history features relevant to Nigerian practice (hypertension, diabetes, HIV, TB, sickle cell disease). We then described the neurological examination: general inspection and higher mental function testing (MMSE); the motor examination sequence (inspection, tone, power by MRC scale, reflexes and plantar response); and sensory and coordination testing (dorsal column modalities, spinothalamic modalities, cerebellar coordination tests, and characteristic gait patterns).

We surveyed the major neurological investigations: CT brain (first-line in acute emergencies: haemorrhage is hyperdense, ischaemia is hypodense), MRI brain (superior soft tissue resolution; DWI for early ischaemic stroke), EEG (epilepsy and encephalopathy and brain death), NCS and EMG (demyelinating versus axonal neuropathy), and LP with CSF analysis (bacterial meningitis: turbid, very low glucose, neutrophilic pleocytosis; viral meningitis: clear, normal glucose, lymphocytic pleocytosis; subarachnoid haemorrhage: xanthochromic). Finally, we addressed consciousness and the GCS: the two components of consciousness (arousal and content); the spectrum from confusion to coma; pupillary signs; the three-component GCS (E, V, M: total 3 to 15); GCS 8 or below as the threshold for airway protection; and the ABCDE management of the unconscious patient in the Nigerian context.

Glossary of Terms

- **Coma:** a state of unarousable unresponsiveness in which the patient does not open eyes, speak, or obey commands even with painful stimulation; GCS score of 8 or below.

- **Decerebrate posturing:** a motor response to pain in the unconscious patient consisting of extension and internal rotation of all four limbs and neck; indicates damage at the level of the midbrain or pons.
- **Decorticate posturing:** a motor response to pain in the unconscious patient consisting of flexion of the arms and extension of the legs; indicates damage above the level of the midbrain (cortex or internal capsule).
- **Diffusion-weighted imaging (DWI):** an MRI sequence that detects restriction of water molecule diffusion in infarcted tissue; the most sensitive sequence for acute ischaemic stroke, showing bright signal within minutes of infarct onset.
- **Dysarthria:** impaired articulation of speech due to weakness, incoordination, or spasticity of the muscles of speech production; a motor problem without impaired language comprehension or formulation.
- **Dysphasia:** a disorder of language (comprehension or production) resulting from damage to the cortical language areas; distinct from dysarthria, which is a motor articulation problem.
- **Glasgow Coma Scale (GCS):** a standardised 15-point scale for assessing level of consciousness; scores eye opening (1 to 4), verbal response (1 to 5), and motor response (1 to 6); a score of 8 or below indicates severe impairment and the need for airway protection.
- **Lumbar puncture:** a procedure in which a needle is inserted into the lumbar subarachnoid space (L3/L4 or L4/L5) to measure CSF pressure and obtain CSF for analysis; contraindicated when raised intracranial pressure is suspected without prior CT brain.
- **MRC power scale:** a standardised six-point scale (0 to 5) for grading muscle strength; 0 is no movement, 3 is movement against gravity but not resistance, and 5 is normal power.
- **Nerve conduction studies (NCS):** electrodiagnostic tests that measure the velocity and amplitude of electrical signals in peripheral nerves; used to diagnose peripheral neuropathies and distinguish demyelinating (slowed velocity) from axonal (reduced amplitude) patterns.
- **Vertigo:** an illusion of rotational movement of self or environment, indicating a disturbance of the vestibular system (peripheral: labyrinthine disorder; or central: brainstem or cerebellar disease).
- **Xanthochromia:** yellow discolouration of CSF due to bilirubin from haemoglobin degradation; indicates subarachnoid haemorrhage when blood has been present in the CSF for more than two hours.

Concept Check Questions [Series 2]

Objective questions

1. A sudden onset neurological deficit suggests a _____ cause, while a gradual progressive onset suggests a structural or degenerative process. (Linked to SLO 2.1)
2. On the MRC power scale, a grade of _____ indicates movement against gravity but not resistance. (Linked to SLO 2.2)
3. On CT brain, fresh blood appears _____ (hyperdense), while ischaemic infarction appears hypodense (dark). (Linked to SLO 2.3)
4. The three components of the GCS are eye opening, verbal response, and _____ response. (Linked to SLO 2.4)
5. A GCS of _____ or below indicates the need for airway protection by endotracheal intubation. (Linked to SLO 2.4)

Short-answer questions

6. Distinguish vertigo from presyncope in terms of symptoms and underlying cause. (Linked to SLO 2.1)
7. Distinguish spasticity from cogwheel rigidity in terms of clinical features and underlying lesion. (Linked to SLO 2.2)
8. Describe the procedure for lumbar puncture and state the main contraindication. (Linked to SLO 2.3)
9. Describe the two components of consciousness and state how each can be impaired. (Linked to SLO 2.4)
10. Describe what pupillary abnormalities indicate in an unconscious patient. (Linked to SLO 2.4)

Long-answer questions

11. Describe the key features that should be elicited when a patient presents with headache. (Linked to SLO 2.1)

12. Describe the motor examination sequence and explain what each component reveals. (Linked to SLO 2.2)
13. Describe the CSF findings that distinguish bacterial from viral meningitis and from subarachnoid haemorrhage. (Linked to SLO 2.3)
14. Describe the Glasgow Coma Scale, including all component scores and clinical thresholds. (Linked to SLO 2.4)
15. Describe the initial management of an unconscious patient in a Nigerian hospital. (Linked to SLO 2.4)

Answers to Concept Check Questions [Series 2]

Objective questions

1. Vascular.
2. 3.
3. Bright or white.
4. Motor.
5. 8.

Short-answer questions

6. Vertigo is a false sense of rotational movement of self or environment, indicating vestibular disease (peripheral: inner ear; or central: brainstem or cerebellum). Presyncope is a feeling of impending faint or lightheadedness, indicating cardiovascular causes (arrhythmia, postural hypotension) or metabolic causes (hypoglycaemia, anaemia), not vestibular disease.
7. Spasticity is a velocity-dependent increase in tone caused by a UMN lesion (loss of descending inhibitory control of spinal stretch reflexes); it gives a clasp-knife quality, with resistance greatest at the start of movement and then suddenly giving way. Cogwheel rigidity is a uniform, ratchet-like resistance throughout the range of passive movement caused by extrapyramidal disease (Parkinson's disease); it is not velocity-dependent.

8. The patient is positioned in the lateral decubitus or sitting position; the L3/L4 or L4/L5 interspace is identified (below the conus medullaris at L1/L2); a spinal needle is inserted under aseptic conditions; opening pressure is measured; CSF is collected for analysis. The main contraindication is suspected raised intracranial pressure (papilloedema, focal signs, deteriorating consciousness): CT brain must be performed first to exclude a space-occupying lesion, as LP in this setting risks fatal brainstem herniation.
9. Consciousness has two components: arousal (the level of wakefulness, maintained by the brainstem reticular activating system: impaired by brainstem lesions or metabolic causes) and content (cognitive functions subserved by the cerebral cortex: impaired by cortical lesions, encephalopathy, or delirium). Both components must be intact for normal consciousness; a patient may be aroused but confused (content impaired), or unarousable (arousal impaired).
10. Fixed and bilaterally dilated pupils indicate midbrain compression or transtentorial herniation, a neurosurgical emergency. Pinpoint pupils indicate a pontine lesion (interrupting descending sympathetic fibres) or opioid toxicity. Asymmetric pupils (anisocoria) with a unilaterally dilated, unreactive pupil indicate compression of the ipsilateral third cranial nerve from uncal herniation by a supratentorial mass.

Long-answer questions

11. **Basic response:** Site (unilateral: migraine or cluster headache; bilateral: tension headache or raised ICP). Onset (thunderclap: worst headache ever, maximal within seconds: subarachnoid haemorrhage until proven otherwise). Character (throbbing: migraine; pressure or band-like: tension headache; stabbing: cluster headache). Severity (visual analogue scale 1 to 10). Duration (episodic: migraine 4 to 72 hours; cluster 15 to 180 minutes; continuous: raised ICP). Associated features (nausea and vomiting and photophobia and phonophobia: migraine; neck stiffness and fever and photophobia: meningitis; visual changes: raised ICP or migraine with aura; focal neurology: intracranial mass). Triggers (menstruation, alcohol, stress, sleep deprivation in migraine). Progression over time (chronic worsening: raised ICP from mass lesion).

Value-added response: The most dangerous headache to identify is the thunderclap headache of subarachnoid haemorrhage: even if the patient looks well and the neurological examination is normal, urgent CT brain and LP (to detect xanthochromia if CT is negative) are mandatory, since 10 to 15 percent of patients with SAH die before reaching hospital and early diagnosis prevents rebleeding.

12. **Basic response:** Inspection: observe for muscle wasting (LMN disease or disuse atrophy), fasciculations (LMN disease: denervation), abnormal posture (hemiparetic arm

held flexed and leg extended in UMN lesion), and involuntary movements (tremor, chorea, dystonia). Tone: passive movement of each joint; spasticity (velocity-dependent, UMN), flaccidity (reduced, LMN or cerebellar), cogwheel rigidity (Parkinson's disease). Power: test key muscle groups against resistance and grade on MRC 0 to 5 scale. Reflexes: elicit tendon reflexes with a tendon hammer (biceps C5/C6, triceps C7, supinator C5/C6, knee L3/L4, ankle S1/S2) and grade 0 to 4+; test plantar response (upgoing Babinski in UMN lesion).

Value-added response: The MRC power scale must be applied consistently: grade 4 (movement against some resistance) is further subdivided in practice into 4 minus (movement against slight resistance), 4 (against moderate resistance), and 4 plus (against strong but submaximal resistance), giving more clinical precision when documenting progressive improvement or deterioration in a patient with weakness.

13. **Basic response:** Bacterial meningitis: turbid or purulent CSF, markedly elevated protein (above 1 g/L), very low glucose (below one-third of plasma glucose), high white cell count (above 1000 per microlitre predominantly neutrophils). Viral meningitis: clear CSF, mildly elevated protein, normal glucose, lymphocytic pleocytosis (10 to 500 cells per microlitre). Subarachnoid haemorrhage: uniformly blood-stained CSF that does not clear between bottles (distinguishes true SAH from a traumatic tap), and xanthochromia (yellow discolouration from bilirubin) if LP is performed more than two hours after the bleed; protein is elevated; glucose is normal.

Value-added response: TB meningitis produces a CSF pattern that is intermediate: clear or slightly turbid, elevated protein, very low glucose (similar to bacterial meningitis), but with a predominantly lymphocytic pleocytosis (100 to 500 cells). The spider-web clot on standing and the very low glucose are characteristic. ADA (adenosine deaminase) in the CSF is elevated and AFB stain and GeneXpert should always be sent in the Nigerian context given the high TB burden.

14. **Basic response:** GCS has three components: Eye opening (E: 4 spontaneous; 3 to voice; 2 to pain; 1 none). Verbal response (V: 5 orientated; 4 confused; 3 inappropriate words; 2 incomprehensible sounds; 1 none). Motor response (M: 6 obeys commands; 5 localises pain; 4 withdrawal from pain; 3 abnormal flexion: decorticate; 2 extension: decerebrate; 1 none). Total score ranges from 3 to 15. Severity: 13 to 15 mild; 9 to 12 moderate; 8 or below severe. Threshold for intubation: GCS 8 or below, as the patient cannot reliably protect their airway. Always document as component scores (e.g. E3 V4 M5) not just the total.

Value-added response: The motor component of the GCS carries the most prognostic information: in a patient who cannot speak (for example due to intubation or facial trauma), the motor score alone can guide clinical decisions. A deteriorating motor score (from localisation to

extension) indicates progressive brainstem compromise and is the most urgent sign of deterioration in an unconscious patient.

15. **Basic response:** Airway: position in the recovery position (lateral decubitus); use oropharyngeal airway; intubate if GCS 8 or below. Breathing: assess respiratory rate and pattern; give high-flow oxygen; note Cheyne-Stokes breathing (bilateral hemisphere or diencephalic dysfunction), central neurogenic hyperventilation (midbrain), or ataxic breathing (medulla). Circulation: two large-bore IV cannulae; blood glucose immediately (give IV 50 percent dextrose if below 3 mmol/L); blood for FBC, urea and electrolytes, LFTs, blood cultures, and toxicology. Disability: document GCS, pupillary responses, eye movements, and best motor response to pain. Exposure: look for signs of meningism, rash (meningococcal), needle marks (drug overdose), medical alert bracelet. Then: urgent CT brain; LP if meningitis suspected and CT shows no contraindication; malaria blood film and RDT in all patients in Nigeria.

Value-added response: In Nigeria, the three most important treatable reversible causes to exclude urgently in any unconscious patient are hypoglycaemia (check blood glucose immediately and treat), cerebral malaria (blood film and RDT), and bacterial meningitis (LP after CT if safe). Missing any one of these is potentially fatal, yet all three are rapidly treatable if identified early.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Sudden onset (maximal within seconds or minutes) suggests a vascular cause such as stroke or subarachnoid haemorrhage. Gradual progressive onset suggests a structural cause (tumour, abscess) or a degenerative process. Episodic symptoms that are fully resolved between attacks suggest epilepsy, migraine, or transient ischaemic attacks. A relapsing-remitting course suggests multiple sclerosis.
2. Vertigo is a false sense of rotational movement of self or the environment, indicating disturbance of the vestibular system: peripheral vertigo arises from the inner ear (benign paroxysmal positional vertigo, Meniere's disease, vestibular neuritis) and is typically severe with nausea; central vertigo arises from the brainstem or cerebellum and is associated with other brainstem or cerebellar signs. Presyncope is a feeling of impending faint or lightheadedness, indicating cardiovascular causes (postural hypotension, arrhythmia) or metabolic causes (hypoglycaemia, anaemia), not vestibular disease.
3. Dysphasia is a disorder of language (comprehension or production of spoken or written language) resulting from cortical damage in the language areas of the dominant

hemisphere: Broca's area (motor speech: non-fluent aphasia) or Wernicke's area (language comprehension: fluent but meaningless aphasia). Dysarthria is impaired motor articulation of speech (slurred or scanning or nasal speech) from damage to the motor pathways controlling the muscles of speech: brainstem (bulbar palsy: LMN), bilateral UMN lesion (pseudobulbar palsy), or cerebellum (scanning dysarthria); language comprehension and formulation are intact.

4. Hypertension (the most important stroke risk factor in Nigeria), diabetes mellitus (common cause of peripheral neuropathy), HIV infection (causes dementia, peripheral neuropathy, and CNS opportunistic infections), tuberculosis (TB meningitis is a major cause of neurological morbidity), and sickle cell disease (causes ischaemic and haemorrhagic stroke, particularly in children).

5. Isoniazid (used in TB treatment) causes peripheral neuropathy by depleting pyridoxine (vitamin B6), and pyridoxine supplementation must always accompany isoniazid in neurological patients. Antiretrovirals (stavudine, didanosine, zalcitabine) cause peripheral neuropathy. Antiepileptic drugs have important interactions with each other and with other medications (rifampicin induces hepatic enzymes and reduces the levels of many antiretrovirals and oral contraceptives). Traditional herbal medicines widely used in Nigeria can cause hepatotoxicity and neurotoxicity and must be specifically asked about.

Part 2.2

1. Observe the patient's level of consciousness and ability to engage. Note the general nutritional status and any obvious asymmetry of the face or limbs. Look for abnormal posture (hemiparetic posture: arm flexed and leg extended, suggesting a UMN lesion). Identify any involuntary movements (tremor, chorea, dystonia, myoclonus). Assess speech as the patient gives their history (fluency, comprehension, articulation). Note any ptosis (third nerve palsy or Horner's syndrome) or facial asymmetry.

2. MRC 0: no movement at all. MRC 1: a visible or palpable flicker of contraction but no movement. MRC 2: full range of movement with gravity eliminated (movement possible in the horizontal plane but not against gravity). MRC 3: movement against gravity through the full range but not against any additional resistance. MRC 4: movement against some external resistance but less than normal. MRC 5: normal strength, equal to the examiner's resistance.

3. Spasticity is a velocity-dependent increase in muscle tone caused by a UMN lesion (stroke, spinal cord compression, MS); the resistance is greatest at the start of passive movement and then suddenly gives way (clasp-knife phenomenon). Cogwheel rigidity is a uniform, ratchet-like resistance present throughout the range of passive movement in all

directions, caused by extrapyramidal disease (Parkinson's disease: from dopamine deficiency in the substantia nigra); it is not velocity-dependent. Flaccidity is reduced or absent tone caused by LMN lesions or cerebellar disease.

4. Light touch (cotton wool dabbed gently on skin: tests both dorsal columns and spinothalamic tract). Pinprick (a fresh sterile pin: tests spinothalamic tract for pain and temperature). Vibration sense (128 Hz tuning fork placed on a bony prominence such as the big toe, medial malleolus, or sternum: tests dorsal columns). Proprioception or joint position sense (small passive movements of the distal interphalangeal joint of the big toe or finger: tests dorsal columns). Two-point discrimination and graphaesthesia (cortical sensory functions testing parietal lobe integrity).
5. Cerebellar disease produces a broad-based, unsteady, lurching or staggering gait (cerebellar ataxia); the patient walks with feet wide apart for balance and cannot walk heel-to-toe in tandem; the gait does not worsen with eye closure (distinguishing it from sensory ataxia). Parkinson's disease produces a shuffling, small-stepped gait with reduced arm swing; the patient may have difficulty initiating gait (start hesitation), festination (progressively faster small steps as if chasing their centre of gravity), and freezing when approaching a doorway or turning.

Part 2.3

1. Haemorrhagic stroke on non-contrast CT brain: an area of high density (hyperdense or bright white signal) within the brain parenchyma, corresponding to acute blood, which is hyperdense due to the high protein content of clotted haemoglobin. Subarachnoid haemorrhage on non-contrast CT: hyperdense (bright white) blood visible within the subarachnoid space, characteristically filling the basal cisterns, Sylvian fissures, and cortical sulci; the sensitivity of CT for SAH is above 98 percent within 6 hours of the ictus but falls rapidly after 24 hours, which is why LP for xanthochromia is required if CT is negative in a patient with thunderclap headache.
2. EEG is the investigation of choice for epilepsy. It identifies epileptiform discharges (spikes and spike-and-slow wave complexes) between seizures (interictal discharges) and during seizures (ictal activity), and classifies seizure types (generalised: bilateral synchronous discharges; focal: unilateral or regional discharges). EEG can also show diffuse slowing in metabolic encephalopathy (hepatic, uraemic, hypoxic), periodic discharges in herpes encephalitis, and electrocerebral silence in brain death.
3. Demyelinating neuropathy (for example Guillain-Barre syndrome): nerve conduction velocity is markedly slowed (below 70 percent of the lower limit of normal) because the myelin sheath, which enables rapid saltatory conduction, is damaged; the amplitude of

the action potential may also be reduced. Axonal neuropathy (for example diabetic peripheral neuropathy): the amplitude of the compound motor action potential (CMAP) and sensory nerve action potential (SNAP) are reduced, reflecting loss of axons; conduction velocity is relatively preserved (above 80 percent of the lower limit of normal) because surviving axons are still myelinated.

4. The patient is positioned in the lateral decubitus (foetal) position with the spine flexed, or sitting leaning forward over a pillow. The L3/L4 or L4/L5 interspace is identified (at or below the iliac crest level). Under full aseptic technique, local anaesthetic is infiltrated and a spinal needle is inserted through the midline ligaments into the subarachnoid space. Opening pressure is measured with a manometer. CSF is collected in three sequential bottles for appearance, protein, glucose, cell count, Gram stain and culture, and any other indicated tests. The main contraindication is suspected raised intracranial pressure (papilloedema, focal neurological signs, or deteriorating consciousness): CT brain must be performed first, as LP in this setting risks fatal downward brainstem herniation.
5. Bacterial meningitis: turbid or purulent CSF, markedly elevated protein (above 1 g/L), very low glucose (below one-third of simultaneous plasma glucose), and high white cell count (above 1000 per microlitre predominantly neutrophils); Gram stain positive in 60 to 80 percent. Viral meningitis: clear CSF, mildly elevated protein (0.5 to 1 g/L), normal glucose, and moderate lymphocytic pleocytosis (10 to 500 cells per microlitre). The key distinguishing features are the CSF glucose ratio (very low in bacterial, normal in viral) and the predominant cell type (neutrophils in bacterial, lymphocytes in viral).

Part 2.4

1. Consciousness has two components: arousal (the level of wakefulness, maintained by the ascending reticular activating system in the brainstem and thalamus: impaired by brainstem lesions, bilateral thalamic lesions, or metabolic and toxic causes) and content (the cognitive functions subserved by the cerebral cortex: impaired by diffuse cortical damage, encephalopathy, or delirium). The spectrum from full consciousness to coma: full consciousness, confusion or delirium (arousal present but content disturbed: the patient is awake but disoriented), obtundation (reduced alertness requiring stimulation to maintain), stupor (arousal only with vigorous stimulation: patient is difficult to rouse), and coma (unarousable even with painful stimulation).
2. Pinpoint pupils (miosis below 2 mm bilaterally) indicate a pontine lesion (interrupting the descending sympathetic fibres from the hypothalamus) or opioid toxicity (opioids cause miosis through muscarinic pupillary constriction); the light reflex is preserved in opioid toxicity but may be impaired in a pontine lesion. Fixed and bilaterally dilated pupils

(mydriasis with absent light reflex) indicate midbrain compression from transtentorial herniation of the uncus of the temporal lobe, compressing the parasympathetic fibres running in the third cranial nerve, or from a direct midbrain lesion.

3. Eye opening (E: 4 spontaneous; 3 to voice; 2 to pain; 1 none). Verbal response (V: 5 orientated; 4 confused; 3 inappropriate words; 2 incomprehensible sounds; 1 none). Motor response (M: 6 obeys commands; 5 localises pain; 4 withdrawal from pain; 3 abnormal flexion: decorticate; 2 extension: decerebrate; 1 none). Total GCS ranges from 3 (deepest coma) to 15 (fully conscious). Record as component scores: for example E2 V3 M4 = total 9 (moderate impairment).

4. The GCS threshold for airway protection is 8 or below. The reason is that at this level of consciousness the patient has lost the ability to maintain their airway safely: the gag reflex and cough reflex are sufficiently impaired that the patient is at high risk of aspiration of oral secretions or vomit, and secretions pool in the posterior pharynx obstructing the airway. Endotracheal intubation at GCS 8 or below secures a definitive airway, ensures adequate oxygenation and ventilation, and prevents aspiration pneumonia, which would significantly worsen the prognosis of the underlying neurological condition.

5. Airway: position in the lateral recovery position; use oropharyngeal airway adjunct; intubate if GCS 8 or below. Breathing: assess rate, pattern, and oxygen saturation; give high-flow oxygen via non-rebreather mask. Circulation: obtain two large-bore IV cannulae; check blood glucose immediately and give IV 50 percent dextrose if below 3 mmol/L; take blood for FBC, urea and electrolytes, LFTs, blood cultures, and toxicology. Disability: document GCS component scores, pupillary responses, eye movements, and best motor response. Exposure: check for meningism, petechial rash, needle marks, medical alert bracelet. Then order urgent CT brain, LP after CT if meningitis is suspected and CT shows no contraindication, and malaria blood film and RDT in all patients in the Nigerian context.

References and Attributions [Series 2]

Textual References

- Fuller, G. and Manford, M. (2010). Neurology: An Illustrated Colour Text (4th ed.). Edinburgh: Churchill Livingstone.
- Longmore, M., Wilkinson, I., Baldwin, A. and Wallin, E. (2014). Oxford Handbook of Clinical Medicine (9th ed.). Oxford: Oxford University Press.
- Teasdale, G. and Jennett, B. (1974). Assessment of Coma and Impaired Consciousness: A Practical Scale. Lancet, 2(7872), 81 to 84.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.

Figures, Plates, Tables, and Boxes

- Figure 2.1: Structure of the neurological history. AI-generated using the prompt: "Create a four-panel diagram titled The Neurological History: presenting complaint characterisation, common neurological complaints with characterisation prompts, past medical history key conditions in Nigeria, and family, drug, and social history." Suggested OER search string: "neurological history taking structured diagram presenting complaint Nigeria OER."
- Figure 2.2: The motor and sensory-coordination neurological examination. AI-generated using the prompt: "Two-panel diagram: left panel sequential motor examination flow (inspection, tone, power with MRC scale, reflexes); right panel sensory modalities with corresponding tracts and coordination and gait assessment." Suggested OER search string: "neurological examination motor sensory coordination gait diagram OER."
- Figure 2.3: Neurological investigations. AI-generated using the prompt: "Three-panel diagram: CT versus MRI comparison; EEG and NCS plus EMG; LP procedure diagram with CSF analysis comparison table." Suggested OER search string: "neurological investigations CT MRI EEG NCS EMG lumbar puncture CSF analysis OER."
- Figure 2.4: The spectrum of consciousness, GCS, and management of the unconscious patient. AI-generated using the prompt: "Two-panel diagram: left consciousness spectrum from full consciousness to coma with causes and pupillary signs; right GCS table with component scores, severity bands, and management steps for the unconscious patient." Suggested OER search string: "Glasgow Coma Scale diagram consciousness spectrum unconscious patient management OER."

Course code: MED 408

Course title: Neurology I

Course credit load: 2 Units

Series 3: Cranial Nerves and Systems

- **Part 3.1: Overview of the Cranial Nerves**
    - Sub Part 3.1.1: Classification and general functions
    - Sub Part 3.1.2: Cranial nerves I to IV
    - Sub Part 3.1.3: Cranial nerves V to VIII
- **Part 3.2: Cranial Nerves IX to XII and the Bulbar System**
    - Sub Part 3.2.1: Cranial nerves IX and X
    - Sub Part 3.2.2: Cranial nerves XI and XII
    - Sub Part 3.2.3: Bulbar and pseudobulbar palsy
- **Part 3.3: The Visual System**
    - Sub Part 3.3.1: The visual pathway
    - Sub Part 3.3.2: Pupillary reflexes and eye movements
    - Sub Part 3.3.3: Visual field defects and their localisation
- **Part 3.4: The Extrapyramidal System and Cerebellum**
    - Sub Part 3.4.1: The basal ganglia and extrapyramidal system
    - Sub Part 3.4.2: Parkinson's disease
    - Sub Part 3.4.3: Other movement disorders

Introduction

The cranial nerves are the twelve paired nerves that emerge directly from the brain and brainstem, supplying the structures of the head, neck, and several viscera. Their examination is a central part of the neurological assessment and provides a precise window into the integrity of the brainstem. Knowledge of what each cranial nerve does and where it originates allows you to

localise lesions with remarkable accuracy. Alongside the cranial nerves, an understanding of the visual system and the extrapyramidal system is essential for managing some of the most common and disabling neurological conditions.

We shall commence with the classification and examination of the cranial nerves, covering cranial nerves I to VIII in the first Part and IX to XII with the bulbar system in the second. We will then explore the visual system in detail, from the retina to the visual cortex, and explain how lesions at different points along the pathway produce characteristic visual field defects. Finally, we will examine the extrapyramidal system, covering the basal ganglia, Parkinson's disease, and other movement disorders.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 3.1:** describe the function and examination of each cranial nerve and identify the deficits produced by lesions of cranial nerves I to XII,
- **SLO 3.2:** describe bulbar and pseudobulbar palsy and distinguish between them,
- **SLO 3.3:** describe the visual pathway and identify the visual field defect produced by lesions at each level, and
- **SLO 3.4:** describe the function of the basal ganglia and the clinical features and management of Parkinson's disease and other movement disorders.

3.1 Overview of the Cranial Nerves

3.1.1 Classification and general functions

There are twelve pairs of cranial nerves, numbered I to XII in order from anterior to posterior along the base of the brain. Each nerve may be purely sensory, purely motor, or mixed (carrying both sensory and motor fibres), and some also carry parasympathetic autonomic fibres. A useful mnemonic for remembering whether each nerve is sensory (S), motor (M), or both (B) is: Some Say Marry Money But My Brother Says Bad Business Marry Money, corresponding to CN I through XII. Cranial nerves I (olfactory) and II (optic) are in fact extensions of the brain rather than true peripheral nerves, which is why they do not regenerate after injury.

The cranial nerve nuclei are arranged within the brainstem: CN III and IV nuclei lie in the midbrain, CN V to VIII in the pons, and CN IX to XII in the medulla. This arrangement means that a brainstem lesion at a given level will affect the cranial nerve nuclei at that level, allowing precise localisation. For example, a lesion at the level of the pons will produce ipsilateral VI

nerve palsy (lateral gaze palsy) and ipsilateral VII nerve palsy (facial palsy), combined with contralateral hemiplegia (from the corticospinal tract): this is the classical Millard-Gubler syndrome.

Fill in the blank: Cranial nerve nuclei III and IV are found in the _____, while nuclei IX to XII are found in the _____. A mnemonic to remember the sensory and motor nature of each cranial nerve begins: Some Say _____ Money.

3.1.2 Cranial nerves I to IV

CN I (olfactory nerve) carries the sense of smell from the olfactory mucosa in the roof of the nasal cavity to the olfactory bulb and cortex. Anosmia (loss of smell) is caused by nasal disease (the most common cause in practice), head injury (shearing of the olfactory filaments at the cribriform plate), or a frontal lobe mass (olfactory groove meningioma: causes unilateral anosmia with ipsilateral optic atrophy and contralateral papilloedema: the Foster-Kennedy syndrome). **CN II (optic nerve)** transmits visual information from the retina to the visual cortex; its examination includes visual acuity, visual fields, colour vision, and the optic disc on fundoscopy. Papilloedema (swelling of the optic disc from raised intracranial pressure) must be distinguished from papillitis (optic disc swelling from inflammation of the optic nerve, causing painful loss of vision: a feature of optic neuritis in multiple sclerosis).

CN III (oculomotor nerve) innervates the superior rectus, inferior rectus, medial rectus, inferior oblique, and levator palpebrae superioris muscles, and carries parasympathetic fibres to the pupillary constrictor and ciliary muscle. A CN III palsy produces a classic triad: ptosis (levator palpebrae paralysis), a fixed dilated pupil (parasympathetic palsy), and the eye deviated down and out (due to unopposed action of the superior oblique CN IV and lateral rectus CN VI). **CN IV (trochlear nerve)** innervates the superior oblique muscle, which depresses and intorts the adducted eye. CN IV palsy causes vertical diplopia worse on looking down and towards the nose (for example on descending stairs), and the patient tilts their head away from the affected side to compensate.

Fill in the blank: A complete CN III palsy produces ptosis, a fixed _____ pupil, and the eye deviated down and _____. CN IV palsy causes vertical diplopia worst on looking _____ and towards the nose.

3.1.3 Cranial nerves V to VIII

CN V (trigeminal nerve) is the largest cranial nerve and has three divisions: V1 (ophthalmic), V2 (maxillary), and V3 (mandibular). It provides sensory innervation to the entire face, scalp, cornea, nasal and oral mucosa, and teeth, and V3 also carries motor fibres to the muscles of mastication (temporalis, masseter, medial and lateral pterygoids). The corneal reflex (touching the cornea causes blinking) has its afferent limb in CN V1 and its efferent limb in CN VII. CN V lesions cause facial sensory loss and, in V3 lesions, weakness of jaw opening (the jaw deviates towards the side of the lesion when CN V is damaged).

CN VI (abducens nerve) innervates the lateral rectus, which abducts the eye. CN VI palsy causes a failure of abduction and horizontal diplopia worst on gaze towards the affected side; it is the most common ocular motor nerve palsy and is vulnerable to raised intracranial pressure (false localising sign). **CN VII (facial nerve)** innervates the muscles of facial expression and carries taste from the anterior two-thirds of the tongue (chorda tympani). A lower motor neuron (LMN) VII palsy (Bell's palsy) affects the entire ipsilateral face including the forehead; an upper motor neuron (UMN) VII lesion (for example from a cortical stroke) spares the forehead because the forehead muscles have bilateral cortical representation. **CN VIII (vestibulocochlear nerve)** carries hearing (cochlear division) and balance (vestibular division); its lesions cause sensorineural deafness and vertigo.

Fill in the blank: The corneal reflex has its afferent limb in CN _____ and its efferent limb in CN VII. Bell's palsy is a _____ motor neuron lesion of CN VII, affecting the entire ipsilateral face including the forehead.

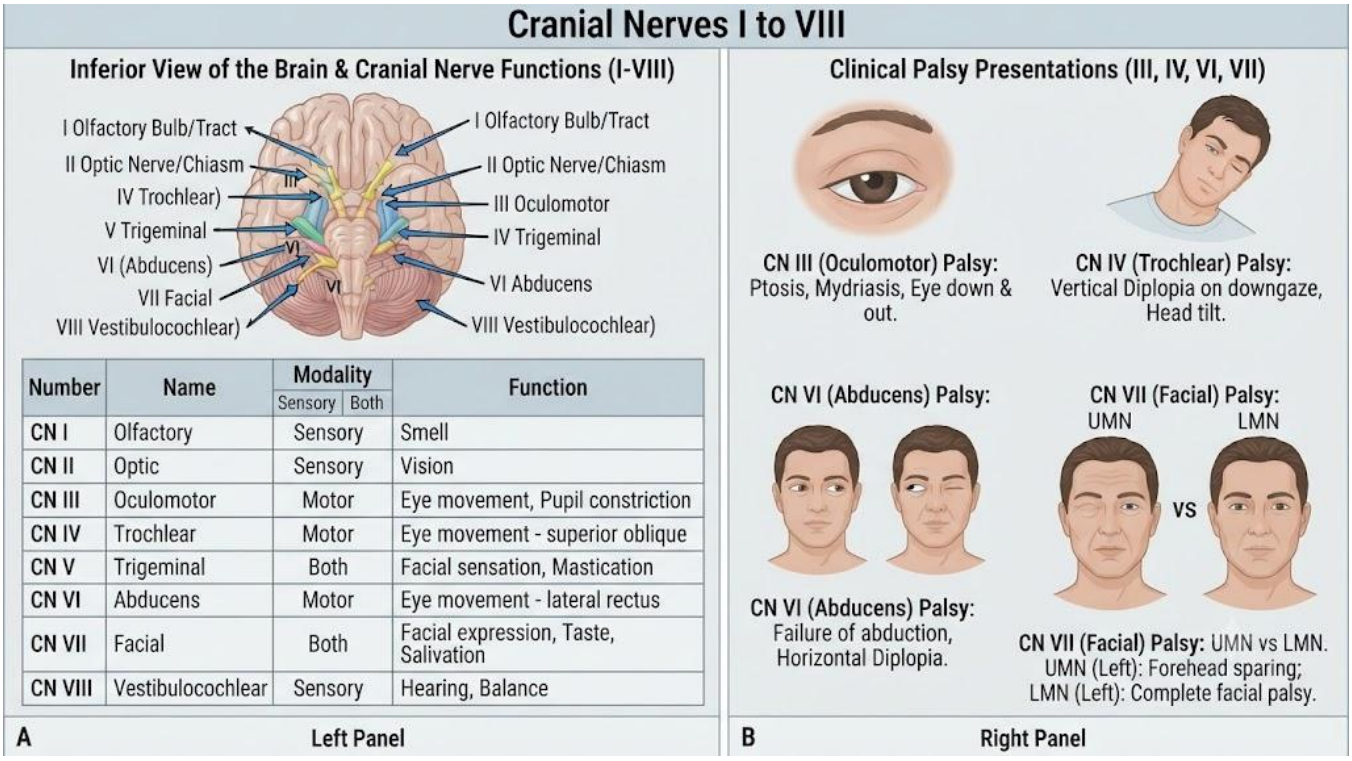


Figure 3.1: Cranial nerves I to VIII: functions and palsy patterns

Pilot questions 3.1

1. Describe the arrangement of cranial nerve nuclei within the brainstem. (Linked to SLO 3.1)
2. Describe the clinical features of a complete CN III palsy and explain its anatomical basis. (Linked to SLO 3.1)

3. Distinguish a UMN from a LMN lesion of CN VII and explain why the forehead is spared in UMN lesions. (Linked to SLO 3.1)
4. Describe the three divisions of CN V and the clinical features of a CN V lesion. (Linked to SLO 3.1)
5. Describe the Foster-Kennedy syndrome and state what causes it. (Linked to SLO 3.1)

3.2 Cranial Nerves IX to XII and the Bulbar System

3.2.1 Cranial nerves IX and X

CN IX (glossopharyngeal nerve) provides sensory innervation to the posterior third of the tongue (including taste), the pharynx, and the middle ear, and it carries the afferent limb of the gag reflex. It also supplies the parotid gland with parasympathetic secretomotor fibres. Isolated CN IX lesions are uncommon; it is most often affected together with CN X and XI in jugular foramen syndrome (from tumours or thrombosis at the jugular foramen).

CN X (vagus nerve) has the most extensive distribution of any cranial nerve, innervating the pharynx, larynx, oesophagus, trachea, heart, and abdominal viscera. It provides the efferent limb of the gag reflex and controls swallowing (via the pharyngeal plexus with CN IX), vocal cord movement (via the recurrent laryngeal nerve: a CN X branch), and the parasympathetic supply to the thoracic and abdominal viscera. A unilateral CN X lesion causes hoarseness (ipsilateral vocal cord paralysis) and ipsilateral uvular deviation away from the side of the lesion (the uvula is pulled towards the intact side).

Fill in the blank: The afferent limb of the gag reflex is carried by CN _____, while the efferent limb is carried by CN X. A unilateral CN X lesion causes the uvula to deviate _____ from the side of the lesion.

3.2.2 Cranial nerves XI and XII

CN XI (accessory nerve) has two components: the cranial root (which joins CN X to supply the larynx and pharynx) and the spinal root (which descends into the posterior triangle of the neck to supply the sternocleidomastoid and trapezius muscles). Clinically, CN XI is tested by asking the patient to turn their head against resistance (sternocleidomastoid: turns the head to the contralateral side) and to shrug their shoulders against resistance (trapezius). A CN XI lesion causes weakness of head turning (towards the contralateral side) and drooping of the ipsilateral shoulder with winging of the scapula.

CN XII (hypoglossal nerve) innervates all the intrinsic and extrinsic muscles of the tongue (except palatoglossus, which is supplied by CN X). A lower motor neuron CN XII lesion causes ipsilateral tongue wasting and fasciculations, and the tongue deviates towards the side of the lesion on protrusion (the intact side pushes the tongue towards the weak side). An upper motor

neuron lesion of CN XII (for example in a cortical stroke) causes the tongue to deviate away from the lesion (towards the side of the hemiplegia), and there is no wasting or fasciculations.

Fill in the blank: CN XI supplies the sternocleidomastoid and _____ muscles. In a LMN CN XII lesion, the tongue deviates _____ the side of the lesion on protrusion.

3.2.3 Bulbar and pseudobulbar palsy

Bulbar palsy is weakness of the muscles supplied by cranial nerves IX, X, XI, and XII from a lower motor neuron lesion of their nuclei in the medulla (bulb) or their peripheral nerves. It presents with a nasal, quiet, or hypernasal voice, dysarthria (slurred speech), dysphagia (difficulty swallowing, with nasal regurgitation of liquids), a weak, wasted, and fasciculating tongue, and an absent gag reflex. Causes in Nigeria include motor neuron disease, Guillain-Barre syndrome, syringobulbia, and brainstem tumours.

Pseudobulbar palsy is caused by bilateral upper motor neuron lesions of the corticobulbar tracts (the fibres carrying motor commands from the cortex to the bulbar nuclei). The clinical features mimic bulbar palsy (dysarthria and dysphagia) but with the opposite signs: the tongue is small, spastic, and slow-moving (not wasted or fasciculating), the jaw jerk is brisk (the hallmark of pseudobulbar palsy), the gag reflex is exaggerated, and the patient has emotional lability (pathological laughing and crying from damage to the corticobulbar fibres that modulate emotional expression). Common causes include bilateral strokes (the most common cause), MS, and motor neuron disease.

Fill in the blank: Bulbar palsy is a _____ motor neuron lesion causing a wasted tongue with fasciculations and an absent gag reflex. Pseudobulbar palsy is a _____ motor neuron lesion with a brisk jaw jerk and emotional _____.

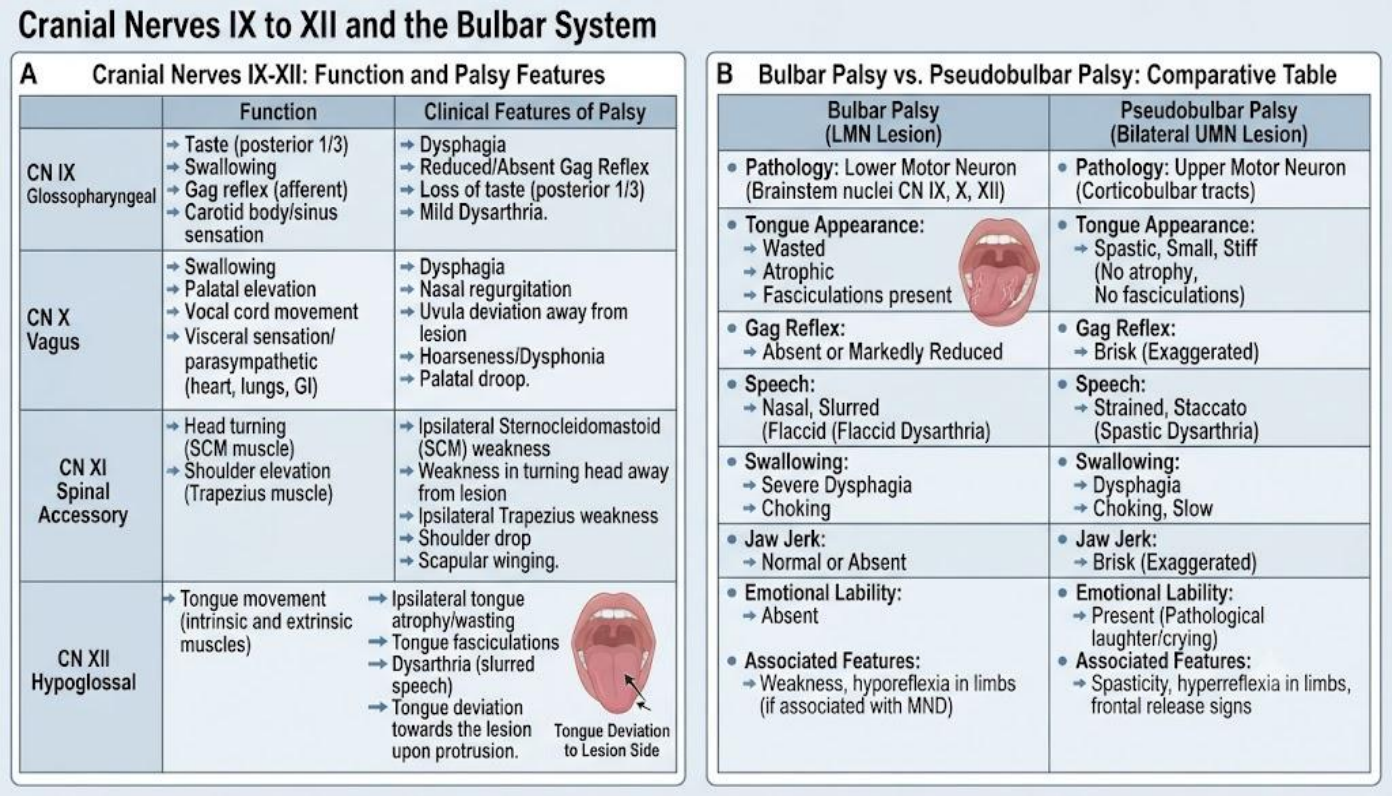


Figure 3.2: Cranial nerves IX to XII and the comparison of bulbar with pseudobulbar palsy

Pilot questions 3.2

1. Describe the clinical features of a unilateral CN X lesion. (Linked to SLO 3.1)
2. Describe how you would test CN XI and state the findings in a CN XI palsy. (Linked to SLO 3.1)
3. Describe the tongue findings in a LMN versus UMN CN XII lesion. (Linked to SLO 3.1)
4. Compare the clinical features of bulbar and pseudobulbar palsy. (Linked to SLO 3.2)
5. State the hallmark sign of pseudobulbar palsy and explain its anatomical basis. (Linked to SLO 3.2)

3.3 The Visual System

3.3.1 The visual pathway

The visual pathway begins at the retina, where photoreceptors (rods and cones) convert light into electrical signals. These signals travel along the optic nerve (CN II) to the optic chiasm, where the fibres from the nasal (medial) half of each retina cross to the opposite side, while fibres from

the temporal (lateral) half remain ipsilateral. After the chiasm, the fibres travel as the optic tract to the lateral geniculate nucleus of the thalamus, then as the optic radiation through the parietal and temporal lobes to the primary visual cortex in the occipital lobe (Brodmann area 17). Each side of the brain processes the contralateral visual field.

Understanding what crosses at the chiasm is the key to localising visual field defects. The nasal retina receives light from the temporal visual field; the temporal retina receives light from the nasal visual field. Because the nasal fibres cross at the chiasm, all information from the left visual field (whether from the left eye's temporal retina or the right eye's nasal retina) ends up in the right optic tract and eventually in the right visual cortex. This is why a lesion in the right optic tract, radiation, or visual cortex causes a loss of the left visual field in both eyes: a left homonymous hemianopia.

Fill in the blank: At the optic chiasm, fibres from the _____ half of each retina cross to the opposite side. A lesion of the right optic tract causes a _____ homonymous hemianopia.

3.3.2 Pupillary reflexes and eye movements

The pupillary light reflex tests the integrity of CN II (afferent) and CN III (efferent). Shining a light into one eye normally causes constriction of both the illuminated pupil (direct reflex) and the opposite pupil (consensual reflex). A relative afferent pupillary defect (RAPD, also called a Marcus Gunn pupil) is detected using the swinging flashlight test: when the light is swung from the normal eye to the affected eye, both pupils dilate (rather than maintaining constriction), indicating an optic nerve lesion on the affected side that reduces the afferent signal.

Conjugate eye movements are controlled by the frontal eye fields (voluntary saccades), the superior colliculus (reflexive saccades), and the brainstem gaze centres. Horizontal gaze is coordinated by the paramedian pontine reticular formation (PPRF) in the pons, which projects to the ipsilateral CN VI nucleus and, via the medial longitudinal fasciculus (MLF), to the contralateral CN III nucleus. Internuclear ophthalmoplegia (INO) is caused by a lesion of the MLF and produces failure of adduction of the ipsilateral eye on lateral gaze, with nystagmus of the abducting eye; it is the most characteristic ocular finding in multiple sclerosis.

Fill in the blank: A _____ afferent pupillary defect (RAPD) indicates an optic nerve lesion detected by the swinging flashlight test. Internuclear ophthalmoplegia (INO) is caused by a lesion of the medial longitudinal _____ and is the most characteristic eye finding in multiple sclerosis.

3.3.3 Visual field defects and their localisation

The pattern of visual field loss precisely localises the site of the lesion along the visual pathway. A monocular visual loss (loss of vision in one eye only) indicates a lesion of the retina or the optic nerve, before the chiasm. A bitemporal hemianopia (loss of both temporal fields, creating tunnel vision) is pathognomonic of a lesion at the optic chiasm, most commonly a pituitary adenoma compressing the crossing nasal fibres from below. A homonymous hemianopia (loss of

the same half of the visual field in both eyes) indicates a lesion of the optic tract, optic radiation, or visual cortex on the contralateral side.

Within homonymous hemianopias, further localisation is possible. A lesion of the temporal lobe optic radiation produces an upper quadrant homonymous defect (pie in the sky), because the inferior fibres of the radiation loop into the temporal lobe (Meyer's loop). A lesion of the parietal lobe radiation produces a lower quadrant defect. A lesion of the visual cortex produces a macular-sparing homonymous hemianopia (the macula has a large cortical representation and a dual blood supply, so it is often spared when the posterior cerebral artery is occluded).

Fill in the blank: A bitemporal hemianopia is pathognomonic of a lesion at the optic _____, most commonly from a _____ adenoma. Macular-sparing homonymous hemianopia indicates a lesion of the _____ cortex.

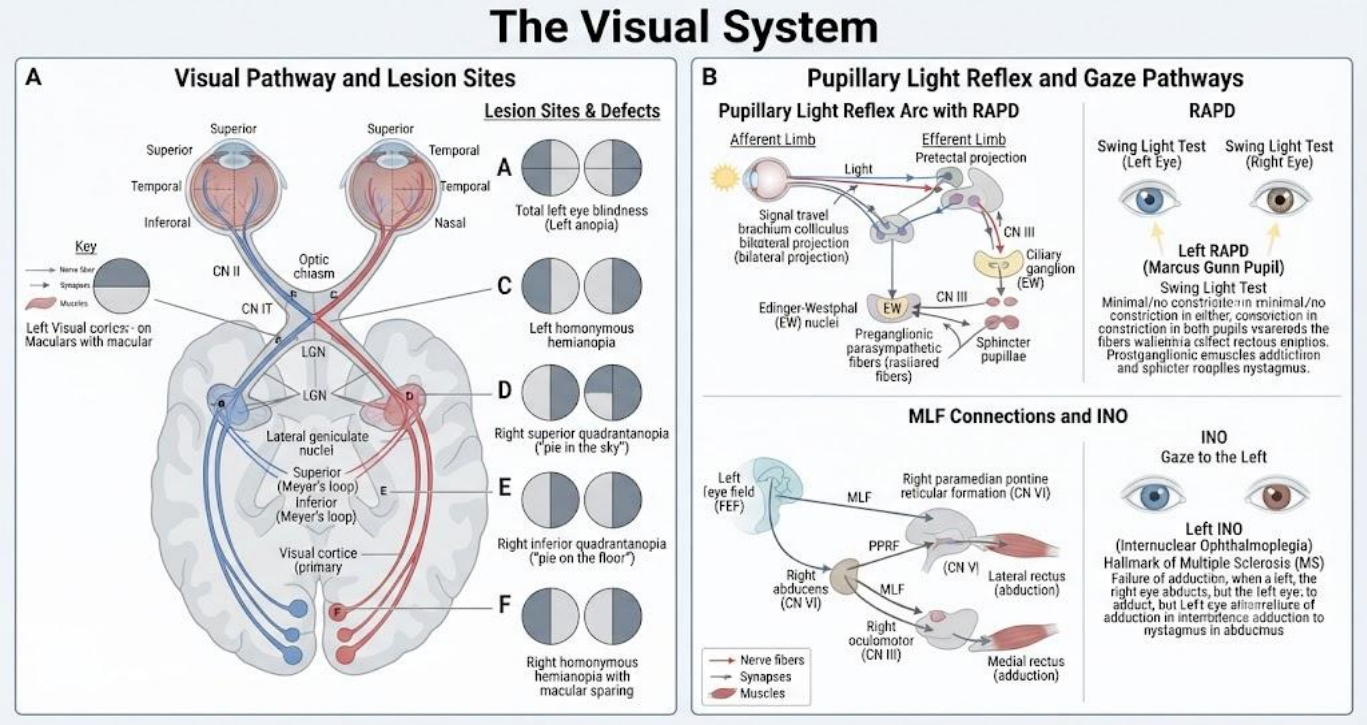


Figure 3.3: The visual pathway with lesion sites and corresponding visual field defects, pupillary reflexes, and INO

Pilot questions 3.3

1. Describe the visual pathway from the retina to the visual cortex. (Linked to SLO 3.3)
2. Explain why a lesion at the optic chiasm causes a bitemporal hemianopia. (Linked to SLO 3.3)
3. Describe the swinging flashlight test and state what a positive RAPD indicates. (Linked to SLO 3.3)

4. Describe internuclear ophthalmoplegia and state its most common cause. (Linked to SLO 3.3)
5. Explain the difference between a parietal and a temporal lobe optic radiation lesion in terms of visual field defect. (Linked to SLO 3.3)

3.4 The Extrapyramidal System and Cerebellum

3.4.1 The basal ganglia and extrapyramidal system

The basal ganglia are a group of subcortical nuclei that modulate voluntary movement through circuits connecting them to the motor cortex and thalamus. The key structures are the striatum (caudate nucleus and putamen), the globus pallidus (internal and external segments), the subthalamic nucleus, and the substantia nigra. Dopaminergic neurons from the substantia nigra pars compacta project to the striatum via the nigrostriatal pathway. The direct pathway (striatum to globus pallidus internal: disinhibitory) facilitates movement, while the indirect pathway (striatum to globus pallidus external to subthalamic nucleus to globus pallidus internal: inhibitory) suppresses unwanted movement.

The extrapyramidal system refers to the motor control system outside the corticospinal (pyramidal) tract, encompassing the basal ganglia, their connections to the thalamus and cortex, and related brainstem structures. Extrapyramidal lesions produce movement disorders characterised by either a paucity of movement (hypokinesia: as in Parkinson's disease, where dopamine depletion disrupts the balance in favour of excessive inhibition of movement) or excessive involuntary movement (hyperkinesia: as in chorea, dystonia, and hemiballismus).

Fill in the blank: The _____ pathway from the striatum facilitates movement, while the indirect pathway suppresses unwanted movement. Dopamine depletion in Parkinson's disease disrupts the balance of these pathways in favour of excessive _____ of movement.

3.4.2 Parkinson's disease

Parkinson's disease is the most common movement disorder and the second most common neurodegenerative disease after Alzheimer's disease. It results from the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, with accumulation of Lewy bodies (intraneuronal inclusions of misfolded alpha-synuclein protein). The cardinal motor features are the TRAP mnemonic: Tremor (resting, pill-rolling, 4 to 6 Hz, suppressed by voluntary movement), Rigidity (cogwheel or lead-pipe, present throughout range of passive movement), Akinesia or bradykinesia (slowness of initiation and execution of movement), and Postural instability (impaired righting reflexes, leading to falls). Non-motor features include anosmia (often the earliest symptom), REM sleep behaviour disorder, autonomic dysfunction, depression, and dementia (in up to 80 percent of patients after 20 years).

Treatment of Parkinson's disease is aimed at restoring dopaminergic neurotransmission. Levodopa (combined with a peripheral decarboxylase inhibitor such as carbidopa or benserazide to prevent peripheral conversion to dopamine) remains the most effective symptomatic treatment; it is converted to dopamine in the brain. Dopamine agonists (pramipexole, ropinirole, rotigotine) stimulate dopamine receptors directly and are used as initial monotherapy in younger patients to delay levodopa use and reduce long-term motor complications. Monoamine oxidase B (MAO-B) inhibitors (selegiline, rasagiline) reduce dopamine breakdown. Long-term levodopa complications include motor fluctuations (wearing-off phenomenon) and dyskinesias (involuntary choreiform movements).

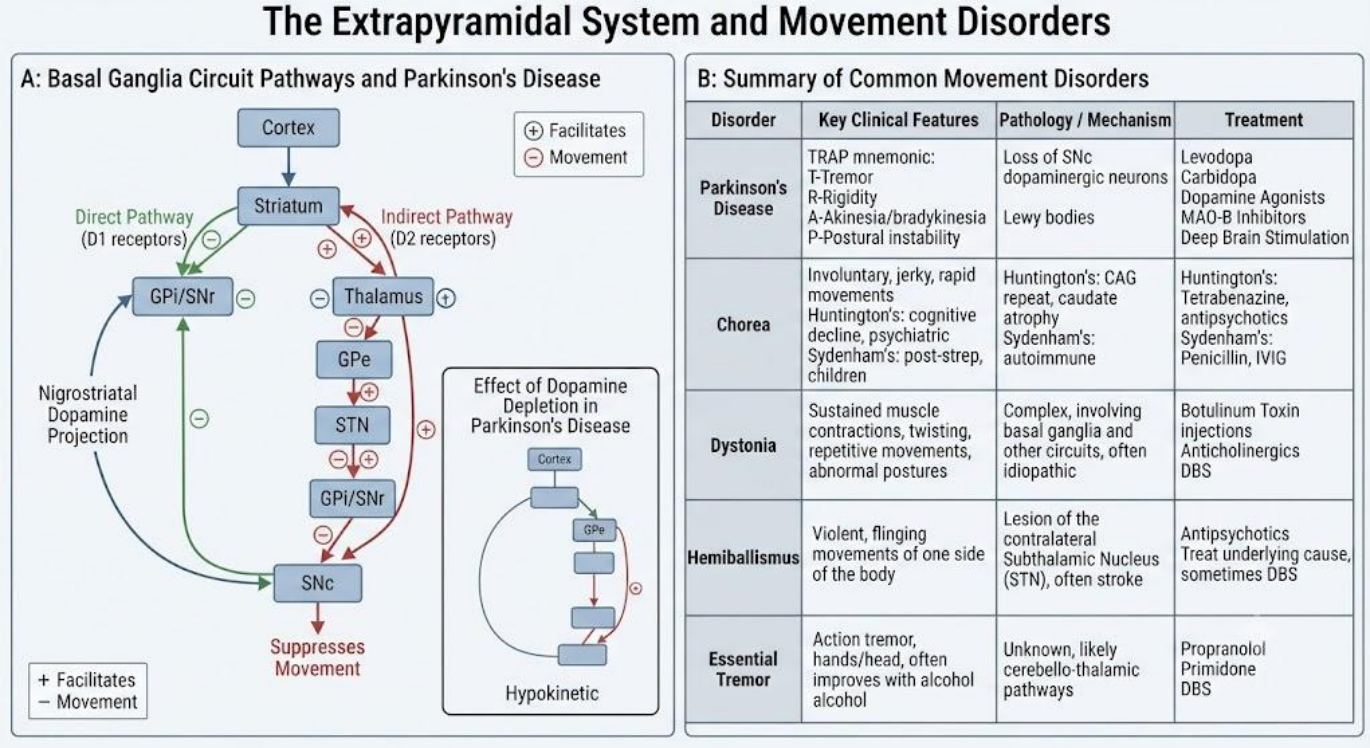
Fill in the blank: The cardinal motor features of Parkinson's disease are summarised by the mnemonic _____: Tremor, Rigidity, Akinesia or bradykinesia, and Postural instability. Levodopa is combined with a peripheral decarboxylase inhibitor such as _____ to prevent its peripheral conversion to dopamine.

3.4.3 Other movement disorders

Chorea is characterised by brief, irregular, unpredictable involuntary movements that flow from one body part to another (the word chorea comes from the Greek for dance). Causes include Huntington's disease (autosomal dominant: CAG trinucleotide repeat expansion in the HTT gene on chromosome 4; presents in the fourth or fifth decade with chorea, progressive dementia, and psychiatric disturbance; no disease-modifying treatment), Sydenham's chorea (a complication of rheumatic fever: group A Streptococcus: seen in children in Nigeria: treat the underlying rheumatic disease), drug-induced chorea (levodopa overdose, antipsychotics causing tardive dyskinesia), and systemic lupus erythematosus.

Dystonia is sustained or intermittent muscle contractions causing abnormal, often repetitive movements or postures. It can be focal (affecting one body part: for example writer's cramp, cervical dystonia or torticollis), segmental, or generalised. Hemiballismus is a dramatic, violent, flinging movement of one half of the body (typically the proximal limbs) caused by a lesion (most commonly an ischaemic or haemorrhagic stroke) of the contralateral subthalamic nucleus. Essential tremor is the most common movement disorder overall; it is a postural and action tremor (worst when the limb is held outstretched or during voluntary movement, unlike the resting tremor of Parkinson's disease) and is treated with propranolol or primidone.

Fill in the blank: Huntington's disease is caused by a CAG _____ repeat expansion in the HTT gene and presents with chorea, dementia, and _____ disturbance. Hemiballismus is caused by a lesion of the contralateral _____ nucleus.



Disorder	Key Clinical Features	Pathology / Mechanism	Treatment
Parkinson's Disease	TRAP mnemonic: T-Tremor R-Rigidity A-Akinesia/bradykinesia P-Postural instability	Loss of SNc dopaminergic neurons Lewy bodies	Levodopa Carbidopa Dopamine Agonists MAO-B Inhibitors Deep Brain Stimulation
Chorea	Involuntary, jerky, rapid movements Huntington's: cognitive decline, psychiatric Sydenham's: post-strep, children	Huntington's: CAG repeat, caudate atrophy Sydenham's: autoimmune	Huntington's: Tetrabenazine, antipsychotics Sydenham's: Penicillin, IVIG
Dystonia	Sustained muscle contractions, twisting, repetitive movements, abnormal postures	Complex, involving basal ganglia and other circuits, often idiopathic	Botulinum Toxin injections Anticholinergics DBS
Hemiballismus	Violent, flinging movements of one side of the body	Lesion of the contralateral Subthalamic Nucleus (STN), often stroke	Antipsychotics Treat underlying cause, sometimes DBS
Essential Tremor	Action tremor, hands/head, often improves with alcohol alcohol	Unknown, likely cerebello-thalamic pathways	Propranolol Primidone DBS

Figure 3.4: Basal ganglia circuitry and movement disorders including Parkinson's disease, chorea, dystonia, hemiballismus, and essential tremor

Pilot questions 3.4

1. Describe the direct and indirect pathways of the basal ganglia and explain how dopamine depletion causes Parkinson's disease. (Linked to SLO 3.4)
2. Describe the cardinal motor features of Parkinson's disease using the TRAP mnemonic. (Linked to SLO 3.4)
3. Describe the treatment of Parkinson's disease and explain the rationale for each drug class. (Linked to SLO 3.4)
4. Describe the clinical features and cause of Huntington's disease. (Linked to SLO 3.4)
5. Distinguish a resting tremor from a postural and action tremor and state a cause of each. (Linked to SLO 3.4)

Series Summary

This Series covered the cranial nerves, visual system, and extrapyramidal system. We began with CN I to IV: anosmia from CN I lesions; CN II examination (acuity, fields, disc); CN III palsy (ptosis, fixed dilated pupil, eye down and out); CN IV palsy (vertical diplopia on downgaze). CN V to VIII: facial sensory loss and jaw weakness from CN V lesions; corneal reflex (V1 afferent, VII efferent); CN VI palsy (failure of abduction, horizontal diplopia); Bell's palsy (LMN VII: entire ipsilateral face) versus UMN VII (forehead spared); CN VIII (sensorineural deafness and vertigo). CN IX to XII: gag reflex (IX afferent, X efferent); uvular deviation in CN X palsy (away from lesion); sternocleidomastoid and trapezius testing for CN XI; tongue deviation towards the lesion (LMN CN XII) versus away from the lesion (UMN). Bulbar versus pseudobulbar: bulbar (LMN: wasted fasciculating tongue, absent gag) versus pseudobulbar (bilateral UMN: brisk jaw jerk, emotional lability, spastic tongue).

The visual pathway: nasal fibres cross at the chiasm; optic tract carries contralateral field; radiation through temporal lobe (inferior fibres: Meyer's loop) and parietal lobe to visual cortex. Lesion patterns: optic nerve (monocular loss); chiasm (bitemporal hemianopia: pituitary adenoma); optic tract or radiation or cortex (homonymous hemianopia). RAPD detected by swinging flashlight test. INO from MLF lesion: failure of adduction with nystagmus: hallmark of MS. Extrapyramidal: direct pathway facilitates movement; indirect inhibits; dopamine depletion tips balance to excessive inhibition (Parkinson's disease: TRAP). Treatment: levodopa plus carbidopa, dopamine agonists, MAO-B inhibitors. Other disorders: chorea (Huntington's: CAG repeat; Sydenham's: rheumatic fever in Nigeria), dystonia (focal: botulinum toxin), hemiballismus (contralateral subthalamic nucleus lesion), essential tremor (propranolol or primidone).

Glossary of Terms

- **Bitemporal hemianopia:** loss of the temporal visual field in both eyes; pathognomonic of a lesion at the optic chiasm, most commonly caused by compression from a pituitary adenoma.
- **Bradykinesia:** abnormal slowness of voluntary movement; a cardinal feature of Parkinson's disease resulting from dopamine depletion in the nigrostriatal pathway.
- **Bulbar palsy:** weakness of the muscles supplied by cranial nerves IX to XII from a lower motor neuron lesion; characterised by a wasted, fasciculating tongue, nasal voice, dysphagia, and absent gag reflex.
- **Cogwheel rigidity:** a ratchet-like resistance to passive joint movement that is uniform throughout the range; characteristic of Parkinson's disease; caused by superimposition of tremor on lead-pipe rigidity.

- **Chorea:** brief, irregular, unpredictable involuntary movements that flow from one body part to another; caused by lesions or functional disturbance of the basal ganglia.
- **Homonymous hemianopia:** loss of the same half of the visual field in both eyes; indicates a lesion of the optic tract, optic radiation, or visual cortex contralateral to the field loss.
- **Internuclear ophthalmoplegia (INO):** a disorder of horizontal conjugate gaze caused by a lesion of the medial longitudinal fasciculus; produces failure of adduction of the ipsilateral eye with nystagmus of the abducting eye; the most characteristic eye finding in multiple sclerosis.
- **Levodopa:** the metabolic precursor of dopamine and the most effective symptomatic treatment for Parkinson's disease; always combined with a peripheral decarboxylase inhibitor (carbidopa or benserazide) to prevent peripheral conversion.
- **Pseudobulbar palsy:** bilateral upper motor neuron lesion of the corticobulbar tracts producing dysarthria and dysphagia with a brisk jaw jerk, exaggerated gag reflex, emotional lability, and a small spastic tongue without wasting or fasciculations.
- **Relative afferent pupillary defect (RAPD):** a sign of optic nerve dysfunction detected by the swinging flashlight test; the affected eye causes relative pupillary dilation when illuminated, indicating reduced afferent signal on that side.
- **TRAP:** a mnemonic for the cardinal motor features of Parkinson's disease: Tremor (resting, pill-rolling), Rigidity (cogwheel or lead-pipe), Akinesia or bradykinesia, and Postural instability.
- **Hemiballismus:** violent, flinging, involuntary movements of the proximal limbs on one side of the body caused by a lesion (usually a stroke) of the contralateral subthalamic nucleus.

Concept Check Questions [Series 3]

Objective questions

1. A complete CN III palsy produces ptosis, a fixed _____ pupil, and the eye deviated down and out. (Linked to SLO 3.1)
2. Bell's palsy is a _____ motor neuron lesion of CN VII affecting the entire ipsilateral face including the forehead. (Linked to SLO 3.1)

3. The hallmark sign of pseudobulbar palsy is a brisk _____ jerk, distinguishing it from bulbar palsy. (Linked to SLO 3.2)
4. A bitemporal hemianopia is pathognomonic of a lesion at the optic _____, most commonly a pituitary adenoma. (Linked to SLO 3.3)
5. The cardinal motor features of Parkinson's disease are summarised by the mnemonic _____, standing for Tremor, Rigidity, Akinesia, and Postural instability. (Linked to SLO 3.4)

Short-answer questions

6. Describe the clinical features of a complete CN III palsy and state two causes. (Linked to SLO 3.1)
7. Compare a LMN and a UMN lesion of CN VII. (Linked to SLO 3.1)
8. Compare bulbar and pseudobulbar palsy in terms of lesion type, tongue findings, jaw jerk, and gag reflex. (Linked to SLO 3.2)
9. Describe the visual field defect produced by a lesion of the optic chiasm and explain its anatomical basis. (Linked to SLO 3.3)
10. Describe the treatment of Parkinson's disease and explain the mechanism of levodopa. (Linked to SLO 3.4)

Long-answer questions

11. Describe the examination of cranial nerves V, VI, VII, and VIII and the deficits produced by lesions of each. (Linked to SLO 3.1)
12. Describe the visual pathway and the characteristic visual field defect produced by lesions at each level from the optic nerve to the visual cortex. (Linked to SLO 3.3)
13. Describe the clinical features, pathophysiology, and treatment of Parkinson's disease. (Linked to SLO 3.4)
14. Describe the causes and clinical features of chorea in the Nigerian context. (Linked to SLO 3.4)
15. Describe internuclear ophthalmoplegia: its anatomical basis, clinical features, and most common cause. (Linked to SLOs 3.1 and 3.3)

Answers to Concept Check Questions [Series 3]

Objective questions

1. Dilated.
2. Lower.
3. Jaw.
4. Chiasm.
5. TRAP.

Short-answer questions

6. A complete CN III palsy produces: ptosis (paralysis of levator palpebrae superioris), a fixed dilated pupil (parasympathetic palsy: loss of pupillary constriction), and the eye deviated down and out (unopposed action of the superior oblique CN IV and lateral rectus CN VI). Two causes: posterior communicating artery aneurysm (a surgical emergency: painful CN III palsy with pupil involvement) and diabetes mellitus (medical CN III palsy: typically pupil-sparing, from microvascular ischaemia of the nerve trunk).
7. LMN (Bell's palsy): affects the entire ipsilateral face including the forehead (complete ipsilateral facial paralysis: inability to raise the eyebrow, close the eye, and show the teeth on the affected side). UMN VII lesion (for example from a contralateral stroke): spares the forehead because the frontalis muscle has bilateral cortical representation; only the contralateral lower face is weak (nasolabial fold flattening and drooping of the corner of the mouth).
8. Bulbar palsy (LMN): wasted, fasciculating tongue; nasal voice; dysphagia with nasal regurgitation; absent or reduced gag reflex; jaw jerk normal or absent. Pseudobulbar palsy (bilateral UMN): small, spastic, slow tongue (no wasting or fasciculations); spastic dysarthria; dysphagia; brisk jaw jerk (the hallmark); exaggerated gag reflex; emotional lability (pathological laughing and crying).
9. A bitemporal hemianopia (loss of both temporal visual fields) is caused by a lesion at the optic chiasm. The anatomical basis: the nasal fibres from each retina (which convey the temporal visual field) cross at the chiasm. A lesion at the chiasm selectively damages these crossing fibres, interrupting the temporal visual field from both eyes

simultaneously. The most common cause is a pituitary adenoma expanding upwards from the sella turcica and compressing the chiasm from below.

10. Levodopa (combined with carbidopa or benserazide) is the most effective symptomatic treatment: levodopa crosses the blood-brain barrier and is converted to dopamine by dopa decarboxylase in the brain, replacing the depleted dopamine in the nigrostriatal pathway. Carbidopa inhibits peripheral dopa decarboxylase, preventing peripheral conversion to dopamine (which causes nausea, vomiting, and cardiac arrhythmias) and allowing more levodopa to enter the brain. Dopamine agonists (pramipexole, ropinirole) stimulate dopamine receptors directly. MAO-B inhibitors (selegiline, rasagiline) inhibit dopamine breakdown, prolonging its action.

Long-answer questions

11. **Basic response:** CN V (trigeminal): three divisions (V1 ophthalmic, V2 maxillary, V3 mandibular). Test: facial sensation with pinprick and light touch in all three divisions; corneal reflex (V1 afferent, VII efferent); jaw opening and clenching (V3 motor: masseter and temporalis). Lesion: facial sensory loss in the distribution of the affected division; V3 lesion causes jaw deviation towards the lesion. CN VI (abducens): test abduction of each eye; palsy causes failure of abduction and horizontal diplopia worst on lateral gaze towards the affected side. CN VII (facial): test forehead raising, eye closure, nasal flaring, mouth opening, and showing teeth; LMN palsy: complete ipsilateral facial weakness including forehead; UMN palsy: contralateral lower face only. CN VIII (vestibulocochlear): test hearing with whispered voice or tuning fork (Rinne and Weber tests: sensorineural deafness: Rinne positive, Weber lateralises away from the affected ear); vestibular: nystagmus and past-pointing and ataxia.

Value-added response: The Rinne test: a 512 Hz tuning fork is placed on the mastoid (bone conduction) then held beside the ear (air conduction). Normally air conduction is better than bone conduction (Rinne positive). In conductive deafness, bone conduction is better (Rinne negative). In sensorineural deafness, both are reduced but air conduction remains better than bone conduction (Rinne positive on the affected side). Weber test: a tuning fork on the vertex lateralises to the worse ear in conductive deafness and to the better ear in sensorineural deafness.

12. **Basic response:** Optic nerve (before the chiasm): monocular visual loss (one eye only) with ipsilateral RAPD. Optic chiasm: bitemporal hemianopia (loss of both temporal fields) from damage to the crossing nasal fibres; most commonly a pituitary adenoma. Optic tract (after the chiasm): contralateral homonymous hemianopia (loss of the same half field in both eyes) with an incongruous field defect (the two eyes have slightly different sized defects). Optic radiation: the fibres split into temporal (inferior: Meyer's

loop) and parietal (superior) pathways. Temporal lobe radiation lesion: contralateral upper quadrant homonymous defect (pie in the sky). Parietal lobe radiation lesion: contralateral lower quadrant homonymous defect. Visual cortex: contralateral macular-sparing homonymous hemianopia (the macula has a large cortical representation and a dual blood supply from the middle and posterior cerebral arteries).

Value-added response: Macular sparing in occipital lobe infarction has important clinical and medico-legal implications: a patient with a complete homonymous hemianopia but with macular sparing retains some central vision in the affected field and may be judged to retain sufficient visual acuity for driving in some jurisdictions, unlike a patient with a complete field defect including the macula.

13. **Basic response:** Pathophysiology: progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, with Lewy body (misfolded alpha-synuclein) formation; dopamine depletion disrupts the direct-indirect pathway balance in the basal ganglia, causing excessive inhibition of the thalamus and reduced cortical motor activation. Clinical features TRAP: resting pill-rolling tremor (4 to 6 Hz, suppressed by voluntary movement), cogwheel or lead-pipe rigidity (uniform throughout range), akinesia or bradykinesia (micrographia, reduced arm swing, hypomimia, hypophonia), and postural instability (falls). Non-motor: anosmia, REM sleep behaviour disorder, autonomic dysfunction, depression, and late dementia. Treatment: levodopa plus carbidopa (most effective), dopamine agonists (younger patients: delay levodopa), MAO-B inhibitors (neuroprotective claim; additive benefit), physiotherapy, and deep brain stimulation (subthalamic nucleus) for refractory motor fluctuations.

Value-added response: Deep brain stimulation (DBS) of the subthalamic nucleus is the most effective surgical treatment for Parkinson's disease with motor complications: it reduces tremor, rigidity, and levodopa-induced dyskinesias and allows significant reduction in levodopa dose; it does not cure the disease or slow neurodegeneration but dramatically improves quality of life in carefully selected patients.

14. **Basic response:** Chorea is brief, irregular, unpredictable involuntary movements flowing from one body part to another. Causes in Nigeria: Huntington's disease (autosomal dominant; CAG trinucleotide repeat expansion in the HTT gene on chromosome 4; progressive chorea with dementia and psychiatric disturbance beginning in the fourth or fifth decade; family history of progressive neurological disease and psychiatric problems; no disease-modifying treatment: manage chorea with tetrabenazine or antipsychotics; genetic counselling essential). Sydenham's chorea (a complication of acute rheumatic fever caused by group A Streptococcus: affects children in Nigeria; involuntary movements with emotional lability and muscle weakness: treat the underlying rheumatic disease with penicillin and manage chorea with haloperidol or sodium valproate). Drug-

induced chorea: levodopa overdose (peak-dose dyskinesias); tardive dyskinesia from long-term antipsychotic use; SLE-associated chorea.

Value-added response: In a Nigerian child presenting with chorea, Sydenham's chorea from rheumatic fever must always be considered first and throat swabs, ASO titre, and echocardiography should be performed to confirm the diagnosis and assess for rheumatic heart disease, which is the main cause of mortality and long-term morbidity in rheumatic fever.

15. **Basic response:** Anatomical basis: horizontal conjugate gaze to the right requires activation of the right PPRF, which projects to the right CN VI nucleus (abducting the right eye) and, via the right MLF, to the left CN III nucleus (adducting the left eye). A left MLF lesion interrupts the signal from the left PPRF to the left CN III nucleus during rightward gaze: the left eye fails to adduct (internuclear ophthalmoplegia of the left eye), while the right eye abducts normally but develops nystagmus from the loss of the balancing adduction signal. Clinical features: on lateral gaze to the right, the left eye fails to cross the midline (adduction palsy) and the right eye shows horizontal nystagmus; convergence may be intact (because convergence uses a separate pathway). Bilateral INO in a young adult is the most characteristic eye finding in multiple sclerosis.

Value-added response: In an older patient, a unilateral INO is more likely to be caused by a brainstem infarct (lacunar or small vessel territory pontine infarct). In a young patient with bilateral INO, multiple sclerosis must be the leading diagnosis and MRI brain with gadolinium should be performed urgently to look for periventricular and juxtacortical white matter plaques.

Answers to Pilot Questions [Series 3]

Part 3.1

1. CN III and IV nuclei lie in the midbrain (CN III at the superior colliculus level, CN IV just below). CN V, VI, VII, and VIII nuclei are in the pons. CN IX, X, XI, and XII nuclei are in the medulla oblongata. This arrangement means that a lesion at any given brainstem level will damage the cranial nerve nuclei at that level, producing ipsilateral cranial nerve signs, combined with contralateral long-tract signs (the crossed deficit pattern).
2. A complete CN III palsy produces three features: (1) ptosis (levator palpebrae superioris paralysis), (2) a fixed dilated pupil (loss of parasympathetic pupillary constriction), and (3) the eye deviated down and out (due to the unopposed action of the superior oblique CN IV which intorts and depresses the eye, and the lateral rectus CN VI which abducts

it). Two causes: posterior communicating artery aneurysm (painful, surgical emergency, pupil involved: parasympathetic fibres run on the outside of CN III and are compressed first) and diabetes mellitus (usually painless, pupil-sparing: microvascular ischaemia affects the core fibres first).

3. LMN CN VII lesion (Bell's palsy): the entire ipsilateral face is paralysed from forehead to chin, because the LMN nucleus in the pons supplies all the ipsilateral facial muscles without any contralateral input. UMN CN VII lesion (for example from a contralateral stroke): only the contralateral lower face is weak, because the upper face (frontalis) has bilateral cortical representation and is therefore protected from a unilateral cortical lesion. The key distinguishing bedside test is asking the patient to raise their eyebrows: in Bell's palsy the ipsilateral eyebrow cannot be raised; in UMN VII palsy both eyebrows can be raised.
4. CN V has three sensory divisions: V1 (ophthalmic: forehead, cornea, upper nasal mucosa), V2 (maxillary: cheek, upper teeth, palate), and V3 (mandibular: lower face, lower teeth, anterior two-thirds of tongue: sensation only). V3 also carries motor fibres to the muscles of mastication (masseter, temporalis, medial and lateral pterygoids). A CN V lesion causes loss of sensation in the distribution of the affected division and loss of the corneal reflex (afferent limb). A V3 motor lesion causes weakness of jaw clenching and lateral jaw movement, with deviation of the jaw towards the side of the lesion on opening (the intact pterygoids push the jaw towards the weak side).
5. Foster-Kennedy syndrome is caused by a mass lesion at the base of the frontal lobe, classically an olfactory groove meningioma. The mass compresses the ipsilateral olfactory nerve (CN I: causing unilateral anosmia) and the ipsilateral optic nerve (CN II: causing ipsilateral optic atrophy from direct compression), while raising intracranial pressure sufficiently to cause papilloedema (swelling of the optic disc from raised ICP) in the contralateral eye. The triad is therefore: ipsilateral anosmia, ipsilateral optic atrophy, and contralateral papilloedema.

Part 3.2

1. A unilateral CN X palsy causes: hoarseness (paralysis of the ipsilateral vocal cord from recurrent laryngeal nerve involvement: the voice is breathy and hoarse), ipsilateral palatal weakness (the soft palate droops on the affected side), and uvular deviation away from the side of the lesion (the intact contralateral palatal muscles pull the uvula towards themselves). Dysphagia may occur if the pharyngeal constrictors are also affected. The gag reflex is impaired on the affected side (CN X carries the efferent limb).

2. Test CN XI by: sternocleidomastoid (ask the patient to turn their head to the contralateral side against the resistance of your hand placed against their cheek: the contracting sternocleidomastoid is visible and palpable on the ipsilateral side) and trapezius (ask the patient to shrug both shoulders against resistance). In a CN XI palsy: weakness of head turning towards the contralateral side (the ipsilateral sternocleidomastoid is weak), drooping of the ipsilateral shoulder (trapezius weakness), and winging of the ipsilateral scapula.
3. LMN CN XII lesion: ipsilateral tongue wasting and fasciculations (denervation atrophy); the tongue deviates towards the side of the lesion on protrusion (the intact contralateral genioglossus muscle pushes the tongue towards the weaker side). UMN CN XII lesion (for example from a contralateral stroke): the tongue is small and spastic (no wasting or fasciculations); the tongue deviates away from the side of the lesion on protrusion (towards the side of the hemiplegia), because the weakened ipsilateral cortical projection allows the contralateral genioglossus to push the tongue to the affected side.
4. Bulbar palsy (LMN): nasal or hypernasal voice; dysarthria (quiet, nasal, slurred speech); dysphagia with nasal regurgitation of liquids; tongue wasted and fasciculating; jaw jerk normal or absent; gag reflex absent. Pseudobulbar palsy (bilateral UMN): spastic dysarthria (strained, strangled, slow speech); dysphagia; tongue small, spastic, and slow (no wasting or fasciculations); jaw jerk brisk (the hallmark); gag reflex exaggerated; emotional lability (pathological laughing and crying out of proportion to the emotional stimulus).
5. The hallmark sign of pseudobulbar palsy is a brisk jaw jerk (hyperreflexic jaw jerk). The anatomical basis: the jaw jerk is a stretch reflex mediated by the trigeminal nerve (CN V), whose motor nucleus is normally under bilateral cortical inhibition via the corticobulbar tracts. When both corticobulbar tracts are damaged (bilateral UMN lesion), the inhibitory control over the trigeminal motor nucleus is lost, and the jaw jerk becomes brisk or even clonic, exactly analogous to the way in which UMN limb lesions cause hyperreflexia and clonus.

Part 3.3

1. Light from the environment enters the eye and is detected by photoreceptors in the retina. Electrical signals travel along the optic nerve (CN II) to the optic chiasm, where nasal (medial) retinal fibres cross to the opposite side and temporal (lateral) fibres remain ipsilateral. The fibres then travel as the optic tract to the lateral geniculate nucleus of the thalamus. From there, the optic radiation carries fibres through the parietal lobe (superior fibres: upper visual field) and temporal lobe (inferior fibres: lower visual field: Meyer's

loop) to the primary visual cortex in the occipital lobe (Brodmann area 17). Each visual cortex processes the contralateral visual field from both eyes.

2. A bitemporal hemianopia occurs because the fibres that cross at the chiasm arise from the nasal half of each retina, which receives light from the temporal visual field. The optic chiasm lies directly above the pituitary gland, and an expanding pituitary adenoma compresses the chiasm from below, selectively interrupting the crossing nasal fibres while sparing the uncrossed temporal fibres. The result is loss of the temporal visual field in both eyes (bitemporal hemianopia), creating the characteristic tunnel vision.
3. The swinging flashlight test is performed in a dimly lit room: shine a bright light into one eye for 2 to 3 seconds and observe both pupils (they should both constrict), then rapidly swing the light to the other eye. Normally, both pupils maintain constriction when the light is swung. A positive RAPD (Marcus Gunn pupil): when the light is swung to the affected eye, both pupils dilate (rather than maintaining constriction), because the optic nerve on the affected side generates a weaker afferent signal, so the Edinger-Westphal nucleus receives less input and reduces its parasympathetic output to both pupils. A RAPD indicates an optic nerve lesion (optic neuritis, glaucoma, anterior ischaemic optic neuropathy) on the affected side.
4. Internuclear ophthalmoplegia is caused by a lesion of the medial longitudinal fasciculus (MLF), which connects the CN VI nucleus in the pons to the contralateral CN III nucleus, coordinating horizontal conjugate gaze. The most common cause is multiple sclerosis (bilateral INO in a young adult is the most characteristic eye finding in MS; the MLF is vulnerable to demyelination because it is a long, slender white matter tract). Other causes include pontine infarction (in older patients with vascular risk factors) and Wernicke's encephalopathy.
5. A parietal lobe optic radiation lesion damages the superior fibres of the radiation (which carry signals from the inferior visual field); it therefore causes a contralateral lower quadrant homonymous visual field defect (inferior quadrantanopia). A temporal lobe optic radiation lesion damages the inferior fibres of the radiation (Meyer's loop, which loops into the temporal lobe and carries signals from the superior visual field); it causes a contralateral upper quadrant homonymous visual field defect (superior quadrantanopia), nicknamed pie in the sky.

Part 3.4

1. The direct pathway (striatum to globus pallidus internal to thalamus: disinhibitory) facilitates movement by releasing the thalamus from inhibition, allowing the thalamus to activate the motor cortex. The indirect pathway (striatum to globus pallidus external to

subthalamic nucleus to globus pallidus internal to thalamus: inhibitory) suppresses unwanted movement by increasing inhibition of the thalamus. Dopamine from the substantia nigra stimulates the direct pathway (via D1 receptors) and inhibits the indirect pathway (via D2 receptors), so dopamine depletion in Parkinson's disease simultaneously underactivates the direct pathway and overactivates the indirect pathway. The net result is excessive inhibition of the thalamus, reduced cortical motor activation, and the clinical features of bradykinesia and rigidity.

2. TRAP: Tremor (resting, pill-rolling, 4 to 6 Hz, typically in the hands or feet, present at rest and suppressed by voluntary movement, worsened by stress and anxiety, unlike the action and postural tremor of essential tremor). Rigidity (cogwheel or lead-pipe uniform resistance to passive joint movement throughout the full range in all directions, present in both flexion and extension, unlike spasticity which is velocity-dependent). Akinesia or bradykinesia (slowness of initiation and execution of voluntary movement: micrographia, hypomimia facial masking, hypophonia, festinant gait). Postural instability (impaired righting reflexes tested by the pull test: examiner pulls the patient's shoulders from behind: more than two steps backward or falling indicates impaired postural reflexes).

3. Levodopa plus carbidopa (or benserazide): levodopa is the precursor to dopamine and crosses the blood-brain barrier; carbidopa inhibits peripheral dopa decarboxylase, reducing peripheral dopamine production and nausea while increasing central levodopa availability. Dopamine agonists (pramipexole, ropinirole, rotigotine): directly stimulate D2 and D3 receptors; preferred in younger patients (below 65 years) to delay levodopa and reduce the risk of motor fluctuations and dyskinesias; side effects include impulse control disorders (gambling, hypersexuality). MAO-B inhibitors (selegiline, rasagiline): inhibit monoamine oxidase B, the main enzyme degrading dopamine in the brain; used as adjunctive therapy; modest symptomatic benefit. Non-pharmacological: physiotherapy (gait and balance), speech therapy (hypophonia), occupational therapy.

4. Huntington's disease is an autosomal dominant neurodegenerative disease caused by an expansion of a CAG trinucleotide repeat in exon 1 of the HTT gene on chromosome 4 (normal: fewer than 36 repeats; pathological: above 36 repeats; above 60 repeats: juvenile onset). The expansion encodes a polyglutamine tract in the huntingtin protein, causing toxic gain of function and progressive neuronal loss in the striatum (caudate and putamen) and cortex. Clinical features: chorea (the presenting feature: brief, irregular, flowing involuntary movements that become more generalised over time), progressive dementia (frontosubcortical: executive function, attention, and memory affected first), and psychiatric disturbance (depression, anxiety, obsessive-compulsive disorder, and psychosis: often preceding the movement disorder). No disease-modifying treatment exists; manage chorea with tetrabenazine or antipsychotics (haloperidol), and psychiatric symptoms with appropriate psychotropic medication.

5. Resting tremor: present when the limb is at rest and supported, and suppressed by voluntary movement; characteristic of Parkinson's disease (4 to 6 Hz pill-rolling tremor of the hand) and caused by dopamine depletion in the nigrostriatal pathway. Postural and action tremor: present when the limb is held against gravity (postural) or during voluntary movement (action), and not present at rest; characteristic of essential tremor (the most common movement disorder: 6 to 12 Hz, often involving the hands and head, autosomal dominant in 50 percent of cases, improved by alcohol) and treated with propranolol or primidone. The distinction is made clinically by observing whether the tremor is present at rest (Parkinson's) or with posture and movement (essential tremor).

References and Attributions [Series 3]

Textual References

- Fuller, G. and Manford, M. (2010). Neurology: An Illustrated Colour Text (4th ed.). Edinburgh: Churchill Livingstone.
- Kandel, E. R., Schwartz, J. H., Jessell, T. M., Siegelbaum, S. A. and Hudspeth, A. J. (2013). Principles of Neural Science (5th ed.). New York: McGraw-Hill.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- Longmore, M., Wilkinson, I., Baldwin, A. and Wallin, E. (2014). Oxford Handbook of Clinical Medicine (9th ed.). Oxford: Oxford University Press.

Figures, Plates, Tables, and Boxes

- Figure 3.1: Cranial nerves I to VIII: functions and palsy patterns. AI-generated using the prompt: "Two-panel diagram: left base of brain with CN I to VIII labelled and a table of functions; right clinical palsy illustrations for CN III, IV, VI, and VII (UMN versus LMN)." Suggested OER search string: "cranial nerves I to VIII functions palsy diagram base of brain OER."
- Figure 3.2: Cranial nerves IX to XII and bulbar versus pseudobulbar palsy. AI-generated using the prompt: "Two-panel diagram: left four-row table for CN IX to XII with

functions and palsy features; right two-column comparison of bulbar and pseudobulbar palsy." Suggested OER search string: "cranial nerves IX X XI XII bulbar pseudobulbar palsy comparison diagram OER."

- Figure 3.3: The visual pathway with visual field defects, pupillary reflexes, and INO. AI-generated using the prompt: "Two-panel diagram: left visual pathway from above with six lesion sites and corresponding visual field defects; right pupillary light reflex arc with RAPD and MLF connections with INO diagram." Suggested OER search string: "visual pathway diagram lesion visual field defects pupillary reflex INO OER."
- Figure 3.4: Basal ganglia circuitry and movement disorders. AI-generated using the prompt: "Two-panel diagram: left basal ganglia circuit with direct and indirect pathways and dopamine depletion effect; right five-row table of movement disorders (Parkinson's, chorea, dystonia, hemiballismus, essential tremor)." Suggested OER search string: "basal ganglia circuit Parkinson's disease movement disorders chorea dystonia hemiballismus diagram OER."

Course code: MED 408

Course title: Neurology I

Course credit load: 2 Units

Series 4: Common Neurological Symptoms

- **Part 4.1: Headache**
    - Sub Part 4.1.1: Approach to headache
    - Sub Part 4.1.2: Primary headache disorders
    - Sub Part 4.1.3: Secondary headache disorders
- **Part 4.2: Seizures and Epilepsy**
    - Sub Part 4.2.1: Classification of seizures
    - Sub Part 4.2.2: Epilepsy syndromes and diagnosis
    - Sub Part 4.2.3: Management of epilepsy
- **Part 4.3: Dizziness and Vertigo**
    - Sub Part 4.3.1: Peripheral vestibular disorders
    - Sub Part 4.3.2: Central causes of dizziness and vertigo
    - Sub Part 4.3.3: Approach to the dizzy patient
- **Part 4.4: Weakness and Sensory Disturbance**
    - Sub Part 4.4.1: Approach to weakness
    - Sub Part 4.4.2: Peripheral neuropathy
    - Sub Part 4.4.3: Approach to sensory disturbance

Introduction

Headache, seizures, dizziness, weakness, and sensory disturbance are among the most common reasons patients seek neurological attention. Knowing how to approach each symptom systematically, from history through examination to investigation and management, is the practical currency of clinical neurology.

This Series examines these common neurological symptoms in turn. We begin with headache, distinguishing the dangerous from the benign and examining the major primary headache disorders. We then cover seizures and epilepsy, including classification, diagnosis, and pharmacological management. Moving on, we address dizziness and vertigo, and we close with a structured approach to weakness and sensory disturbance.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 4.1:** take a structured history in a patient presenting with headache and distinguish primary from secondary headache disorders,
- **SLO 4.2:** classify seizures, describe the major epilepsy syndromes, and outline the management of epilepsy,
- **SLO 4.3:** distinguish peripheral from central causes of dizziness and vertigo and describe the clinical approach to the dizzy patient, and
- **SLO 4.4:** apply a structured approach to weakness and sensory disturbance and describe the clinical features of peripheral neuropathy.

4.1 Headache

4.1.1 Approach to headache

Headache is one of the most common symptoms in medicine, and the clinician's primary task is to distinguish the dangerous secondary headache from the benign primary headache. The history is the most important tool. Key features to characterise: onset (thunderclap onset, reaching maximal intensity within seconds, is a neurological emergency suggesting subarachnoid haemorrhage until proven otherwise), site, character, severity, duration, frequency, associated features (nausea, photophobia, phonophobia, neck stiffness, fever, focal neurology, visual changes), and triggers.

Red flag features that mandate urgent investigation include: thunderclap onset, headache with fever and neck stiffness (meningitis), headache with focal neurological signs, new headache in a patient over 50 years, headache worsening progressively over days to weeks, headache waking the patient from sleep, and headache in a patient with known malignancy or HIV (which raises the possibility of CNS metastases or opportunistic infection).

Fill in the blank: A headache reaching maximal intensity within _____ is called a thunderclap headache and is a neurological emergency until _____ haemorrhage has been excluded.

4.1.2 Primary headache disorders

Migraine is the most common disabling primary headache. It presents with recurrent attacks of moderate to severe unilateral throbbing headache lasting 4 to 72 hours, accompanied by nausea and vomiting and sensitivity to light (photophobia) and sound (phonophobia), worsened by physical activity. Approximately one-third of migraineurs experience an aura: a reversible focal neurological symptom that precedes or accompanies the headache, most commonly a visual aura (zigzag lines, scintillating scotoma). Acute treatment: analgesics (paracetamol, NSAIDs), triptans (5-HT1B/1D agonists: sumatriptan) for moderate to severe attacks. Prophylaxis for frequent attacks: propranolol, amitriptyline, topiramate, or sodium valproate.

Tension-type headache is the most prevalent headache overall: bilateral, pressing or tightening in quality (band-like), mild to moderate severity, not worsened by physical activity, and without prominent nausea, photophobia, or phonophobia. It is managed with simple analgesics and lifestyle measures. Cluster headache is rare but intensely painful: strictly unilateral orbital or periorbital pain, lasting 15 to 180 minutes, occurring in clusters of attacks over weeks followed by remission periods, associated with ipsilateral autonomic features (lacrimation, nasal congestion, miosis, ptosis, facial flushing). Acute treatment: high-flow oxygen (100 percent, 10 to 15 L/min for 15 minutes) and sumatriptan injection.

Fill in the blank: Migraine aura is a reversible focal neurological symptom, most commonly _____ (zigzag lines or scintillating scotoma). Cluster headache is associated with ipsilateral autonomic features and is acutely treated with high-flow _____ and sumatriptan.

4.1.3 Secondary headache disorders

Subarachnoid haemorrhage (SAH) classically presents as a sudden, severe thunderclap headache described as the worst headache of the patient's life, often with nausea, vomiting, neck stiffness, photophobia, and transient or persistent loss of consciousness. The most common cause is rupture of an intracranial saccular (berry) aneurysm. CT brain without contrast is the first investigation (sensitivity above 98 percent within 6 hours); if CT is negative, lumbar puncture is performed to detect xanthochromia. Confirmed SAH requires urgent neurosurgical referral for aneurysm clipping or endovascular coiling.

Meningitis presents with headache, fever, neck stiffness, and photophobia (the meningeal triad); the non-blanching petechial or purpuric rash of meningococcal meningitis is a medical emergency requiring immediate IV benzylpenicillin. Raised intracranial pressure (from hydrocephalus, tumour, or idiopathic intracranial hypertension) produces a chronic progressive headache worse in the morning, on straining, or on lying down, associated with papilloedema and sixth nerve palsy (false localising sign). Temporal arteritis (giant cell arteritis) in patients over 50 causes unilateral temporal headache with scalp tenderness, jaw claudication, and

elevated ESR; urgent high-dose prednisolone prevents irreversible visual loss from ischaemic optic neuropathy.

Fill in the blank: Subarachnoid haemorrhage presents as the worst headache of the patient's life with _____ onset. Temporal arteritis requires urgent _____ to prevent irreversible visual loss.

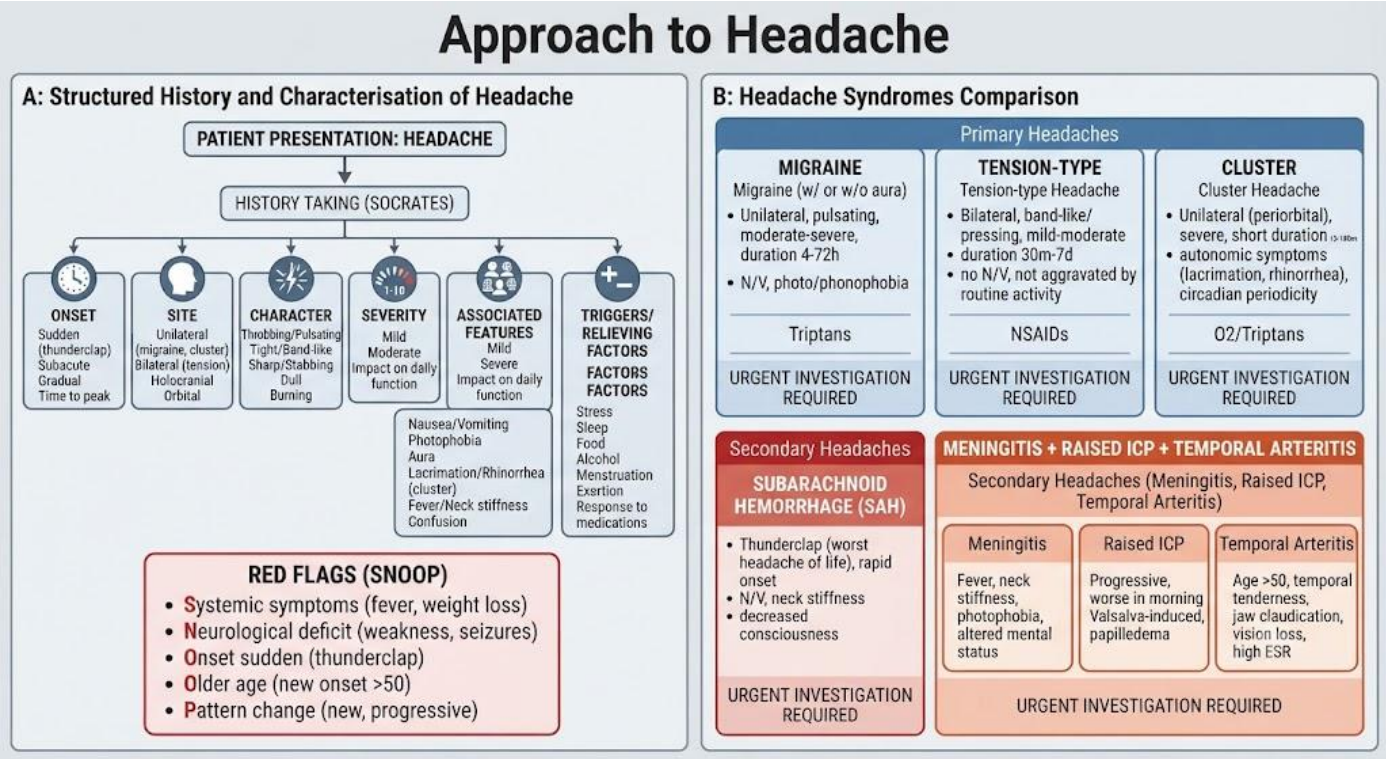


Figure 4.1: Structured approach to headache: history, red flags, and primary versus secondary headache disorders

Pilot questions 4.1

1. State four red flag features that mandate urgent investigation in a patient with headache. (Linked to SLO 4.1)
2. Describe the clinical features of migraine and distinguish it from tension-type headache. (Linked to SLO 4.1)
3. Describe the clinical features of cluster headache and state the acute treatment. (Linked to SLO 4.1)
4. Describe the investigation of a patient with suspected subarachnoid haemorrhage. (Linked to SLO 4.1)
5. Describe temporal arteritis and explain why it is a medical emergency. (Linked to SLO 4.1)

4.2 Seizures and Epilepsy

4.2.1 Classification of seizures

A seizure is a transient episode of abnormal, excessive, or synchronous neuronal activity in the brain, manifesting as a sudden change in behaviour, movement, sensation, or consciousness. Seizures are classified by their onset: focal (previously called partial) seizures begin in one hemisphere and produce symptoms referable to the region of onset, with or without alteration of awareness; generalised seizures begin simultaneously across both hemispheres and always involve impairment of consciousness. A focal seizure may secondarily generalise, spreading to involve both hemispheres and producing a tonic-clonic seizure.

Generalised seizures include absence seizures (brief episodes of staring and unresponsiveness lasting 5 to 30 seconds without a post-ictal phase; characteristic 3 Hz spike-and-wave on EEG), myoclonic seizures (sudden brief muscle jerks, often in the morning), tonic seizures (sudden stiffening), atonic seizures (drop attacks: sudden loss of postural tone), and generalised tonic-clonic (GTC) seizures (the classic convulsion: tonic phase with stiffening and apnoea followed by clonic phase with rhythmic jerking, then post-ictal confusion and drowsiness). The post-ictal period (confusion and drowsiness lasting minutes to hours after a seizure) is an important feature distinguishing a seizure from a syncopal episode.

Fill in the blank: Focal seizures begin in _____ hemisphere, while generalised seizures begin simultaneously across _____. The _____ period of confusion and drowsiness after a GTC seizure helps distinguish it from syncope.

4.2.2 Epilepsy syndromes and diagnosis

Epilepsy is defined as a disorder of the brain characterised by an enduring predisposition to generate seizures. The diagnosis requires at least two unprovoked seizures occurring more than 24 hours apart (or one unprovoked seizure with a risk of recurrence above 60 percent). Epilepsy syndromes are classified by the age of onset, seizure type, EEG findings, and aetiology. Common syndromes include childhood absence epilepsy (absence seizures with 3 Hz spike-and-wave, remits in adolescence), juvenile myoclonic epilepsy (the most common genetic epilepsy: onset in adolescence, morning myoclonus, GTC seizures, requires lifelong treatment), and temporal lobe epilepsy (the most common focal epilepsy in adults: often associated with hippocampal sclerosis from perinatal injury or febrile convulsions; auras of a rising epigastric sensation, deja vu, or fear).

Diagnosis of epilepsy requires a careful clinical history, an EEG (shows epileptiform discharges: normal EEG does not exclude epilepsy), and MRI brain to identify structural causes (mesial temporal sclerosis, cortical dysplasia, tumours, vascular malformations). In Nigeria, common causes of symptomatic epilepsy include neurocysticercosis (Taenia solium larvae in the brain: a leading cause of new-onset seizures in adults), cerebral malaria (as an acute symptomatic seizure), tuberculous meningitis (with cortical involvement), stroke, and traumatic brain injury.

Fill in the blank: Epilepsy requires at least _____ unprovoked seizures more than 24 hours apart. In Nigeria, _____ (Taenia solium larvae in the brain) is a leading cause of new-onset seizures in adults.

4.2.3 Management of epilepsy

The goal of epilepsy treatment is complete seizure freedom with minimal side effects, using the lowest effective dose of a single antiepileptic drug (monotherapy). First-line drugs are chosen according to seizure type: sodium valproate (the most broad-spectrum antiepileptic: effective for generalised and focal seizures; teratogenic and should be avoided in women of childbearing age if possible), lamotrigine and levetiracetam (broad-spectrum; safer in women of childbearing age), carbamazepine and oxcarbazepine (focal seizures: avoid in generalised epilepsies as they can worsen absence and myoclonic seizures), and ethosuximide (absence seizures only). If monotherapy fails, a second drug is added before switching to combination therapy.

Patients with epilepsy must receive safety counselling: avoid driving until seizure-free for the required period (12 months in Nigeria as per road traffic regulations), avoid heights and unsupervised swimming, take showers rather than baths, inform relevant authorities of the diagnosis, and take medication consistently. Status epilepticus is a neurological emergency defined as a seizure lasting more than 5 minutes or two seizures without full recovery between them: give IV lorazepam (or diazepam if IV access is unavailable) immediately, followed by IV phenytoin or valproate if the seizure continues, and general anaesthesia with midazolam, propofol, or thiopentone for refractory status.

Fill in the blank: The most broad-spectrum antiepileptic drug is sodium _____, but it is _____ and should be avoided in women of childbearing age if possible. Status epilepticus is defined as a seizure lasting more than _____ minutes.

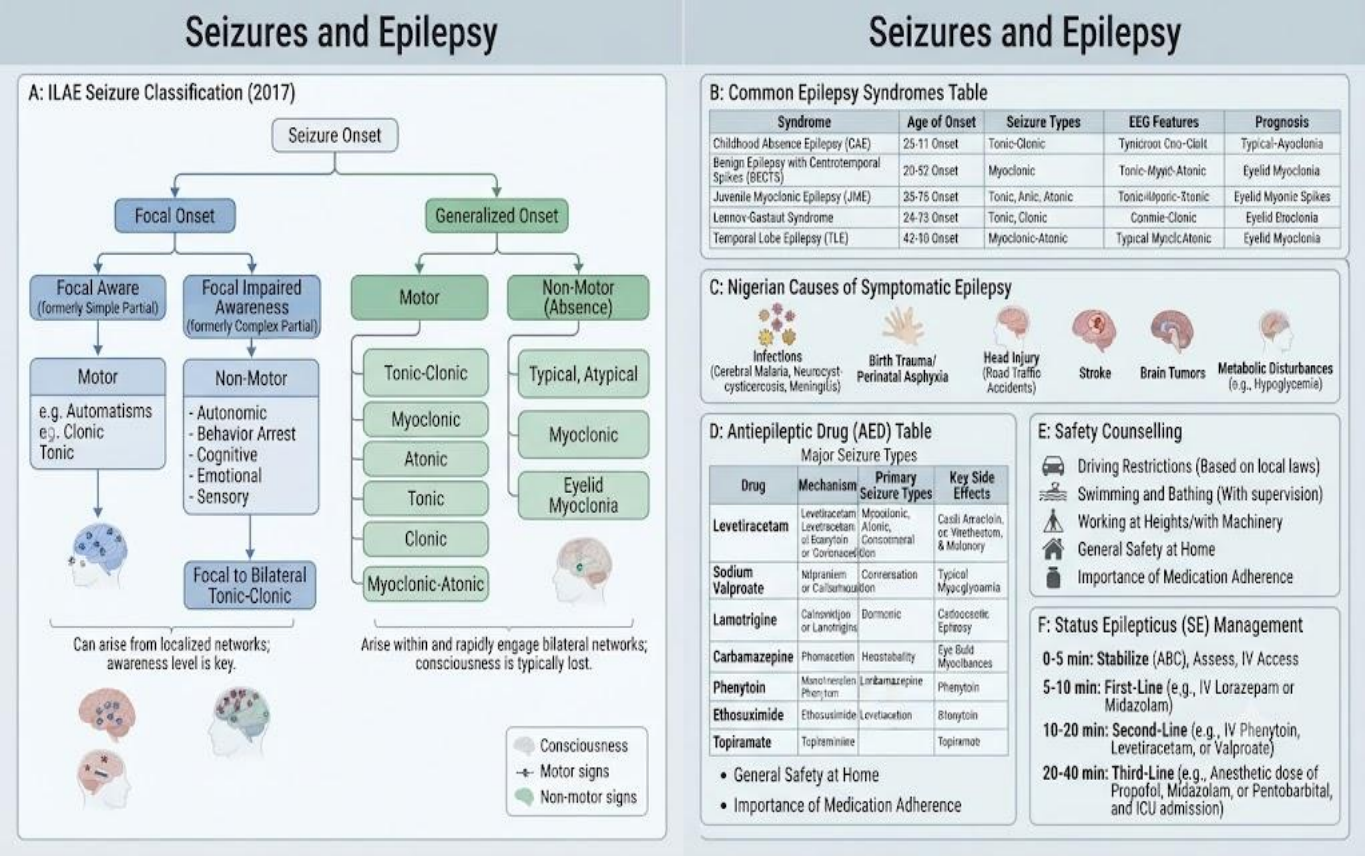


Figure 4.2: Seizure classification, epilepsy syndromes, and management

Pilot questions 4.2

1. Distinguish focal from generalised seizures and describe the post-ictal period. (Linked to SLO 4.2)
2. Describe the clinical and EEG features of childhood absence epilepsy. (Linked to SLO 4.2)
3. State five causes of symptomatic epilepsy relevant to Nigerian clinical practice. (Linked to SLO 4.2)
4. Describe the first-line antiepileptic drug for each major seizure type. (Linked to SLO 4.2)
5. Define status epilepticus and describe its immediate management. (Linked to SLO 4.2)

4.3 Dizziness and Vertigo

4.3.1 Peripheral vestibular disorders

Peripheral vertigo arises from disorders of the inner ear or vestibular nerve and is characterised by severe vertigo that is often positional, accompanied by nausea and vomiting, horizontal nystagmus with a rotational component (fast phase away from the affected ear), and hearing symptoms in some conditions. Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo overall: brief episodes (seconds) triggered by changes in head position (for example rolling over in bed) from displaced otoconia (calcium crystals) in the semicircular canals. Diagnosis is confirmed by the Dix-Hallpike manoeuvre (the affected ear is placed downward: positive if this reproduces the vertigo with a burst of nystagmus after a short latency). Treatment is the Epley manoeuvre (a series of head movements to reposition the otoconia).

Vestibular neuritis is sudden-onset severe continuous vertigo without hearing loss, lasting days, caused by inflammation (often post-viral) of the vestibular nerve; treated with vestibular sedatives (prochlorperazine) acutely and vestibular exercises for rehabilitation. Meniere's disease is a triad of episodic vertigo (lasting 20 minutes to 24 hours), fluctuating sensorineural hearing loss, and tinnitus, caused by endolymphatic hydrops (excess fluid in the inner ear); treated with a low-salt diet, thiazide diuretics, and betahistine.

Fill in the blank: BPPV is diagnosed by the _____ manoeuvre and treated by the _____ manoeuvre. Meniere's disease is a triad of episodic vertigo, fluctuating sensorineural _____ loss, and tinnitus.

4.3.2 Central causes of dizziness and vertigo

Central vertigo arises from lesions of the brainstem or cerebellum and must always be excluded, as it may indicate a serious and potentially life-threatening cause. Features suggesting central vertigo: abrupt onset with severe neurological symptoms disproportionate to the vertigo, vertical nystagmus or nystagmus that does not suppress with visual fixation, other brainstem or cerebellar signs (diplopia, dysarthria, dysphagia, ataxia, facial numbness, Horner's syndrome), failure to improve over days, and risk factors for stroke (hypertension, diabetes, atrial fibrillation, smoking). The most important central cause to exclude is a posterior fossa stroke or TIA: a cerebellar or lateral medullary (Wallenberg syndrome) infarct.

Wallenberg syndrome (lateral medullary syndrome) from posterior inferior cerebellar artery (PICA) occlusion produces: ipsilateral facial pain and temperature loss (trigeminal nucleus), ipsilateral Horner's syndrome (descending sympathetic fibres), ipsilateral cerebellar ataxia, and contralateral limb pain and temperature loss (spinothalamic tract), together with vertigo, nausea, dysphagia, and hoarseness. MRI brain (diffusion-weighted imaging) is the investigation of choice for posterior fossa stroke and is far superior to CT in the posterior fossa.

Fill in the blank: Vertical nystagmus that does not suppress with visual fixation suggests _____ vertigo. Wallenberg syndrome is caused by occlusion of the _____ inferior cerebellar artery.

4.3.3 Approach to the dizzy patient

The clinical approach to dizziness begins with clarifying what the patient means: vertigo (a sense of rotational movement: vestibular disease), presyncope (a feeling of impending faint: cardiovascular or metabolic), or disequilibrium (unsteadiness without vertigo: proprioceptive or cerebellar disease). The most important clinical question in a patient with vertigo is: peripheral or central? Central vertigo mandates urgent MRI brain.

Distinguishing features: peripheral vertigo is severe, positional, associated with horizontal-rotational nystagmus that suppresses with fixation, accompanied by hearing symptoms in some cases (Meniere's), and the patient is able to walk (albeit unsteadily). Central vertigo may be milder but is associated with other brainstem or cerebellar signs, does not suppress with fixation, and may have vertical nystagmus; inability to stand or walk (truncal ataxia) without limb ataxia is a red flag for a central lesion. The HINTS examination (Head Impulse, Nystagmus, Test of Skew) performed at the bedside can reliably distinguish peripheral from central vertigo in patients with acute vestibular syndrome.

Fill in the blank: The HINTS examination distinguishes _____ from central vertigo at the bedside. _____ nystagmus that does not suppress with fixation is a red flag for central vertigo.

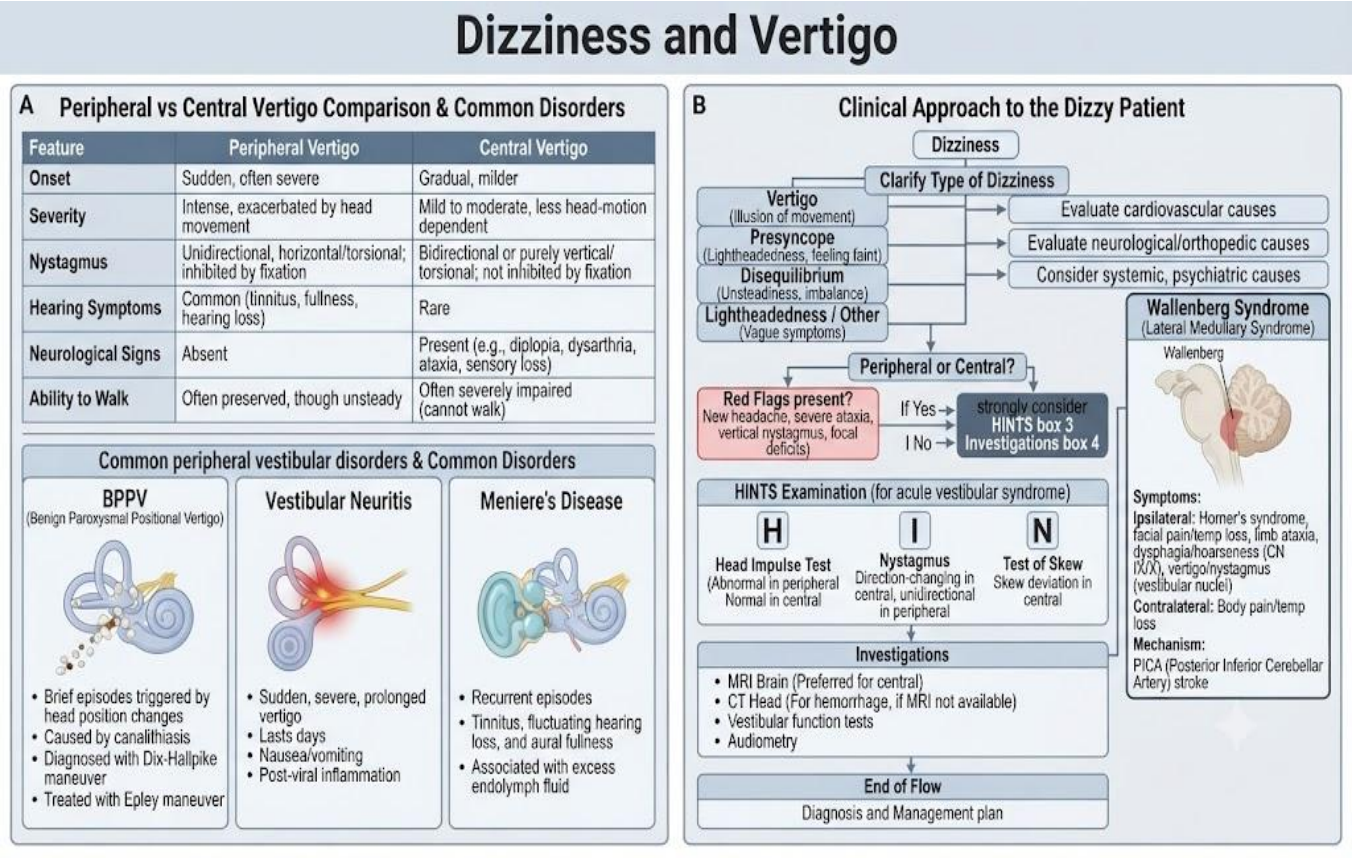


Figure 4.3: Peripheral versus central vertigo, the three main peripheral disorders, and clinical approach to the dizzy patient

Pilot questions 4.3

1. Describe the clinical features of BPPV and describe its diagnosis and treatment. (Linked to SLO 4.3)
2. Describe the triad of Meniere's disease and state the treatment. (Linked to SLO 4.3)
3. State five features that suggest central rather than peripheral vertigo. (Linked to SLO 4.3)
4. Describe the clinical features of Wallenberg syndrome. (Linked to SLO 4.3)
5. Describe the HINTS examination and explain how it distinguishes peripheral from central vertigo. (Linked to SLO 4.3)

4.4 Weakness and Sensory Disturbance

4.4.1 Approach to weakness

The approach to a patient presenting with weakness follows the localisation principles established in Series 1: first determine the pattern of weakness (hemiplegia: cortex or internal capsule or brainstem; monoplegia: cortical or nerve root; paraplegia: spinal cord; proximal: myopathy or inflammatory muscle disease; distal: peripheral neuropathy), then determine whether it is upper or lower motor neuron (UMN or LMN) in character (Series 1, Part 1.4), and finally consider the time course (acute: stroke, Guillain-Barre syndrome; subacute: motor neuron disease, MS; chronic and progressive: hereditary neuropathies, muscular dystrophy).

Acute onset of hemiplegia (face, arm, and leg on the same side) with or without speech difficulty, visual field defect, or sensory loss points strongly to a stroke and mandates urgent CT brain. Bilateral leg weakness (paraparesis or paraplegia) with a sensory level and bladder disturbance indicates spinal cord compression until proven otherwise and requires urgent MRI of the spine. Ascending weakness with areflexia following an infection suggests Guillain-Barre syndrome, a potentially life-threatening peripheral neuropathy requiring urgent nerve conduction studies and assessment of respiratory function.

Fill in the blank: Acute hemiplegia mandates urgent _____ brain to exclude stroke. Bilateral leg weakness with a sensory level and bladder disturbance indicates _____ cord compression until proven otherwise.

4.4.2 Peripheral neuropathy

Peripheral neuropathy refers to disease of the peripheral nerves and can be classified by the pattern of involvement (mononeuropathy: single nerve; mononeuritis multiplex: multiple individual nerves; polyneuropathy: symmetrical and diffuse), the nerve fibres predominantly affected (motor, sensory, autonomic, or mixed), and the underlying pathology (axonal: reduced amplitude on NCS; demyelinating: slowed conduction velocity). Common causes in Nigeria

include diabetes mellitus (the most common cause of polyneuropathy globally: distal symmetrical, predominantly sensory, axonal), HIV and antiretroviral drugs (particularly stavudine), leprosy (Mycobacterium leprae: the most common cause of peripheral neuropathy worldwide: thickened cutaneous nerves, skin patches, and mononeuritis multiplex), vitamin B12 deficiency, alcohol, and isoniazid (treated with pyridoxine).

Guillain-Barre syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy, typically triggered by a preceding infection (Campylobacter jejuni most commonly). It presents with ascending symmetrical weakness and areflexia, beginning in the legs and rising to involve the arms, respiratory muscles, and cranial nerves (facial diplegia in 50 percent). Autonomic involvement (tachycardia, blood pressure fluctuations) is common and may be life-threatening. CSF shows the characteristic albuminocytological dissociation (elevated protein with normal cell count). Treatment: IV immunoglobulin or plasma exchange (equally effective); supportive care including respiratory monitoring and ventilatory support when the FVC falls below 15 to 20 mL/kg.

Fill in the blank: The most common cause of peripheral neuropathy in Nigeria is _____ mellitus. Guillain-Barre syndrome shows albuminocytological _____ on CSF analysis (elevated protein with normal cell count).

4.4.3 Approach to sensory disturbance

Sensory symptoms include numbness, tingling (paraesthesiae), pain, and hypersensitivity. The distribution of sensory loss is the key to localisation: a dermatomal pattern (following a spinal root map) indicates a nerve root lesion or radiculopathy; a peripheral nerve territory pattern indicates a mononeuropathy; a distal symmetrical glove-and-stocking pattern indicates a polyneuropathy; hemibody sensory loss (face and limbs on one side) indicates a thalamic or cortical lesion; and a sensory level (all modalities lost below a specific dermatome bilaterally) indicates a spinal cord lesion.

Dissociated sensory loss (loss of one sensory modality with preservation of others in the same region) is particularly useful for localisation. Loss of pain and temperature with preserved fine touch and vibration in a region indicates spinothalamic tract damage on the contralateral side (for example in Brown-Sequard syndrome, syringomyelia, or the lateral medullary syndrome). Loss of fine touch and vibration with preserved pain and temperature indicates dorsal column damage. Painful sensory symptoms (burning pain, allodynia, hyperpathia) in a glove-and-stocking distribution suggest a painful peripheral neuropathy (diabetic, alcoholic, HIV-related, or drug-induced).

Fill in the blank: A dermatomal distribution of sensory loss indicates a nerve _____ lesion. Loss of pain and temperature with preserved fine touch indicates damage to the _____ tract.

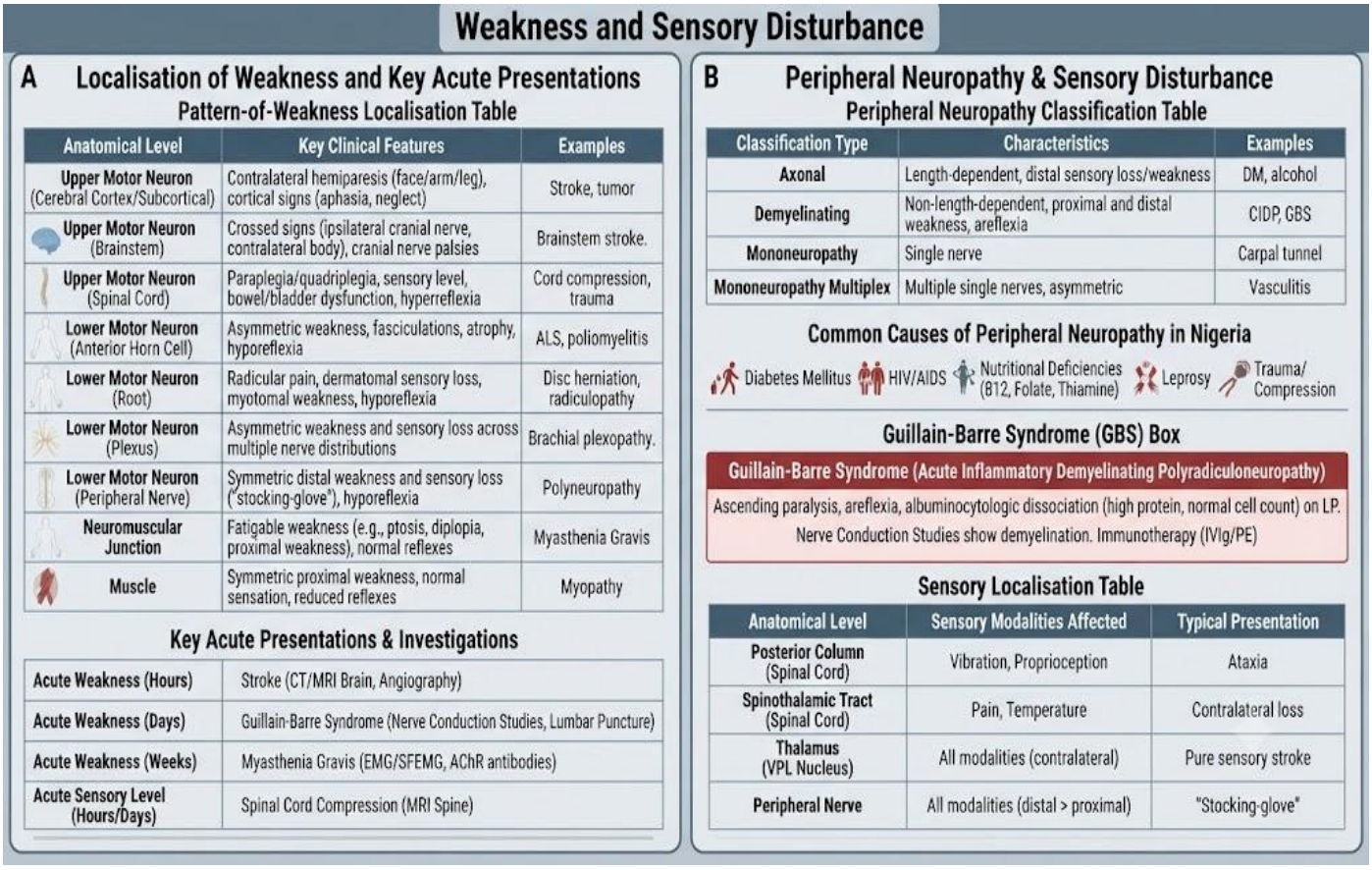


Figure 4.4: Approach to weakness and sensory disturbance, peripheral neuropathy causes, and sensory localisation

Pilot questions 4.4

1. Describe the pattern of weakness that suggests a spinal cord lesion and state the urgent investigation. (Linked to SLO 4.4)
2. State five causes of peripheral neuropathy relevant to Nigerian practice. (Linked to SLO 4.4)
3. Describe the clinical features and CSF findings in Guillain-Barre syndrome. (Linked to SLO 4.4)
4. Describe the treatment of Guillain-Barre syndrome and the respiratory threshold for ventilation. (Linked to SLO 4.4)
5. Describe the sensory localisation pattern of a polyneuropathy and a spinal cord lesion. (Linked to SLO 4.4)

Series Summary

This Series examined the common neurological symptoms encountered in clinical practice. We explored headache (distinguishing thunderclap onset and red flag features from primary disorders: migraine, tension-type, and cluster headache), seizures and epilepsy (focal versus generalised classification, Nigerian causes of symptomatic epilepsy, antiepileptic drug selection, and management of status epilepticus), dizziness and vertigo (peripheral causes: BPPV, vestibular neuritis, and Meniere's disease; central causes: posterior fossa stroke and Wallenberg syndrome; and the HINTS examination), and finally weakness and sensory disturbance (pattern localisation, peripheral neuropathy, Guillain-Barre syndrome, and sensory dissociation).

Having completed this Series, you should now be able to take a structured headache history and identify red flags (SLO 4.1), classify seizures and outline epilepsy management (SLO 4.2), distinguish peripheral from central vertigo and apply the HINTS examination (SLO 4.3), and use the pattern of weakness and sensory loss to localise neurological lesions (SLO 4.4). These skills provide the clinical foundation for the disease-specific content in Series 5.

Glossary of Terms

- **Absence seizure:** a generalised seizure characterised by brief (5 to 30 seconds) episodes of staring and unresponsiveness without a post-ictal phase; associated with a 3 Hz spike-and-wave discharge on EEG.
- **Albuminocytological dissociation:** a CSF finding of elevated protein with a normal white cell count; characteristic of Guillain-Barre syndrome and some other demyelinating neuropathies.
- **BPPV (benign paroxysmal positional vertigo):** the most common cause of vertigo; caused by displaced otoconia in the semicircular canals; presents with brief positional vertigo; diagnosed by the Dix-Hallpike manoeuvre and treated by the Epley manoeuvre.
- **Epley manoeuvre:** a repositioning procedure for BPPV consisting of a series of head movements designed to move displaced otoconia out of the affected semicircular canal.
- **Guillain-Barre syndrome (GBS):** an acute inflammatory demyelinating polyradiculoneuropathy, typically triggered by a preceding infection; presents with ascending symmetrical weakness and areflexia; treated with IV immunoglobulin or plasma exchange.
- **HINTS examination:** a bedside clinical test (Head Impulse, Nystagmus, Test of Skew) used to distinguish peripheral from central acute vestibular syndrome; a normal head

impulse test with direction-changing nystagmus or vertical skew deviation indicates a central lesion.

- **Meniere's disease:** a disorder of the inner ear causing episodic vertigo (20 minutes to 24 hours), fluctuating sensorineural hearing loss, and tinnitus from endolymphatic hydrops.
- **Migraine:** a primary headache disorder characterised by recurrent attacks of moderate to severe unilateral throbbing headache lasting 4 to 72 hours with nausea, photophobia, and phonophobia; may be preceded by an aura.
- **Neurocysticercosis:** infection of the brain with larvae of Taenia solium (the pork tapeworm); a leading cause of new-onset seizures in adults in Nigeria and other endemic regions.
- **Status epilepticus:** a neurological emergency defined as a seizure lasting more than 5 minutes or two seizures without full recovery between them; managed with IV lorazepam followed by IV phenytoin or valproate.
- **Thunderclap headache:** a headache reaching maximal intensity within seconds; always a neurological emergency requiring urgent CT brain and lumbar puncture to exclude subarachnoid haemorrhage.
- **Wallenberg syndrome:** the lateral medullary syndrome caused by posterior inferior cerebellar artery occlusion; produces ipsilateral facial sensory loss and Horner's syndrome with contralateral limb pain and temperature loss, vertigo, dysphagia, and cerebellar ataxia.

Concept Check Questions [Series 4]

Objective questions

1. A headache reaching maximal intensity within seconds is called a _____ headache and requires urgent CT brain to exclude subarachnoid haemorrhage. (Linked to SLO 4.1)
2. Absence seizures show a characteristic _____ Hz spike-and-wave discharge on EEG and do not have a post-ictal period. (Linked to SLO 4.2)
3. BPPV is diagnosed by the _____ manoeuvre and treated by the Epley manoeuvre. (Linked to SLO 4.3)
4. Guillain-Barre syndrome shows _____ dissociation on CSF analysis: elevated protein with a normal cell count. (Linked to SLO 4.4)

5. The most common cause of peripheral neuropathy in Nigerian practice is _____ mellitus. (Linked to SLO 4.4)

Short-answer questions

6. State four red flag features in headache that mandate urgent investigation. (Linked to SLO 4.1)
7. Distinguish migraine from tension-type headache in terms of character, severity, and associated features. (Linked to SLO 4.1)
8. Define status epilepticus and describe its immediate management. (Linked to SLO 4.2)
9. State five features that suggest central rather than peripheral vertigo. (Linked to SLO 4.3)
10. Describe the sensory distribution that indicates a polyneuropathy and explain why it has this pattern. (Linked to SLO 4.4)

Long-answer questions

11. Describe the clinical features and management of migraine. (Linked to SLO 4.1)
12. Describe the classification of seizures and the antiepileptic drugs used for each type. (Linked to SLO 4.2)
13. Describe the clinical features of Guillain-Barre syndrome and outline its management. (Linked to SLO 4.4)
14. Describe the clinical features of Wallenberg syndrome and state the artery involved. (Linked to SLO 4.3)
15. Describe the approach to a patient presenting with acute onset of bilateral leg weakness and bladder disturbance. (Linked to SLO 4.4)

Answers to Concept Check Questions [Series 4]

Objective questions

1. Thunderclap.

2. 3.
3. Dix-Hallpike.
4. Albuminocytological.
5. Diabetes.

Short-answer questions

6. Thunderclap onset (SAH until proven otherwise), headache with fever and neck stiffness (meningitis), new headache with focal neurological signs, new headache in a patient over 50 years.
7. Migraine: unilateral, throbbing, moderate to severe, with nausea and photophobia and phonophobia, lasting 4 to 72 hours, worsened by activity, may have aura. Tension-type: bilateral, pressing or band-like, mild to moderate, without significant nausea or photophobia or phonophobia, not worsened by activity.
8. Status epilepticus: a seizure lasting more than 5 minutes or two seizures without full recovery between them. Immediate management: IV lorazepam (0.1 mg/kg) as first-line; if seizure continues, IV phenytoin (18 mg/kg) or IV valproate; if still refractory, general anaesthesia with propofol, midazolam, or thiopentone in an ICU setting.
9. Vertical nystagmus or direction-changing nystagmus not suppressed by visual fixation; other brainstem or cerebellar signs (diplopia, dysarthria, dysphagia, facial numbness, Horner's syndrome, limb ataxia); truncal ataxia with inability to stand or walk without limb ataxia; risk factors for stroke; failure to improve over days.
10. A polyneuropathy affects all peripheral nerves diffusely; the longest fibres (those supplying the feet) are most vulnerable because they have the greatest distance for axoplasmic transport and the highest metabolic demands, so they are affected first. Deficits begin at the feet and spread proximally, producing a symmetrical glove-and-stocking distribution of sensory loss and weakness.

Long-answer questions

11. **Basic response:** Migraine presents with recurrent attacks of moderate to severe, unilateral, throbbing headache lasting 4 to 72 hours, accompanied by nausea and vomiting, photophobia, and phonophobia, worsened by physical activity. One-third of patients experience an aura (most commonly visual: scintillating scotoma or zigzag

lines). Acute treatment: simple analgesics (paracetamol, NSAIDs) for mild to moderate attacks; triptans (sumatriptan: 5-HT1B/1D agonists, which cause vasoconstriction and inhibit trigeminovascular inflammation) for moderate to severe attacks. Prophylaxis (for attacks occurring more than four per month): propranolol, amitriptyline, topiramate, or sodium valproate (teratogenic in women of childbearing age); lifestyle: identify and avoid triggers, maintain regular sleep and meal patterns.

Value-added response: Triptans are contraindicated in patients with ischaemic heart disease or a history of stroke, because their vasoconstrictive mechanism of action may precipitate coronary or cerebral ischaemia. In Nigeria, the cost and availability of triptans can be a barrier, and simple analgesics combined with antiemetics (metoclopramide) remain a practical acute treatment option.

12. **Basic response:** Seizure classification: Focal (begin in one hemisphere: focal aware: consciousness preserved; focal impaired awareness: consciousness impaired with automatisms) and Generalised (begin simultaneously in both hemispheres: absence: 3 Hz spike-and-wave, no post-ictal period; myoclonic: brief jerks; tonic; atonic: drop attacks; generalised tonic-clonic: tonic then clonic phases with post-ictal confusion). Antiepileptic drugs by type: absence seizures (ethosuximide or sodium valproate); juvenile myoclonic epilepsy (sodium valproate or levetiracetam: avoid carbamazepine as it worsens myoclonus); generalised tonic-clonic (sodium valproate, lamotrigine, levetiracetam); focal seizures (carbamazepine, lamotrigine, levetiracetam: avoid sodium valproate as teratogen in women if possible). Monotherapy is the goal; avoid sodium valproate in women of childbearing age.

Value-added response: The choice between antiepileptic drugs in Nigerian practice must consider cost, availability, and teratogenicity. Phenobarbitone remains widely used as it is cheap and effective for GTC and focal seizures; however its sedative side effects, enzyme-inducing properties, and cognitive effects make it less desirable than modern first-line drugs where available.

13. **Basic response:** GBS presents with ascending symmetrical weakness beginning in the legs and rising to the arms, respiratory muscles, and cranial nerves; it is typically triggered by a preceding infection (Campylobacter jejuni most commonly, also CMV, EBV, and COVID-19). All tendon reflexes are absent (areflexia). Facial diplegia (bilateral VII nerve palsy) occurs in 50 percent. Autonomic dysfunction (tachycardia, blood pressure swings) is common and life-threatening. CSF shows albuminocytological dissociation (elevated protein, normal cell count). NCS shows demyelinating pattern (markedly slowed conduction velocity). Management: IV immunoglobulin (2 g/kg over 5 days) or plasma exchange (equally effective); neither combination of both; monitor FVC (forced vital capacity) every 4 to 6 hours and ventilate if FVC falls below 15 to 20

mL/kg. Supportive care: analgesia (neuropathic pain is common and severe), DVT prophylaxis, physiotherapy, and early rehabilitation.

Value-added response: The need for ventilatory support in GBS should be anticipated early rather than waiting for respiratory failure: the 20-15-12 rule is a useful aide-memoire for respiratory parameters requiring consideration of intubation (FVC below 20 mL/kg, maximal inspiratory pressure below 30 cmH2O, maximal expiratory pressure below 40 cmH2O: any two of these three warrants ICU admission).

14. **Basic response:** Wallenberg syndrome (lateral medullary syndrome) is caused by occlusion of the posterior inferior cerebellar artery (PICA), which supplies the lateral medulla and inferior cerebellum. The clinical features reflect the structures damaged in the lateral medulla: ipsilateral facial pain and temperature loss (descending trigeminal nucleus and tract), ipsilateral Horner's syndrome (descending hypothalamospinal sympathetic fibres: ptosis, miosis, and anhidrosis), ipsilateral cerebellar ataxia (inferior cerebellar peduncle), ipsilateral palatal and pharyngeal weakness (nucleus ambiguus: CN IX and X: causing hoarseness and dysphagia), and contralateral limb pain and temperature loss (spinothalamic tract). The patient also has vertigo, nausea, and vomiting from vestibular nucleus involvement. Importantly, limb weakness is absent (the corticospinal tract is located medially in the medulla and is not affected by a lateral medullary infarct).

Value-added response: The absence of hemiplegia in Wallenberg syndrome despite severe neurological deficits is a key distinguishing feature: if the patient has vertigo with crossed sensory deficits (ipsilateral face and contralateral limb) but no limb weakness, a lateral medullary infarct is the most likely diagnosis. MRI DWI confirms the infarct within minutes and is far superior to CT in the posterior fossa.

15. **Basic response:** Bilateral leg weakness with a sensory level and bladder disturbance (retention or incontinence) indicates spinal cord compression until proven otherwise. Initial management: ABCDE stabilisation; immediate MRI of the whole spine with gadolinium (the investigation of choice: identifies the level, extent, and cause of cord compression); while awaiting MRI, do not move the patient unnecessarily (cervical spine injury precautions if trauma is suspected). Causes: metastatic spinal cord compression (from breast, prostate, lung cancer: the most common cause in adults), disc prolapse, epidural abscess, haematoma, vertebral tuberculosis (Pott's disease: common in Nigeria), and multiple sclerosis. If malignant cord compression is suspected, give IV dexamethasone immediately (reduces oedema and buys time). Urgent neurosurgical or radiation oncology referral for definitive decompression or radiotherapy within 24 hours of onset is essential to maximise neurological recovery.

Value-added response: The outcome of spinal cord compression is strongly time-dependent: patients who are still ambulatory at the time of treatment have a significantly better chance of remaining ambulatory than those who are paraplegic before treatment begins. Recognising the red flag combination of bilateral leg weakness, sensory level, and bladder symptoms and treating it as an emergency is one of the most important neurological skills in Nigerian practice.

Answers to Pilot Questions [Series 4]

Part 4.1

1. Thunderclap onset (SAH until proven otherwise), headache with fever and neck stiffness (meningitis), headache with focal neurological signs (stroke or mass lesion), new headache in a patient over 50 years (temporal arteritis or intracranial mass).
2. Migraine: unilateral, throbbing, moderate to severe, 4 to 72 hours, nausea and photophobia and phonophobia, worsened by activity, may have aura (visual most common). Tension-type: bilateral, pressing or band-like, mild to moderate, not worsened by activity, no significant nausea or photophobia.
3. Cluster headache: strictly unilateral orbital or periorbital pain rated 10 out of 10, lasting 15 to 180 minutes, occurring in clusters over weeks followed by remission, with ipsilateral autonomic features (lacrimation, nasal congestion, miosis, ptosis, facial flushing). Acute treatment: 100 percent oxygen at 10 to 15 L/min for 15 minutes via non-rebreather mask (most effective), and subcutaneous or nasal sumatriptan.
4. Urgent CT brain without contrast (sensitivity above 98 percent within 6 hours of ictus: blood appears hyperdense in the subarachnoid space). If CT is negative and clinical suspicion remains high, lumbar puncture at least 12 hours after symptom onset to detect xanthochromia (yellow CSF from bilirubin: persists for up to 2 weeks). If confirmed SAH, urgent CT angiography or digital subtraction angiography to identify the aneurysm and urgent neurosurgical or interventional radiology referral.
5. Temporal arteritis (giant cell arteritis) is an inflammatory vasculitis affecting medium and large vessels, particularly the temporal and ophthalmic arteries. It causes severe visual loss from anterior ischaemic optic neuropathy (occlusion of the posterior ciliary arteries supplying the optic disc); once visual loss occurs in one eye, the other eye is at immediate risk. High-dose prednisolone (1 mg/kg/day) must be started immediately on clinical suspicion, before biopsy confirmation, because biopsy can be performed within 7 to 10 days and will still be positive despite steroid treatment.

Part 4.2

1. Focal seizures begin in one hemisphere and produce symptoms referable to the cortical region of onset (for example a motor focal seizure from the motor cortex produces unilateral jerking of the contralateral limbs). Generalised seizures begin simultaneously across both hemispheres and always involve impairment of consciousness from onset. The post-ictal period (confusion, drowsiness, headache, and muscle aching lasting minutes to hours after a GTC seizure) is an important feature that distinguishes a generalised seizure from a syncopal episode, which has immediate recovery of full consciousness.

2. Childhood absence epilepsy: age of onset 4 to 10 years; brief (5 to 30 seconds) episodes of staring and unresponsiveness with sudden onset and offset and no post-ictal confusion; consciousness is transiently interrupted and the child resumes their activity as if nothing happened. EEG: characteristic 3 Hz (three per second) generalised spike-and-slow wave discharges, often provoked by hyperventilation during the recording. Treatment: ethosuximide (first-line) or sodium valproate; the condition usually remits in adolescence.

3. Neurocysticercosis (Taenia solium larvae in the brain: leading cause of new-onset seizures in adults in Nigeria and sub-Saharan Africa), cerebral malaria (acute symptomatic seizures from P. falciparum), tuberculous meningitis with cortical involvement, stroke (the most common cause of epilepsy in the elderly in Nigeria), and traumatic brain injury.

4. Absence seizures: ethosuximide (first-line) or sodium valproate; avoid carbamazepine (worsens absence). Juvenile myoclonic epilepsy: sodium valproate or levetiracetam; avoid carbamazepine (worsens myoclonus). Generalised tonic-clonic: sodium valproate, lamotrigine, or levetiracetam. Focal seizures: carbamazepine or lamotrigine or levetiracetam; avoid sodium valproate as teratogen if monotherapy alternatives exist for women of childbearing age.

5. Status epilepticus: a seizure lasting more than 5 minutes, or two or more seizures without return to baseline between them. Immediate management: IV lorazepam 0.1 mg/kg (or diazepam 0.15 mg/kg IV or rectally if no IV access); if the seizure continues after 5 minutes, IV phenytoin 18 mg/kg (at no more than 50 mg/min) or IV sodium valproate 25 to 30 mg/kg; if refractory after 20 to 30 minutes, general anaesthesia with propofol, midazolam, or thiopentone in an ICU. Always check blood glucose immediately and correct hypoglycaemia.

Part 4.3

1. BPPV: brief (seconds) episodes of intense vertigo triggered by specific changes in head position (rolling over in bed, looking up). Diagnosis: the Dix-Hallpike manoeuvre (the patient is moved from sitting to lying with the head turned 45 degrees to the affected side and extended over the edge of the couch): a positive test reproduces the vertigo with a burst of rotatory nystagmus (upbeating and towards the affected ear) after a latency of 1 to 5 seconds; the nystagmus fatigues with repeated testing. Treatment: the Epley repositioning manoeuvre (a sequence of four head positions rotating the head through 270 degrees to move the otoconia from the posterior semicircular canal into the utricle); it is curative in 80 percent after a single session.

2. Meniere's disease: episodic vertigo lasting 20 minutes to 24 hours (not seconds as in BPPV), fluctuating low-frequency sensorineural hearing loss, tinnitus (a roaring or ringing sound), and a feeling of aural fullness or pressure. Caused by endolymphatic hydrops (excess fluid in the endolymphatic system of the inner ear). Treatment: low-sodium diet (below 1500 mg per day: reduces endolymph production), thiazide diuretics (bendroflumethiazide: reduces endolymph volume), betahistine (increases cochlear blood flow: reduces frequency of attacks), and vestibular sedatives (prochlorperazine) for acute attacks.

3. Vertical nystagmus or direction-changing nystagmus that does not suppress with visual fixation; other brainstem or cerebellar signs (diplopia, dysarthria, dysphagia, facial sensory loss, Horner's syndrome, limb ataxia); truncal ataxia with inability to stand or walk without limb involvement; new onset in a patient with vascular risk factors (hypertension, diabetes, atrial fibrillation, smoking); failure of symptoms to improve over days.

4. Wallenberg syndrome (lateral medullary syndrome) from PICA occlusion: ipsilateral facial pain and temperature loss (trigeminal nucleus), ipsilateral Horner's syndrome (descending sympathetic fibres: ptosis, miosis, anhidrosis), ipsilateral cerebellar ataxia (inferior cerebellar peduncle), ipsilateral palatal and vocal cord paralysis (nucleus ambiguus: hoarseness and dysphagia), contralateral limb pain and temperature loss (spinothalamic tract), and vertigo with nausea and vomiting (vestibular nucleus). Notably absent: limb weakness (the corticospinal tract is in the medial medulla and is spared).

5. HINTS examination for acute vestibular syndrome: Head Impulse test (the examiner rapidly rotates the patient's head to one side while the patient fixates on a target; an abnormal test: a corrective saccade after the head movement: indicates peripheral vestibular disease, as the VOR is impaired; a normal head impulse test in a patient with acute vertigo is a red flag for central disease); Nystagmus (direction-changing nystagmus on lateral gaze or vertical nystagmus: central; unidirectional horizontal-rotational nystagmus: peripheral); Test of Skew (vertical misalignment of the eyes: skew deviation:

central). A normal head impulse with nystagmus or skew deviation indicates a central lesion; this combination has higher sensitivity for posterior fossa stroke than early MRI.

Part 4.4

1. Bilateral leg weakness (paraparesis) with a sensory level (a clear dermatomal boundary below which all sensation is impaired bilaterally) and bladder disturbance (retention or incontinence) is the classic presentation of spinal cord compression. Urgent investigation: MRI of the whole spine with gadolinium (the investigation of choice: identifies the level, extent, and cause of compression; CT spine without MRI misses soft tissue causes). While awaiting MRI, IV dexamethasone is given if malignant cord compression is suspected, and the patient is kept flat with cervical spine precautions if trauma is a possibility.
2. Diabetes mellitus (most common cause in Nigeria: distal symmetrical sensory axonal polyneuropathy), HIV infection and antiretroviral drugs especially stavudine (sensory neuropathy), leprosy (Mycobacterium leprae: mononeuritis multiplex with thickened cutaneous nerves and skin patches), vitamin B12 deficiency (subacute combined degeneration: dorsal column and spinothalamic involvement), and isoniazid toxicity (pyridoxine deficiency: prevents with pyridoxine supplementation).
3. GBS presents with ascending symmetrical weakness starting in the legs (difficulty walking, then climbing stairs, then rising from a chair) and rising over days to weeks to involve the arms, respiratory muscles, and cranial nerves (facial diplegia in 50 percent, ophthalmoplegia, dysphagia). All tendon reflexes are absent from the outset. Autonomic instability (tachycardia, hypertension alternating with hypotension, urinary retention) is common. CSF: albuminocytological dissociation (elevated protein, typically above 0.45 g/L, with a normal white cell count of fewer than 10 per microlitre). NCS: demyelinating pattern with markedly slowed conduction velocities and prolonged distal latencies.
4. IV immunoglobulin (2 g/kg over 5 days: the most practical in Nigerian settings) or plasma exchange (five exchanges over 10 to 14 days) are equally effective and should be started as soon as the diagnosis is confirmed; the combination of both is not more effective than either alone. Respiratory monitoring is critical: measure FVC (forced vital capacity) every 4 to 6 hours; intubate and ventilate when FVC falls below 15 to 20 mL/kg (approximately one litre in an average adult), maximal inspiratory pressure below 30 cmH2O, or maximal expiratory pressure below 40 cmH2O. Supportive care: analgesia for severe neuropathic pain (gabapentin, amitriptyline), DVT prophylaxis (heparin and compression stockings), physiotherapy and early rehabilitation.

5. Polyneuropathy: distal symmetrical glove-and-stocking pattern of sensory loss (affects feet first because the longest fibres are most vulnerable; spreads proximally as the disease progresses); loss of all sensory modalities (or predominantly large fibre: fine touch and vibration and proprioception; or small fibre: pain and temperature depending on the neuropathy type). Spinal cord lesion: a sensory level (all modalities bilaterally impaired below a specific dermatomal level); may show a dissociated pattern (Brown-Sequard: ipsilateral dorsal column loss with contralateral spinothalamic loss; or central cord: suspended dissociated sensory level from syringomyelia).

References and Attributions [Series 4]

Textual References

- Fuller, G. and Manford, M. (2010). Neurology: An Illustrated Colour Text (4th ed.). Edinburgh: Churchill Livingstone.
- Headache Classification Committee of the International Headache Society. (2018). The International Classification of Headache Disorders (3rd ed.). Cephalalgia, 38(1), 1 to 211.
- Fisher, R. S., Cross, J. H., French, J. A. et al. (2017). Operational Classification of Seizure Types by the International League Against Epilepsy. Epilepsia, 58(4), 522 to 530.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.

Figures, Plates, Tables, and Boxes

- Figure 4.1: Structured approach to headache. AI-generated using the prompt: "Two-panel diagram: structured headache history flowchart with red flags box; primary headache comparison boxes (migraine, tension-type, cluster) and secondary headache boxes (SAH, meningitis, raised ICP, temporal arteritis)." Suggested OER search string: "headache classification primary secondary migraine cluster tension subarachnoid haemorrhage diagram OER."
- Figure 4.2: Seizure classification, epilepsy syndromes, and management. AI-generated using the prompt: "Two-panel diagram: hierarchical seizure classification tree (focal versus generalised with subtypes); epilepsy syndrome table, antiepileptic drug table,

safety counselling box, and status epilepticus management box." Suggested OER search string: "seizure classification epilepsy syndromes antiepileptic drugs diagram OER."

- Figure 4.3: Peripheral versus central vertigo and clinical approach. AI-generated using the prompt: "Two-panel diagram: peripheral versus central vertigo comparison table; three peripheral disorder boxes (BPPV, vestibular neuritis, Meniere's); clinical approach flowchart with HINTS examination and Wallenberg syndrome box." Suggested OER search string: "peripheral central vertigo comparison BPPV Meniere's Wallenberg HINTS examination diagram OER."
- Figure 4.4: Weakness and sensory disturbance. AI-generated using the prompt: "Two-panel diagram: pattern-of-weakness localisation table and acute presentations; peripheral neuropathy classification, Nigerian causes, Guillain-Barre syndrome box, and sensory localisation table." Suggested OER search string: "weakness sensory disturbance peripheral neuropathy Guillain-Barre localisation diagram OER."