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MED 604

NEUROLOGY II

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Course title: Neurology II

Course credit load: 2 Units

Series 1: Higher Cerebral Function, Dementia, and Stroke

- **Part 1.1: Higher Cerebral Function**
    - Sub Part 1.1.1: Assessment of higher cerebral function
    - Sub Part 1.1.2: Aphasia and language disorders
    - Sub Part 1.1.3: Agnosia, apraxia, and other cortical syndromes
- **Part 1.2: Dementia**
    - Sub Part 1.2.1: Classification and approach to dementia
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- **Part 1.3: Stroke: Pathophysiology and Assessment**
    - Sub Part 1.3.1: Classification of stroke
    - Sub Part 1.3.2: Clinical presentation and localisation of stroke
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    - Sub Part 1.4.1: Acute management of ischaemic stroke
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    - Sub Part 1.4.3: Secondary prevention and rehabilitation after stroke

Introduction

This opening Series of **Neurology II** begins where cognition itself can be examined directly, working through the assessment of higher cerebral function, the major dementia syndromes that disrupt it over time, and stroke, the sudden vascular insult that can disrupt it in an instant. Each of these three areas demands the same careful, structured localisation skill that underpins the whole of clinical neurology, working out precisely where in the nervous system a lesion sits from the pattern of deficit it produces.

The aim of this Series is to equip you with the ability to assess higher cerebral function and recognise aphasia and other cortical syndromes, classify and describe the major dementia syndromes, classify stroke and describe its clinical presentation, and describe the acute assessment, management, and secondary prevention of stroke. This foundation in cognitive assessment and cerebrovascular disease anchors much of what follows across the rest of this course.

This Series opens with **higher cerebral function**, moves through **dementia** and **stroke: pathophysiology and assessment**, and closes with **stroke: management and prevention**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** describe the assessment of higher cerebral function
- **SLO 1.2:** describe aphasia and other cortical syndromes including agnosia and apraxia
- **SLO 1.3:** classify dementia and describe the general approach to its assessment
- **SLO 1.4:** describe Alzheimer's disease, vascular dementia, and other dementias
- **SLO 1.5:** classify stroke and describe its clinical presentation and localisation
- **SLO 1.6:** describe the acute assessment and investigation of stroke
- **SLO 1.7:** describe the acute management of ischaemic and haemorrhagic stroke
- **SLO 1.8:** discuss secondary prevention and rehabilitation after stroke

1.1 Higher Cerebral Function

1.1.1 assessment of higher cerebral function

A structured bedside evaluation of cognition

Assessment of higher cerebral function systematically examines orientation, attention, memory, language, visuospatial function, and executive function, typically using a structured, validated screening tool alongside targeted bedside testing directed by the specific clinical concern, since a brief global screening test alone can miss a focal cognitive deficit affecting only one specific domain while sparing others entirely.

Interpreting cognitive assessment findings requires accounting for the patient's baseline education level, language, and cultural background, since a low score on a standardised test can reflect these factors rather than genuine cognitive impairment, and comparing performance against an estimated premorbid baseline, where available, considerably strengthens the confidence with which a genuine cognitive decline can be identified.

Fill in the blank: A brief global screening test alone can miss a focal cognitive deficit affecting only one specific _____.

1.1.2 aphasia and language disorders

Disorders of language, distinct from disorders of speech

Aphasia, an acquired disorder of language rather than of speech articulation itself, is classified according to the pattern of fluency, comprehension, and repetition affected: **Broca's aphasia**, non-fluent speech with relatively preserved comprehension, localises to the posterior inferior frontal lobe, while **Wernicke's aphasia**, fluent but often meaningless speech with impaired comprehension, localises to the posterior superior temporal lobe.

Conduction aphasia, fluent speech and relatively preserved comprehension but disproportionately impaired repetition, reflects damage to the arcuate fasciculus connecting Broca's and Wernicke's areas, and correctly classifying the specific aphasia subtype present provides genuinely valuable localising information about the underlying lesion before any imaging is even obtained, a skill this sub-Part aims to build through systematic bedside assessment of these three language components.

Fill in the blank: Conduction aphasia reflects damage to the arcuate fasciculus connecting Broca's and Wernicke's _____.

1.1.3 agnosia, apraxia, and other cortical syndromes

Failures of recognition and skilled movement, not of the senses themselves

Agnosia, an inability to recognise objects, faces, or sounds despite intact primary sensory function, and **apraxia**, an inability to perform learned, skilled motor tasks despite intact strength and coordination, both reflect disruption of higher-order cortical processing rather than a primary sensory or motor deficit, a distinction genuinely important to establish clearly through careful examination before attributing either finding to a more basic sensory or motor problem.

Neglect, most commonly affecting the left side of space following a right parietal lesion, describes a failure to attend to or respond to stimuli on the affected side despite intact sensory pathways, and, alongside agnosia and apraxia, forms part of a broader group of cortical syndromes that localise reliably to specific, recognisable brain regions, reinforcing the genuinely powerful localising value of careful higher cerebral function assessment.

Fill in the blank: Neglect most commonly affects the left side of space following a right _____ lesion.

Figure 1.1: A clinician assessing language function through object naming during bedside examination.

Pilot questions 1.1

1. List the domains assessed in higher cerebral function testing. (Linked to SLO 1.1)
2. Explain why baseline education and language must be considered when interpreting cognitive testing. (Linked to SLO 1.1)
3. Distinguish Broca's from Wernicke's aphasia. (Linked to SLO 1.2)
4. Describe conduction aphasia and its anatomical basis. (Linked to SLO 1.2)
5. Distinguish agnosia from apraxia. (Linked to SLO 1.2)

1.2 Dementia

1.2.1 classification and approach to dementia

A syndrome, not a single disease, with several distinct causes

Dementia, an acquired, progressive decline in cognitive function sufficient to interfere with independent daily functioning, is a syndrome rather than a single disease, with **Alzheimer's disease**, **vascular dementia**, **dementia with Lewy bodies**, and **frontotemporal dementia** together accounting for the great majority of cases, each with a distinct, though sometimes overlapping, clinical and pathological pattern.

A systematic diagnostic approach distinguishes genuine dementia from **delirium**, an acute, fluctuating confusional state with a demonstrable medical cause, and from **depression**, which can produce a pattern of cognitive slowing sometimes termed depressive pseudodementia, since each of these alternative explanations requires an entirely different management approach, making accurate initial differentiation a genuinely essential first step before any specific dementia diagnosis is pursued further.

Fill in the blank: Dementia must be distinguished from delirium, an acute, fluctuating confusional state with a demonstrable medical _____.

1.2.2 alzheimer's disease

The most common cause of dementia

Alzheimer's disease, the most common cause of dementia, characteristically presents with insidious onset and gradual progression, typically beginning with prominent short-term memory impairment before extending to language, visuospatial, and executive difficulties as the disease advances, a pattern reflecting the disease's typical early involvement of the medial temporal lobe structures responsible for new memory formation.

Diagnosis relies primarily on the characteristic clinical history and pattern of cognitive decline, supported by structural imaging to identify a pattern of atrophy consistent with the diagnosis and to exclude an alternative structural cause, and management combines cholinesterase inhibitor medication, which can offer modest symptomatic benefit in appropriately selected patients, alongside environmental support, caregiver education, and planning for anticipated future care needs.

Fill in the blank: Alzheimer's disease typically begins with prominent short-term _____ impairment before extending to other domains.

1.2.3 vascular and other dementias

Different underlying mechanisms, distinct clinical clues

Vascular dementia, resulting from cerebrovascular disease, characteristically presents with a stepwise, rather than smoothly progressive, decline, following discrete vascular events, and often with more prominent executive dysfunction relative to memory impairment compared with Alzheimer's disease, a distinguishing clinical pattern that, alongside vascular risk factors and supporting imaging findings, helps differentiate it from other dementia causes.

Dementia with Lewy bodies presents with fluctuating cognition, recurrent visual hallucinations, and spontaneous parkinsonism, alongside marked sensitivity to antipsychotic medication, while **frontotemporal dementia** presents earlier in life with prominent personality and behavioural change or progressive language impairment, with memory relatively preserved in the early stages, two further distinct patterns worth recognising confidently alongside the more common Alzheimer's and vascular presentations already discussed.

Fill in the blank: Dementia with Lewy bodies shows marked sensitivity to _____ medication.

Table 1.1: Comparing the major dementia subtypes

Dementia type	Typical early feature	Course
Alzheimer's disease	Short-term memory impairment	Gradual, progressive
Vascular dementia	Executive dysfunction	Stepwise, following vascular events
Frontotemporal dementia	Personality or language change	Gradual, earlier onset, memory preserved early

Pilot questions 1.2

1. List the four major dementia subtypes accounting for most cases. (Linked to SLO 1.3)
2. Distinguish dementia from delirium. (Linked to SLO 1.3)
3. Describe the typical early presentation of Alzheimer's disease. (Linked to SLO 1.4)
4. Describe the typical course of vascular dementia. (Linked to SLO 1.4)
5. Describe dementia with Lewy bodies and frontotemporal dementia. (Linked to SLO 1.4)

1.3 Stroke: Pathophysiology and Assessment

1.3.1 classification of stroke

Two fundamentally different mechanisms, one clinical presentation

Stroke, a sudden focal neurological deficit resulting from a vascular cause, is classified as **ischaemic stroke**, resulting from arterial occlusion cutting off blood supply to a specific brain territory, accounting for the great majority of strokes, or **haemorrhagic stroke**, resulting from bleeding into the brain parenchyma, a distinction that cannot be reliably made on clinical grounds alone and requires urgent imaging to confirm, given the genuinely opposite treatment implications each type carries.

Ischaemic stroke is further subclassified by underlying mechanism, including large artery atherosclerosis, cardioembolism from a cardiac source such as atrial fibrillation, and small vessel disease affecting the deep perforating arteries, each carrying distinct implications for secondary prevention strategy, a topic returned to in more detail in the closing Part of this Series once acute management has first been covered.

Fill in the blank: The distinction between ischaemic and haemorrhagic stroke cannot be reliably made on clinical grounds alone and requires urgent _____.

1.3.2 clinical presentation and localisation of stroke

Reading the deficit to localise the vascular territory

Stroke presents with sudden-onset focal neurological deficit, the specific pattern reflecting the vascular territory affected: **middle cerebral artery** territory stroke typically produces contralateral weakness and sensory loss affecting the face and arm more than the leg, alongside aphasia if the dominant hemisphere is affected, while **anterior cerebral artery** territory stroke produces contralateral weakness affecting the leg more than the arm.

Posterior circulation stroke, affecting the vertebrobasilar territory, can produce a genuinely wide range of presentations including vertigo, diplopia, ataxia, and crossed sensorimotor

findings, patterns that are sometimes less immediately recognisable as stroke compared with the more classically taught anterior circulation presentations, making active clinical suspicion genuinely important in any patient presenting with these less typical symptoms and a suggestive onset pattern.

Fill in the blank: Middle cerebral artery territory stroke typically affects the face and arm more than the _____.

1.3.3 acute assessment and investigation of stroke

Confirming the diagnosis and characterising it precisely, fast

Acute stroke assessment prioritises rapid recognition, using a structured screening tool to identify likely stroke at first contact, alongside precise documentation of symptom onset time, a genuinely critical piece of information since it directly determines eligibility for time-sensitive acute treatments covered in the following Part of this Series, making onset time establishment a genuine priority equal to the clinical examination itself.

Urgent non-contrast computed tomography of the head is the standard first-line investigation, primarily to exclude haemorrhage before considering thrombolytic therapy, with further imaging, including vascular imaging to identify a large vessel occlusion potentially amenable to mechanical thrombectomy, obtained where clinically indicated and where local resources and time constraints allow this additional characterisation.

Fill in the blank: Precise documentation of symptom onset time directly determines eligibility for time-sensitive acute _____.

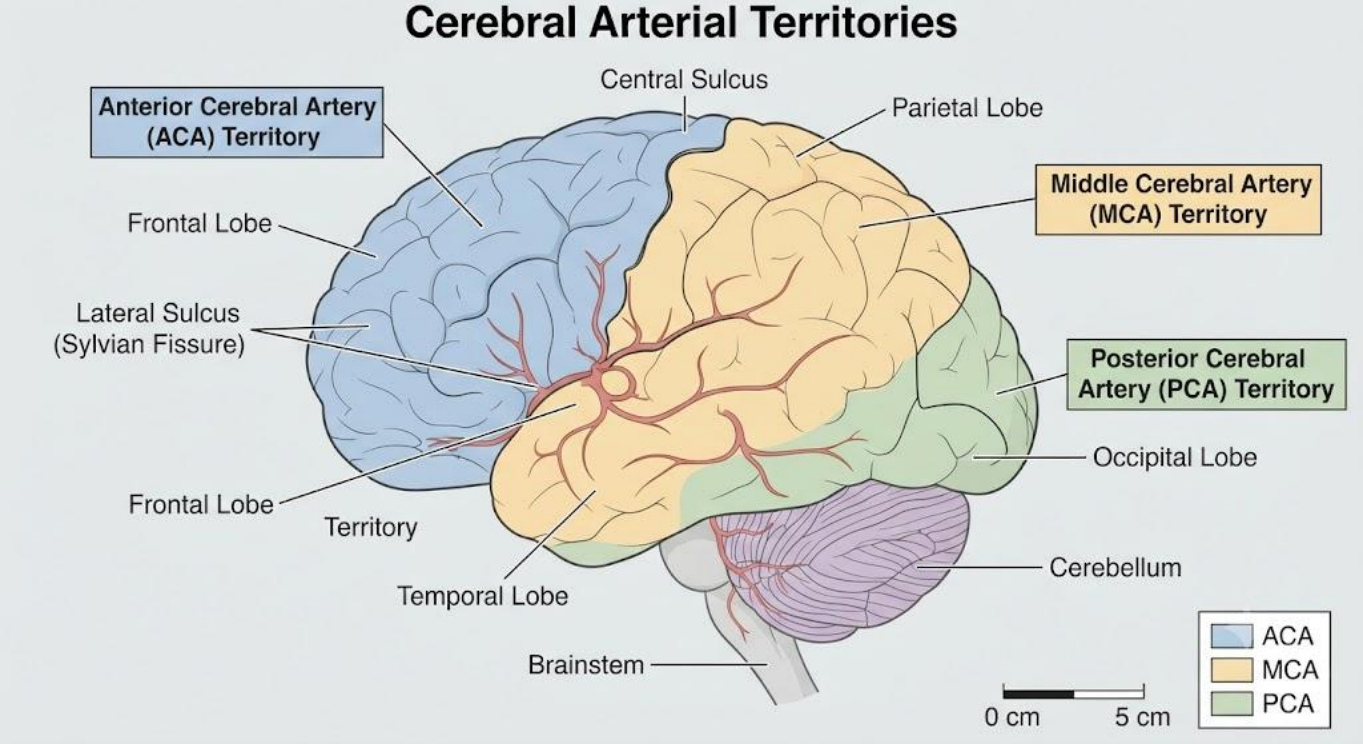

Figure 1.2: A labelled diagram of the major cerebral arterial territories.

Pilot questions 1.3

1. Distinguish ischaemic from haemorrhagic stroke. (Linked to SLO 1.5)
2. List the subclassifications of ischaemic stroke by mechanism. (Linked to SLO 1.5)
3. Describe the typical presentation of middle cerebral artery territory stroke. (Linked to SLO 1.5)
4. Explain why precise onset time documentation is a genuine priority in acute stroke assessment. (Linked to SLO 1.6)
5. Describe the purpose of urgent non-contrast CT in acute stroke assessment. (Linked to SLO 1.6)

1.4 Stroke: Management and Prevention

1.4.1 acute management of ischaemic stroke

Restoring blood flow within a narrow, genuinely critical window

Intravenous thrombolysis, using a clot-dissolving medication, is indicated for appropriately selected patients with ischaemic stroke presenting within a defined, genuinely narrow time window from symptom onset, once haemorrhage has been excluded on imaging, and the benefit

of thrombolysis is genuinely time-dependent, diminishing progressively the longer treatment is delayed, underlining why rapid assessment and treatment decision-making, already emphasised in the previous Part, matters so considerably in acute stroke care.

Mechanical thrombectomy, physically removing a clot from a large occluded vessel using an endovascular catheter-based technique, offers a further treatment option for appropriately selected patients with a confirmed large vessel occlusion, available within a somewhat longer, though still genuinely time-limited, window than thrombolysis, and can be combined with thrombolysis in eligible patients rather than necessarily used as an alternative to it.

Fill in the blank: The benefit of thrombolysis is genuinely time-dependent, diminishing progressively the longer treatment is _____.

1.4.2 management of haemorrhagic stroke

A fundamentally different acute treatment priority

Management of haemorrhagic stroke prioritises careful blood pressure control to reduce the risk of haematoma expansion, reversal of any anticoagulation the patient may be taking, and close monitoring for signs of raised intracranial pressure, since the initial period following haemorrhagic stroke carries a genuine risk of further clinical deterioration from haematoma growth or the development of secondary hydrocephalus, both requiring active, deliberate surveillance.

Surgical evacuation of the haematoma is considered in selected cases, particularly for a cerebellar haemorrhage causing significant mass effect or hydrocephalus, where surgical decompression can be genuinely life-saving, while for many supratentorial haemorrhages, surgery offers less clear-cut benefit, and the decision for surgical intervention requires careful, individualised assessment of haematoma location, size, and the patient's overall clinical trajectory.

Fill in the blank: Surgical evacuation is particularly considered for a cerebellar haemorrhage causing significant mass effect or _____.

1.4.3 secondary prevention and rehabilitation after stroke

Reducing recurrence risk and restoring function

Secondary prevention following stroke addresses the underlying mechanism identified during the acute assessment already discussed earlier in this Series, including antiplatelet or anticoagulant therapy depending on the specific stroke subtype, blood pressure and lipid

management, and addressing modifiable risk factors such as smoking and diabetes, a comprehensive approach genuinely reducing the risk of subsequent stroke.

Stroke rehabilitation, involving physiotherapy, occupational therapy, and speech and language therapy where relevant, ideally begins as early as safely possible following the acute event, and structured, multidisciplinary rehabilitation genuinely improves functional outcome and independence, closing this Series on the same theme of coordinated, multidisciplinary care that has recurred throughout the whole of this course.

Fill in the blank: Structured, multidisciplinary rehabilitation genuinely improves functional outcome and _____.

Table 1.2: Comparing acute management priorities in ischaemic and haemorrhagic stroke

Feature	Ischaemic stroke	Haemorrhagic stroke
Key acute treatment	Thrombolysis or thrombectomy	Blood pressure control, anticoagulation reversal
Time-critical window	Yes, narrow	Ongoing surveillance for expansion
Surgical role	Rarely indicated acutely	Selected cases, especially cerebellar haemorrhage

Pilot questions 1.4

1. Describe intravenous thrombolysis and why timing matters so considerably. (Linked to SLO 1.7)
2. Describe mechanical thrombectomy. (Linked to SLO 1.7)
3. Describe the acute management priorities for haemorrhagic stroke. (Linked to SLO 1.7)
4. Explain when surgical evacuation is particularly considered in haemorrhagic stroke. (Linked to SLO 1.7)
5. Describe the components of secondary prevention and rehabilitation after stroke. (Linked to SLO 1.8)

Series Summary

This opening Series worked through higher cerebral function, dementia, and stroke. Part 1.1 covered the assessment of higher cerebral function, aphasia, and other cortical syndromes, while

Part 1.2 turned to the classification and major subtypes of dementia. Part 1.3 covered the classification, presentation, and acute assessment of stroke, and Part 1.4 closed with the acute management, secondary prevention, and rehabilitation of stroke.

You should now be able to assess higher cerebral function, classify and describe dementia and stroke, and describe their management across all four Parts of this Series, meeting the outcomes set for this Series. Series 2 turns next to epilepsy, sleep disorders, Parkinson's disease, and headache.

Glossary of Terms

- **Agnosia:** an inability to recognise objects, faces, or sounds despite intact primary sensory function.
- **Aphasia:** an acquired disorder of language, distinct from a disorder of speech articulation.
- **Apraxia:** an inability to perform learned, skilled motor tasks despite intact strength and coordination.
- **Broca's aphasia:** non-fluent aphasia with relatively preserved comprehension.
- **Delirium:** an acute, fluctuating confusional state with a demonstrable medical cause.
- **Dementia:** an acquired, progressive decline in cognitive function interfering with daily functioning.
- **Haemorrhagic stroke:** stroke resulting from bleeding into the brain parenchyma.
- **Ischaemic stroke:** stroke resulting from arterial occlusion cutting off blood supply to a brain territory.
- **Mechanical thrombectomy:** endovascular removal of a clot from a large occluded cerebral vessel.
- **Neglect:** a failure to attend to or respond to stimuli on one side of space despite intact sensation.
- **Thrombolysis:** clot-dissolving medication used in appropriately selected acute ischaemic stroke.
- **Wernicke's aphasia:** fluent but often meaningless speech with impaired comprehension.

Concept Check Questions [Series 1]

Objective questions

1. A brief global screening test alone can miss a focal cognitive deficit affecting only one specific _____. (Linked to SLO 1.1)

2. Dementia must be distinguished from delirium, an acute, fluctuating confusional state with a demonstrable medical _____. (Linked to SLO 1.3)
3. The distinction between ischaemic and haemorrhagic stroke cannot be reliably made on clinical grounds alone and requires urgent _____. (Linked to SLO 1.5)
4. The benefit of thrombolysis is genuinely time-dependent, diminishing progressively the longer treatment is _____. (Linked to SLO 1.7)
5. Structured, multidisciplinary rehabilitation genuinely improves functional outcome and _____. (Linked to SLO 1.8)

Short-answer questions

6. Distinguish Broca's from Wernicke's aphasia. (Linked to SLO 1.2)
7. Describe the typical early presentation of Alzheimer's disease. (Linked to SLO 1.4)
8. Describe the typical presentation of middle cerebral artery territory stroke. (Linked to SLO 1.5)
9. Describe mechanical thrombectomy. (Linked to SLO 1.7)
10. Describe the components of secondary prevention after stroke. (Linked to SLO 1.8)

Long-answer questions

11. Discuss the assessment of higher cerebral function and describe aphasia and other cortical syndromes. (Linked to SLO 1.1, 1.2)
12. Describe the classification and major subtypes of dementia. (Linked to SLO 1.3, 1.4)
13. Discuss the classification, presentation, and acute assessment of stroke. (Linked to SLO 1.5, 1.6)
14. Describe the acute management of ischaemic and haemorrhagic stroke. (Linked to SLO 1.7)
15. Discuss secondary prevention and rehabilitation after stroke. (Linked to SLO 1.8)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Domain.
2. Cause.
3. Imaging.
4. Delayed.
5. Independence.

Short-answer questions

6. Broca's aphasia is non-fluent with preserved comprehension, while Wernicke's aphasia is fluent with impaired comprehension.
7. Alzheimer's disease typically begins with prominent short-term memory impairment before extending to other domains.
8. Middle cerebral artery stroke typically causes contralateral face and arm weakness, sensory loss, and aphasia if dominant.
9. Mechanical thrombectomy is endovascular removal of a clot from a large occluded cerebral vessel.
10. Secondary prevention includes antiplatelet or anticoagulant therapy, blood pressure and lipid management, and risk factor modification.

Long-answer questions

11. **Basic response:** Assessment covers orientation, attention, memory, language, visuospatial, and executive function; aphasia is classified by fluency, comprehension, and repetition; agnosia, apraxia, and neglect reflect higher-order cortical dysfunction rather than primary sensory or motor deficit.

    Value-added response: A value-added response notes that baseline education and language background must be considered when interpreting cognitive test results.

12. **Basic response:** Dementia is a syndrome with several causes, most commonly Alzheimer's disease, vascular dementia, Lewy body dementia, and frontotemporal dementia, each with a distinct clinical pattern.

    Value-added response: A value-added response notes that dementia must be actively distinguished from delirium and depressive pseudodementia before a specific diagnosis is pursued.

13. **Basic response:** Stroke is classified as ischaemic or haemorrhagic, requiring urgent imaging to distinguish; presentation localises to the affected arterial territory; acute assessment prioritises rapid recognition and precise onset time documentation.

    Value-added response: A value-added response notes that posterior circulation stroke can present less typically, requiring active clinical suspicion.

14. **Basic response:** Ischaemic stroke management uses time-critical thrombolysis or thrombectomy; haemorrhagic stroke management prioritises blood pressure control, anticoagulation reversal, and monitoring, with surgery reserved for selected cases.

    Value-added response: A value-added response notes that thrombectomy can be combined with thrombolysis in eligible patients rather than used as an alternative.

15. **Basic response:** Secondary prevention addresses the underlying stroke mechanism through antithrombotic therapy, risk factor management, and lifestyle modification; rehabilitation begins early and involves multidisciplinary therapy.

    Value-added response: A value-added response notes that structured, early rehabilitation genuinely improves functional outcome and independence.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Domains include orientation, attention, memory, language, visuospatial function, and executive function.
2. A low score can reflect education or language background rather than genuine cognitive impairment.
3. Broca's aphasia is non-fluent with preserved comprehension, while Wernicke's aphasia is fluent with impaired comprehension.
4. Conduction aphasia reflects damage to the arcuate fasciculus connecting Broca's and Wernicke's areas.
5. Agnosia is a failure of recognition, while apraxia is a failure of skilled movement, despite intact sensation and strength respectively.

Part 1.2

1. The four subtypes are Alzheimer's disease, vascular dementia, dementia with Lewy bodies, and frontotemporal dementia.
2. Delirium is acute and fluctuating with a demonstrable cause, while dementia is chronic and progressive.
3. Alzheimer's disease typically begins with prominent short-term memory impairment.
4. Vascular dementia typically follows a stepwise decline after discrete vascular events.
5. Dementia with Lewy bodies presents with fluctuating cognition and hallucinations; frontotemporal dementia presents with personality or language change with preserved memory early on.

Part 1.3

1. Ischaemic stroke results from arterial occlusion, while haemorrhagic stroke results from bleeding into the brain.
2. Subclassifications include large artery atherosclerosis, cardioembolism, and small vessel disease.

3. Middle cerebral artery stroke typically causes face and arm weakness and sensory loss, with aphasia if dominant.
4. Onset time directly determines eligibility for time-sensitive treatments such as thrombolysis.
5. Urgent CT primarily excludes haemorrhage before considering thrombolytic therapy.

Part 1.4

1. Thrombolysis dissolves the clot; its benefit diminishes progressively the longer treatment is delayed.
2. Mechanical thrombectomy physically removes a clot from a large occluded vessel using an endovascular technique.
3. Priorities include blood pressure control, anticoagulation reversal, and monitoring for deterioration.
4. Surgical evacuation is particularly considered for cerebellar haemorrhage causing significant mass effect or hydrocephalus.
5. Components include antithrombotic therapy, risk factor management, and early multidisciplinary rehabilitation.

References and Attributions [Series 1]

- Ropper, A. H., Samuels, M. A., & Klein, J. P. (2019). Adams and Victor's Principles of Neurology (11th ed.). McGraw-Hill.
- Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
- Warlow, C. P., van Gijn, J., Dennis, M. S., Wardlaw, J. M., Bamford, J. M., Hankey, G. J., & Sandercock, P. A. (2008). Stroke: Practical Management (3rd ed.). Wiley-Blackwell.
- National Institute for Health and Care Excellence (2022). Stroke and Transient Ischaemic Attack in Over 16s: Diagnosis and Initial Management (NG128). NICE.

Figures, Plates, Tables, and Boxes

- Figure 1.1: A clinician assessing language function through object naming during bedside examination. AI-generated using the prompt: "Create a realistic clinical illustration titled Bedside Language Assessment, showing a clinician holding up a common object and

asking a seated patient to name it during a cognitive examination, consultation room setting. Clean clinical illustration style, no text."

- Table 1.1: Comparing the major dementia subtypes. AI-generated using the prompt: "Create a clean reference table titled Comparing the Major Dementia Subtypes, listing dementia type, typical early feature, and course. Simple clinical reference table style with outside border."
- Figure 1.2: A labelled diagram of the major cerebral arterial territories. AI-generated using the prompt: "Create a clean, accurate labelled anatomical diagram titled Cerebral Arterial Territories, showing a lateral view of the brain with the anterior cerebral artery, middle cerebral artery, and posterior circulation territories clearly labelled and shaded distinctly. Educational anatomical diagram style, no photographic content."
- Table 1.2: Comparing acute management priorities in ischaemic and haemorrhagic stroke. AI-generated using the prompt: "Create a clean reference table titled Comparing Acute Management Priorities in Ischaemic and Haemorrhagic Stroke, listing feature, ischaemic stroke, and haemorrhagic stroke. Simple clinical reference table style with outside border."

Series 2: Epilepsy, Sleep Disorders, Parkinson's Disease, and Headache

- **Part 2.1: Epilepsy**
  - Sub Part 2.1.1: Classification of seizures and epilepsy
  - Sub Part 2.1.2: Clinical presentation and diagnosis of epilepsy
  - Sub Part 2.1.3: Management of epilepsy, including status epilepticus
- **Part 2.2: Sleep Disorders**
  - Sub Part 2.2.1: Normal sleep physiology and classification of sleep disorders
  - Sub Part 2.2.2: Insomnia and sleep apnoea
  - Sub Part 2.2.3: Parasomnias and narcolepsy
- **Part 2.3: Parkinson's Disease**
  - Sub Part 2.3.1: Pathophysiology of Parkinson's disease
  - Sub Part 2.3.2: Clinical presentation of Parkinson's disease
  - Sub Part 2.3.3: Management of Parkinson's disease
- **Part 2.4: Headache**
  - Sub Part 2.4.1: Classification and approach to headache
  - Sub Part 2.4.2: Primary headache disorders: migraine and tension-type headache
  - Sub Part 2.4.3: Secondary headache and red flag features

Introduction

This Series turns to four genuinely common presenting problems in neurological practice, epilepsy, sleep disorders, Parkinson's disease, and headache, conditions that together account for a substantial proportion of everyday neurology clinic referrals. Though clinically distinct, each demands the same careful history-taking discipline, since the diagnosis in every one of these four areas rests overwhelmingly on the clinical history rather than any single confirmatory test.

The aim of this Series is to equip you with the ability to classify seizures and epilepsy, describe its presentation, diagnosis, and management including status epilepticus, describe common sleep disorders, describe the pathophysiology, presentation, and management of Parkinson's disease, and classify headache, describing both primary headache disorders and the red flag features suggesting a secondary cause.

This Series opens with **epilepsy**, moves through **sleep disorders** and **Parkinson's disease**, and closes with **headache**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 2.1:** classify seizures and epilepsy
- **SLO 2.2:** describe the clinical presentation, diagnosis, and management of epilepsy
- **SLO 2.3:** discuss status epilepticus
- **SLO 2.4:** describe sleep disorders, including insomnia, sleep apnoea, parasomnias, and narcolepsy
- **SLO 2.5:** describe the pathophysiology and clinical presentation of Parkinson's disease
- **SLO 2.6:** discuss the management of Parkinson's disease
- **SLO 2.7:** classify headache and describe primary headache disorders
- **SLO 2.8:** discuss secondary headache and red flag features

2.1 Epilepsy

2.1.1 classification of seizures and epilepsy

Where the electrical disturbance begins, and how it spreads

Seizures are classified according to their site of onset: **focal seizures**, beginning in a specific brain region and producing symptoms reflecting that region's normal function, and **generalised seizures**, involving both hemispheres from the outset, including the familiar **tonic-clonic seizure**, with stiffening followed by rhythmic jerking, and **absence seizures**, brief episodes of impaired awareness without significant motor activity, most commonly seen in children.

Epilepsy, a tendency towards recurrent, unprovoked seizures, is distinguished from a single, isolated seizure or a seizure clearly provoked by an acute cause such as fever, alcohol withdrawal, or acute metabolic disturbance, since this distinction carries direct implications for

both the required investigation and the appropriateness of long-term anticonvulsant treatment, a genuinely important classification step before any specific epilepsy diagnosis is confirmed.

Fill in the blank: Focal seizures begin in a specific brain region, producing symptoms reflecting that region's normal _____.

2.1.2 clinical presentation and diagnosis of epilepsy

A diagnosis resting overwhelmingly on the history

Diagnosis of epilepsy relies primarily on a detailed history of the seizure event itself, ideally supplemented by an eyewitness account, since the patient themselves often cannot recall the event clearly, covering the specific sequence of symptoms, duration, any preceding aura, and the pattern of recovery afterwards, information that together allows a confident distinction between epilepsy and its common mimics, particularly syncope and psychogenic non-epileptic seizures.

Electroencephalography, recording the brain's electrical activity, and **magnetic resonance imaging**, identifying a structural cause where present, support the clinical diagnosis and help characterise the specific epilepsy syndrome, though a normal electroencephalogram between seizures does not exclude epilepsy, since the interictal recording can genuinely appear normal in many patients with a well-established, unequivocal clinical diagnosis.

Fill in the blank: A normal electroencephalogram between seizures does not exclude epilepsy given the recording can appear normal _____.

2.1.3 management of epilepsy, including status epilepticus

Long-term control, and a genuine acute emergency

Anticonvulsant medication, selected according to the specific seizure type and epilepsy syndrome, patient factors including age and comorbidities, and, in women of childbearing potential, the specific teratogenic risk profile of the chosen agent, forms the mainstay of long-term epilepsy management, with the goal of achieving complete seizure freedom with minimal side effects wherever genuinely achievable.

Status epilepticus, a prolonged seizure or repeated seizures without full recovery of consciousness between them, is a genuine medical emergency requiring urgent treatment with intravenous benzodiazepine as first-line therapy, followed by a second-line anticonvulsant if the seizure continues, since prolonged, unaddressed seizure activity carries a genuine risk of permanent neurological injury the longer it continues untreated.

Fill in the blank: Status epilepticus is a prolonged seizure or repeated seizures without full recovery of _____ between them.

Figure 2.1: A technician placing electroencephalography electrodes on a patient's scalp.

Pilot questions 2.1

- Distinguish focal from generalised seizures. (Linked to SLO 2.1)
- Distinguish epilepsy from a single, provoked seizure. (Linked to SLO 2.1)
- Explain why eyewitness account matters in diagnosing epilepsy. (Linked to SLO 2.2)
- Explain why a normal interictal electroencephalogram does not exclude epilepsy. (Linked to SLO 2.2)
- Describe the emergency management of status epilepticus. (Linked to SLO 2.3)

2.2 Sleep Disorders

2.2.1 normal sleep physiology and classification of sleep disorders

A structured, cyclical process, and what happens when it fails

Normal sleep cycles through distinct stages, alternating between **rapid eye movement sleep**, associated with vivid dreaming and characteristic muscle atonia, and **non-rapid eye movement sleep**, itself comprising lighter and deeper stages, across several cycles through the night, a structured architecture that, when disrupted, underlies many of the clinical sleep disorders covered across this Part.

Sleep disorders are broadly classified into disorders of initiating or maintaining sleep, such as insomnia, disorders of excessive daytime sleepiness, such as sleep apnoea and narcolepsy, and disorders of abnormal behaviour or movement during sleep, termed **parasomnias**, a classification framework that guides the more detailed discussion of specific conditions across the following two sub-Parts of this Part.

Fill in the blank: Rapid eye movement sleep is associated with vivid dreaming and characteristic muscle _____.

2.2.2 insomnia and sleep apnoea

Two of the most commonly encountered sleep complaints

Insomnia, persistent difficulty initiating or maintaining sleep causing daytime impairment, is frequently associated with anxiety, depression, poor sleep hygiene, or an underlying medical condition, and management prioritises addressing any identifiable underlying cause alongside structured cognitive behavioural therapy for insomnia, which offers genuinely more durable benefit than pharmacological treatment alone for most patients with chronic insomnia.

Obstructive sleep apnoea, repeated upper airway collapse during sleep causing recurrent breathing interruption, presents with loud snoring, witnessed apnoeic episodes, and excessive daytime sleepiness, and is strongly associated with obesity, and untreated obstructive sleep apnoea carries genuine cardiovascular consequences over time, making confirmation through overnight sleep study and, where indicated, treatment with continuous positive airway pressure therapy genuinely important beyond simply addressing the sleep disturbance itself.

Fill in the blank: Untreated obstructive sleep apnoea carries genuine _____ consequences over time.

2.2.3 parasomnias and narcolepsy

Abnormal behaviours during sleep, and an inability to stay awake

Parasomnias, including sleepwalking and night terrors typically arising from non-rapid eye movement sleep, and rapid eye movement sleep behaviour disorder, characterised by loss of the normal muscle atonia during rapid eye movement sleep allowing patients to physically act out dreams, sometimes with genuine injury risk to themselves or a bed partner, each present with distinctive, recognisable clinical patterns worth confidently distinguishing from one another.

Narcolepsy, a disorder of excessive daytime sleepiness with sudden, irresistible sleep episodes, is frequently accompanied by **cataplexy**, sudden loss of muscle tone triggered by strong emotion, particularly laughter, a specific and genuinely diagnostically valuable associated feature, and management combines behavioural strategies, including scheduled daytime naps, with specific pharmacological treatment targeting both the excessive sleepiness and, where present, cataplexy.

Fill in the blank: Cataplexy is sudden loss of muscle tone triggered by strong emotion, particularly _____.

Table 2.1: Comparing common sleep disorders

Disorder	Core feature	Key associated finding
Insomnia	Difficulty initiating or maintaining sleep	Often linked to anxiety or depression
Obstructive sleep apnoea	Repeated airway collapse during sleep	Loud snoring, obesity association
Narcolepsy	Irresistible daytime sleep episodes	Cataplexy triggered by emotion

Pilot questions 2.2

1. Describe the stages of normal sleep. (Linked to SLO 2.4)
2. List the broad classification categories of sleep disorders. (Linked to SLO 2.4)
3. Describe the management approach to chronic insomnia. (Linked to SLO 2.4)
4. Describe the clinical presentation of obstructive sleep apnoea. (Linked to SLO 2.4)
5. Describe narcolepsy and cataplexy. (Linked to SLO 2.4)

2.3 Parkinson's Disease

2.3.1 pathophysiology of parkinson's disease

Progressive loss of dopamine-producing neurons

Parkinson's disease results from progressive degeneration of dopamine-producing neurons within the substantia nigra, a structure deep within the midbrain, leading to progressive dopamine deficiency within the basal ganglia circuitry that normally coordinates smooth, controlled voluntary movement, and this specific, well-characterised neurochemical deficiency directly underlies both the characteristic clinical features and the pharmacological treatment approach covered later in this Part.

Pathologically, affected neurons characteristically contain **Lewy bodies**, abnormal protein aggregates, and the underlying cause of this specific pattern of neuronal degeneration in most cases remains genuinely incompletely understood, involving a probable combination of genetic susceptibility and environmental factors, though a small proportion of cases are attributable to specific identified genetic mutations, particularly in patients with an early age of onset.

Fill in the blank: Parkinson's disease results from progressive degeneration of dopamine-producing neurons within the substantia _____.

2.3.2 clinical presentation of parkinson's disease

A recognisable triad, and features extending well beyond it

The classic **motor triad** of Parkinson's disease comprises **resting tremor**, characteristically a pill-rolling tremor most prominent at rest and reducing with voluntary movement, **rigidity**, increased resistance to passive movement often described as cogwheel in character, and **bradykinesia**, slowness of movement initiation, alongside postural instability emerging typically later in the disease course, together producing the characteristic shuffling gait and reduced facial expression recognisable even to non-specialists.

Non-motor features, including autonomic dysfunction, sleep disturbance, cognitive impairment, and depression, are increasingly recognised as genuinely significant contributors to overall disease burden and quality of life, sometimes preceding the classic motor features by several years, and actively screening for and addressing these non-motor symptoms, rather than focusing exclusively on the motor triad, forms a genuinely essential part of comprehensive Parkinson's disease care.

Fill in the blank: Resting tremor in Parkinson's disease is characteristically pill-rolling and reduces with voluntary _____.

2.3.3 management of parkinson's disease

Replacing dopamine, and supporting the whole patient

Levodopa, converted to dopamine within the brain, remains the most effective symptomatic treatment for the motor features of Parkinson's disease, though long-term use is associated with **motor fluctuations** and **dyskinesias**, involuntary movements developing over years of treatment, complications that genuinely complicate long-term management and require careful, experienced adjustment of medication timing and dosing as the disease and treatment response evolve.

Other pharmacological options, including dopamine agonists and monoamine oxidase-B inhibitors, offer alternative or adjunctive treatment, particularly earlier in the disease course, and **deep brain stimulation**, a surgical option for appropriately selected patients with more advanced disease and significant motor fluctuations despite optimised medical therapy, alongside physiotherapy, occupational therapy, and speech therapy, together form a genuinely comprehensive, multidisciplinary approach to management extending well beyond medication alone.

Fill in the blank: Long-term levodopa use is associated with motor fluctuations and _____, involuntary movements developing over years of treatment.

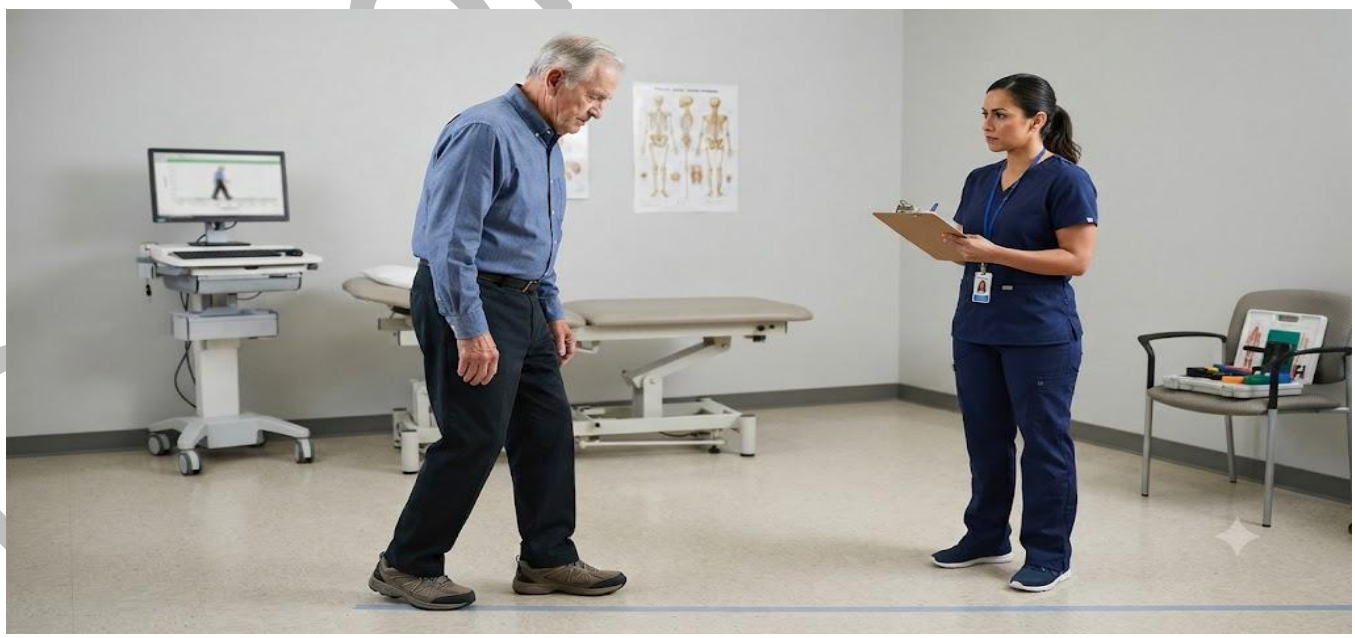

Figure 2.2: A clinician observing a patient's gait during a Parkinson's disease assessment.

Pilot questions 2.3

1. Describe the underlying pathophysiology of Parkinson's disease. (Linked to SLO 2.5)
2. Describe the classic motor triad of Parkinson's disease. (Linked to SLO 2.5)
3. List non-motor features of Parkinson's disease. (Linked to SLO 2.5)
4. Describe levodopa and its long-term complications. (Linked to SLO 2.6)
5. Describe deep brain stimulation and its typical indication. (Linked to SLO 2.6)

2.4 Headache

2.4.1 classification and approach to headache

Distinguishing primary from secondary at the very first assessment

Headache is broadly classified as **primary**, where the headache itself is the disorder, including migraine and tension-type headache, or **secondary**, where headache is a symptom of an underlying identifiable cause, and this fundamental distinction guides the entire subsequent assessment, since a systematic approach actively screening for red flag features, covered later in this Part, must accompany every headache assessment regardless of how typical the presentation initially appears.

A thorough headache history covers onset pattern, character, location, associated symptoms, triggers, and response to previous treatment, and, in the great majority of patients presenting with headache, a confident primary headache diagnosis can be reached through careful history alone without requiring neuroimaging, reserved instead for patients with red flag features or an atypical presentation genuinely warranting further investigation.

Fill in the blank: A systematic approach actively screening for red flag features must accompany every headache _____.

2.4.2 primary headache disorders: migraine and tension-type headache

The two most commonly encountered primary headache disorders

Migraine presents with recurrent, typically unilateral, throbbing headache, often accompanied by nausea, photophobia, phonophobia, and worsening with routine physical activity, and, in a proportion of patients, preceded by a characteristic **aura**, transient neurological symptoms, most

commonly visual, immediately preceding or accompanying the headache itself, a specific pattern with direct relevance to both diagnosis and, in some cases, treatment choice.

Tension-type headache, the most common primary headache disorder overall, presents with bilateral, pressing or tightening pain of mild to moderate intensity, typically without the significant nausea or marked light and sound sensitivity characteristic of migraine, and management for both conditions combines acute treatment for individual attacks with preventive treatment for patients experiencing frequent or significantly disabling episodes.

Fill in the blank: Migraine aura describes transient neurological symptoms, most commonly _____, preceding or accompanying the headache.

2.4.3 secondary headache and red flag features

The features that must never be overlooked

Red flag features in headache assessment, including sudden-onset severe headache reaching maximal intensity within seconds, headache with fever and neck stiffness, new headache in a patient over fifty, headache with focal neurological deficit or papilloedema, and headache significantly worsened by coughing, straining, or postural change, each warrant urgent further evaluation given their association with a serious underlying secondary cause.

Recognising these red flags promptly is genuinely essential, since conditions including subarachnoid haemorrhage, meningitis, and raised intracranial pressure from an intracranial mass, each already discussed in earlier courses of this programme, can present initially with headache as the predominant or even sole symptom, making active, deliberate screening for these features a genuinely non-negotiable part of every headache assessment regardless of how typical or reassuring the overall clinical picture otherwise appears.

Fill in the blank: A sudden-onset severe headache reaching maximal intensity within seconds is a recognised red _____.

Table 2.2: Comparing migraine and tension-type headache

Feature	Migraine	Tension-type headache
Location	Typically unilateral	Typically bilateral
Character	Throbbing	Pressing or tightening
Associated symptoms	Nausea, photophobia, phonophobia	Usually absent or mild

Pilot questions 2.4

1. Distinguish primary from secondary headache. (Linked to SLO 2.7)
2. Describe the components of a thorough headache history. (Linked to SLO 2.7)
3. Describe the clinical features of migraine, including aura. (Linked to SLO 2.7)
4. Distinguish tension-type headache from migraine. (Linked to SLO 2.7)
5. List red flag features in headache assessment. (Linked to SLO 2.8)

Series Summary

This Series covered four commonly encountered neurological presentations. Part 2.1 covered the classification, presentation, diagnosis, and management of epilepsy, including status epilepticus, while Part 2.2 turned to sleep disorders, insomnia, sleep apnoea, parasomnias, and narcolepsy. Part 2.3 covered the pathophysiology, presentation, and management of Parkinson's disease, and Part 2.4 closed with headache, primary headache disorders, and the red flag features suggesting a secondary cause.

You should now be able to classify and describe epilepsy, sleep disorders, Parkinson's disease, and headache across all four Parts of this Series, meeting the outcomes set for this Series. Series 3 turns next to spinal cord lesions, peripheral neuropathy, and neuromuscular diseases.

Glossary of Terms

1. **Aura:** transient neurological symptoms, most commonly visual, preceding or accompanying migraine headache.
2. **Bradykinesia:** slowness of movement initiation, a core feature of Parkinson's disease.
3. **Cataplexy:** sudden loss of muscle tone triggered by strong emotion, associated with narcolepsy.
4. **Dyskinesia:** involuntary movements developing as a long-term complication of levodopa therapy.
5. **Epilepsy:** a tendency towards recurrent, unprovoked seizures.
6. **Focal seizure:** a seizure beginning in a specific brain region.
7. **Generalised seizure:** a seizure involving both hemispheres from the outset.
8. **Levodopa:** the most effective symptomatic treatment for the motor features of Parkinson's disease.

9. **Migraine:** a primary headache disorder with recurrent, typically unilateral, throbbing headache.
10. **Obstructive sleep apnoea:** repeated upper airway collapse during sleep causing breathing interruption.
11. **Status epilepticus:** a prolonged seizure or repeated seizures without recovery of consciousness between them.
12. **Tension-type headache:** the most common primary headache disorder, bilateral and pressing in character.

Concept Check Questions [Series 2]

Objective questions

- Focal seizures begin in a specific brain region, producing symptoms reflecting that region's normal _____. (Linked to SLO 2.1)
- Status epilepticus is a prolonged seizure or repeated seizures without full recovery of _____ between them. (Linked to SLO 2.3)
- Untreated obstructive sleep apnoea carries genuine _____ consequences over time. (Linked to SLO 2.4)
- Parkinson's disease results from progressive degeneration of dopamine-producing neurons within the substantia _____. (Linked to SLO 2.5)
- A sudden-onset severe headache reaching maximal intensity within seconds is a recognised red _____. (Linked to SLO 2.8)

Short-answer questions

1. Distinguish epilepsy from a single, provoked seizure. (Linked to SLO 2.1)
2. Describe the clinical presentation of obstructive sleep apnoea. (Linked to SLO 2.4)
3. Describe the classic motor triad of Parkinson's disease. (Linked to SLO 2.5)
4. Describe levodopa and its long-term complications. (Linked to SLO 2.6)
5. Distinguish tension-type headache from migraine. (Linked to SLO 2.7)

Long-answer questions

6. Discuss the classification, presentation, diagnosis, and management of epilepsy, including status epilepticus. (Linked to SLO 2.1, 2.2, 2.3)
7. Describe sleep disorders, including insomnia, sleep apnoea, parasomnias, and narcolepsy. (Linked to SLO 2.4)
8. Discuss the pathophysiology, presentation, and management of Parkinson's disease. (Linked to SLO 2.5, 2.6)

9. Describe the classification and clinical features of primary headache disorders. (Linked to SLO 2.7)
10. Discuss secondary headache and the red flag features that warrant further evaluation. (Linked to SLO 2.8)

Answers to Concept Check Questions [Series 2]

Objective questions

11. Function.
12. Consciousness.
13. Cardiovascular.
14. Nigra.
15. Flag.

Short-answer questions

16. Epilepsy is a tendency towards recurrent, unprovoked seizures, unlike a single seizure clearly provoked by an acute cause.
17. Obstructive sleep apnoea presents with loud snoring, witnessed apnoeic episodes, and excessive daytime sleepiness.
18. The triad is resting tremor, rigidity, and bradykinesia, alongside later postural instability.
19. Levodopa is converted to dopamine in the brain; long-term use causes motor fluctuations and dyskinesias.
20. Tension-type headache is bilateral and pressing without significant nausea, unlike unilateral, throbbing migraine with associated symptoms.

Long-answer questions

21. **Basic response:** Seizures are classified as focal or generalised; diagnosis relies on detailed history supported by EEG and MRI; management uses anticonvulsant medication; status epilepticus is a genuine emergency treated with intravenous benzodiazepine.

    Value-added response: A value-added response notes that a normal interictal EEG does not exclude a well-established clinical diagnosis of epilepsy.

22. **Basic response:** Insomnia involves difficulty initiating or maintaining sleep; obstructive sleep apnoea involves repeated airway collapse; parasomnias involve abnormal sleep behaviours; narcolepsy involves irresistible daytime sleep episodes often with cataplexy.

Value-added response: A value-added response notes that cognitive behavioural therapy offers more durable benefit than medication alone for chronic insomnia.

23. **Basic response:** Parkinson's disease results from dopaminergic neuron loss in the substantia nigra, presenting with resting tremor, rigidity, and bradykinesia; management uses levodopa and other agents, with deep brain stimulation for selected advanced cases.

    Value-added response: A value-added response notes that non-motor features can precede the classic motor triad by several years and deserve active screening.

24. **Basic response:** Headache is classified as primary or secondary; migraine presents with unilateral, throbbing pain with associated symptoms and sometimes aura; tension-type headache presents with bilateral, pressing pain without significant associated symptoms.

    Value-added response: A value-added response notes that most primary headache diagnoses can be confidently reached through history alone without neuroimaging.

25. **Basic response:** Red flags include sudden severe onset, fever with neck stiffness, new headache over fifty, focal deficit or papilloedema, and worsening with coughing or straining.

    Value-added response: A value-added response notes that serious secondary causes can present with headache as the predominant or sole initial symptom.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Focal seizures begin in a specific region, while generalised seizures involve both hemispheres from onset.
2. Epilepsy is recurrent and unprovoked, unlike a single seizure with a clear acute provoking cause.
3. Eyewitness account provides objective detail the patient often cannot recall themselves.
4. The interictal recording can genuinely appear normal even in a well-established, unequivocal clinical diagnosis.
5. Status epilepticus is treated urgently with intravenous benzodiazepine, followed by a second-line agent if needed.

Part 2.2

6. Sleep cycles through rapid eye movement and non-rapid eye movement stages across several cycles nightly.
7. Categories include disorders of initiating or maintaining sleep, excessive daytime sleepiness, and parasomnias.
8. Management prioritises addressing underlying cause alongside cognitive behavioural therapy for insomnia.
9. Obstructive sleep apnoea presents with loud snoring, witnessed apnoeic episodes, and daytime sleepiness.
10. Narcolepsy is irresistible daytime sleep episodes, often with cataplexy triggered by strong emotion.

Part 2.3

11. Parkinson's disease results from progressive dopaminergic neuron loss in the substantia nigra.
12. The triad is resting tremor, rigidity, and bradykinesia.
13. Non-motor features include autonomic dysfunction, sleep disturbance, cognitive impairment, and depression.
14. Levodopa is converted to dopamine in the brain but causes motor fluctuations and dyskinesias with long-term use.
15. Deep brain stimulation is a surgical option for selected patients with advanced disease and significant motor fluctuations.

Part 2.4

1. Primary headache is itself the disorder, while secondary headache is a symptom of an identifiable underlying cause.
2. A thorough history covers onset, character, location, associated symptoms, triggers, and treatment response.
3. Migraine is unilateral and throbbing with nausea and photophobia, sometimes with a preceding visual aura.
4. Tension-type headache is bilateral and pressing, without the significant associated symptoms seen in migraine.
5. Red flags include sudden severe onset, fever with neck stiffness, new headache over fifty, and focal deficit.

References and Attributions [Series 2]

1. Ropper, A. H., Samuels, M. A., & Klein, J. P. (2019). Adams and Victor's Principles of Neurology (11th ed.). McGraw-Hill.
2. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
3. International League Against Epilepsy (2017). Classification of Seizures and Epilepsy. Epilepsia.
4. International Headache Society (2018). The International Classification of Headache Disorders (3rd ed.). Cephalalgia.

Figures, Plates, Tables, and Boxes

1. Figure 2.1: A technician placing electroencephalography electrodes on a patient's scalp. AI-generated using the prompt: "Create a realistic clinical illustration titled Electroencephalography Electrode Placement, showing a technician carefully attaching small electrodes to a seated patient's scalp using a measuring cap, neurophysiology department setting. Clean clinical illustration style, no text."
2. Table 2.1: Comparing common sleep disorders. AI-generated using the prompt: "Create a clean reference table titled Comparing Common Sleep Disorders, listing disorder, core feature, and key associated finding. Simple clinical reference table style with outside border."
3. Figure 2.2: A clinician observing a patient's gait during a Parkinson's disease assessment. AI-generated using the prompt: "Create a realistic clinical illustration titled Gait Assessment in Parkinson's Disease, showing a clinician observing a patient walking across a clinic room with a stooped posture and reduced arm swing, clinical examination setting. Clean clinical illustration style, no text."
4. Table 2.2: Comparing migraine and tension-type headache. AI-generated using the prompt: "Create a clean reference table titled Comparing Migraine and Tension-Type Headache, listing feature, migraine, and tension-type headache. Simple clinical reference table style with outside border."

Course code: MED 604

Course title: Neurology II

Course credit load: 2 Units

Series 3: Spinal Cord Lesions, Peripheral Neuropathy, and Neuromuscular Diseases

- **Part 3.1: Spinal Cord Lesions**
    - Sub Part 3.1.1: Anatomy and localisation of spinal cord lesions
    - Sub Part 3.1.2: Clinical syndromes of spinal cord disease
    - Sub Part 3.1.3: Approach to myelopathy
- **Part 3.2: Peripheral Neuropathy**
    - Sub Part 3.2.1: Classification of peripheral neuropathy
    - Sub Part 3.2.2: Clinical presentation and pattern recognition in peripheral neuropathy
    - Sub Part 3.2.3: Common causes and investigation of peripheral neuropathy
- **Part 3.3: Neuromuscular Junction and Muscle Disease**
    - Sub Part 3.3.1: Myasthenia gravis: pathophysiology and presentation
    - Sub Part 3.3.2: Myasthenia gravis: diagnosis and management
    - Sub Part 3.3.3: Other neuromuscular junction disorders
- **Part 3.4: Muscular Dystrophy and Myopathy**
    - Sub Part 3.4.1: Classification and approach to myopathy
    - Sub Part 3.4.2: Duchenne and other muscular dystrophies
    - Sub Part 3.4.3: Management of muscular dystrophy

Introduction

This Series moves progressively down the neuraxis, from the spinal cord itself, through the peripheral nerves carrying signals to and from it, to the neuromuscular junction and muscle tissue that ultimately execute movement. **Spinal cord lesions, peripheral neuropathy, and neuromuscular disease** each produce weakness or sensory disturbance, but the specific pattern each produces differs in genuinely recognisable, learnable ways once the anatomical level of the lesion is correctly identified.

The aim of this Series is to equip you with the ability to describe the anatomy, localisation, and clinical syndromes of spinal cord lesions, classify peripheral neuropathy and describe its presentation and causes, describe the pathophysiology, presentation, diagnosis, and management of myasthenia gravis and other neuromuscular junction disorders, and classify myopathy and describe muscular dystrophy and its management.

This Series opens with **spinal cord lesions**, moves through **peripheral neuropathy** and **neuromuscular junction and muscle disease**, and closes with **muscular dystrophy and myopathy**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 3.1:** describe the anatomy and localisation of spinal cord lesions
- **SLO 3.2:** describe the clinical syndromes of spinal cord disease
- **SLO 3.3:** classify peripheral neuropathy and describe its clinical presentation
- **SLO 3.4:** discuss the common causes and investigation of peripheral neuropathy
- **SLO 3.5:** describe the pathophysiology, presentation, diagnosis, and management of myasthenia gravis
- **SLO 3.6:** describe other neuromuscular junction disorders
- **SLO 3.7:** classify myopathy and describe muscular dystrophy
- **SLO 3.8:** discuss the management of muscular dystrophy

3.1 Spinal Cord Lesions

3.1.1 anatomy and localisation of spinal cord lesions

Reading the deficit to place the lesion precisely

The spinal cord contains several genuinely distinct functional tracts running its full length: the **corticospinal tract**, carrying voluntary motor signals, the **dorsal columns**, carrying vibration and proprioception, and the **spinothalamic tract**, carrying pain and temperature sensation, each occupying a specific, consistent anatomical position within the cord's cross-section, a genuinely reliable anatomical arrangement that allows the specific pattern of deficit produced to localise a lesion with real precision.

Localising a spinal cord lesion requires identifying both the **level** of the lesion, from the specific dermatome and myotome pattern of sensory and motor involvement, and the **transverse extent**, whether the lesion affects the full cross-section of the cord or only specific tracts, a two-dimensional localisation exercise that this Part builds through the specific clinical syndromes covered in the following sub-Part.

Fill in the blank: The dorsal columns carry vibration and _____ sensation within the spinal cord.

3.1.2 clinical syndromes of spinal cord disease

Recognisable patterns from partial cord involvement

Brown-Sequard syndrome, from hemisection of the cord, produces ipsilateral motor weakness and loss of vibration and proprioception below the lesion, with contralateral loss of pain and temperature sensation, a pattern directly reflecting the specific tracts affected on each side of the injured cord, while **anterior cord syndrome**, affecting the anterior two-thirds of the cord, produces motor loss and loss of pain and temperature with relatively preserved dorsal column function.

Central cord syndrome, disproportionately affecting the centrally located fibres of the corticospinal tract, produces weakness affecting the upper limbs more than the lower limbs, and a full transverse cord lesion, affecting the entire cross-section, produces complete loss of motor and sensory function below the level of the lesion, alongside bladder and bowel dysfunction, a comprehensive pattern reflecting the involvement of every functional tract simultaneously.

Fill in the blank: Central cord syndrome produces weakness disproportionately affecting the upper limbs more than the _____ limbs.

3.1.3 approach to myelopathy

Working through a genuinely wide differential systematically

Myelopathy, spinal cord dysfunction from any cause, has a genuinely wide differential diagnosis including compressive causes such as disc disease or tumour, inflammatory causes such as transverse myelitis, infective causes, and metabolic causes such as subacute combined degeneration from vitamin B12 deficiency, each requiring a distinct diagnostic and management pathway, making systematic, structured clinical reasoning genuinely essential when a patient presents with a myelopathic pattern of deficit.

Magnetic resonance imaging of the spine is the key investigation for suspected myelopathy, both to identify a compressive cause requiring potentially urgent surgical intervention and to characterise the pattern of signal change that can support a specific non-compressive diagnosis, and prompt investigation and diagnosis genuinely matters given that several causes of myelopathy carry a genuine risk of permanent neurological deficit if treatment is delayed.

Fill in the blank: Prompt investigation of myelopathy matters given the genuine risk of permanent neurological deficit if treatment is _____.

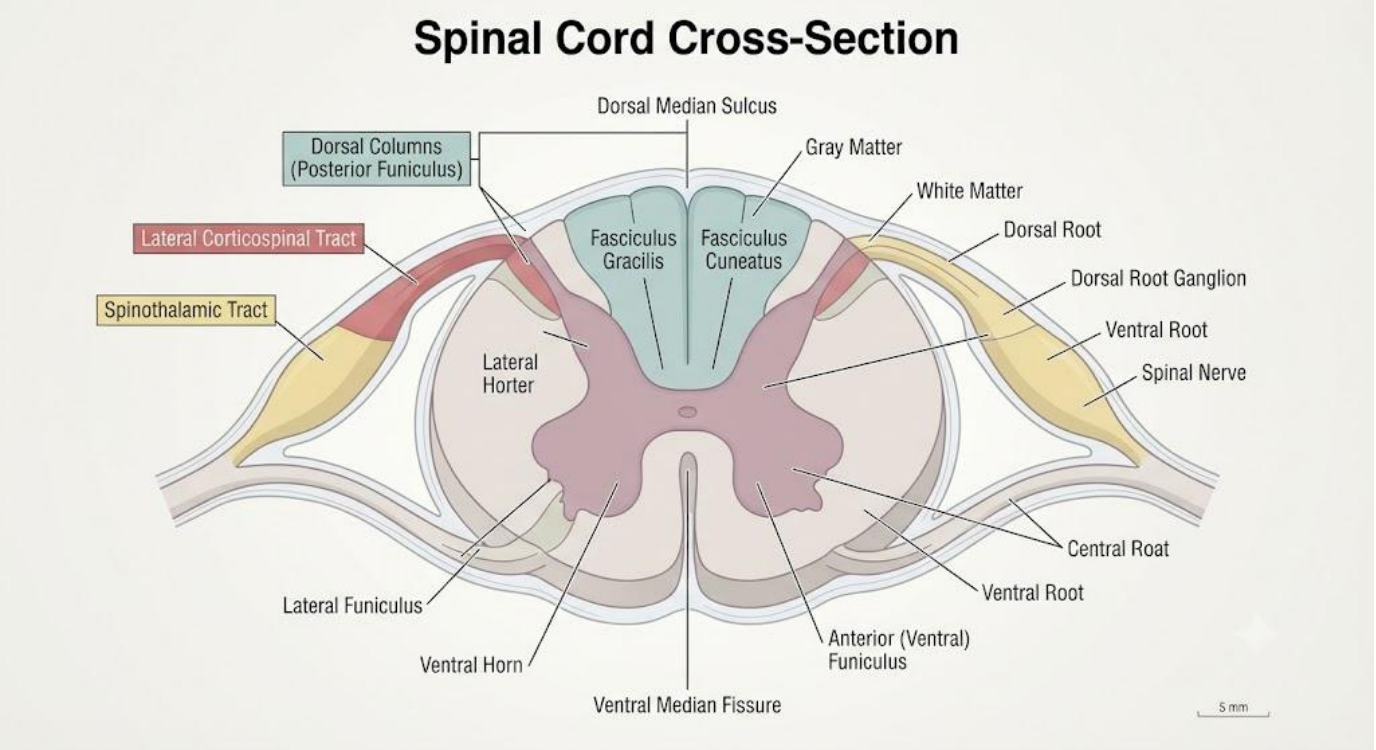

Figure 3.1: A labelled cross-sectional diagram of the spinal cord showing the major functional tracts.

Pilot questions 3.1

- List the major functional tracts of the spinal cord and what each carries. (Linked to SLO 3.1)
- Explain the two dimensions involved in localising a spinal cord lesion. (Linked to SLO 3.1)
- Describe Brown-Sequard syndrome. (Linked to SLO 3.2)
- Distinguish anterior cord syndrome from central cord syndrome. (Linked to SLO 3.2)
- List causes within the differential diagnosis of myelopathy. (Linked to SLO 3.2)

3.2 Peripheral Neuropathy

3.2.1 classification of peripheral neuropathy

Sorting neuropathy by pattern and fibre type

Peripheral neuropathy is classified by distribution, as **polyneuropathy**, a symmetrical, typically length-dependent process affecting the longest nerves first and therefore beginning distally in the feet, **mononeuropathy**, affecting a single nerve, and **mononeuritis multiplex**, affecting several individual nerves asynchronously, a distinction with genuine relevance to the likely underlying cause and required investigation pathway.

Neuropathy is further classified by the type of nerve fibre predominantly affected, **sensory**, **motor**, or **autonomic**, alone or in combination, and by the underlying pathological process, **axonal**, primarily damaging the nerve fibre itself, or **demyelinating**, primarily damaging the myelin sheath surrounding the fibre, a distinction genuinely important since it directly shapes both the differential diagnosis and the specific investigation, particularly nerve conduction studies, used to characterise the neuropathy further.

Fill in the blank: Polyneuropathy is typically length-dependent, affecting the longest nerves first and beginning _____.

3.2.2 clinical presentation and pattern recognition in peripheral neuropathy

Reading the specific pattern to narrow the differential

Sensory polyneuropathy typically presents with numbness, tingling, and, sometimes, pain in a symmetrical, glove and stocking distribution, beginning in the feet and progressing proximally over time, while **motor neuropathy** presents with distal weakness and, over time, muscle

wasting, and recognising which fibre type or types are predominantly affected provides genuinely valuable diagnostic information before specific investigation is even pursued.

Autonomic neuropathy, affecting the autonomic nervous system, can produce postural hypotension, gastrointestinal dysmotility, and bladder dysfunction, symptoms that are sometimes overlooked or misattributed unless autonomic involvement is actively considered as part of a comprehensive neuropathy assessment, reinforcing the genuine value of a systematic review of all three fibre types, sensory, motor, and autonomic, in every patient presenting with suspected peripheral neuropathy.

Fill in the blank: Autonomic neuropathy can produce postural hypotension, gastrointestinal dysmotility, and bladder _____.

3.2.3 common causes and investigation of peripheral neuropathy

A wide differential, narrowed through structured investigation

Diabetes mellitus remains the most common cause of peripheral neuropathy overall, already discussed in an earlier course of this programme, alongside other genuinely common causes including alcohol excess, vitamin B12 deficiency, chronic kidney disease, and specific medication toxicity, with **Guillain-Barre syndrome**, an acute, immune-mediated demyelinating polyneuropathy typically following an antecedent infection, representing a genuinely important cause of rapidly progressive weakness requiring urgent recognition given its potential to affect respiratory muscles.

Investigation of peripheral neuropathy combines basic blood tests screening for the common causes already mentioned with **nerve conduction studies**, distinguishing axonal from demyelinating pathology and characterising the specific distribution affected, and, where the cause remains genuinely unclear despite this initial evaluation, further specific investigation, including nerve biopsy in selected cases, may be required to reach a definitive diagnosis.

Fill in the blank: Guillain-Barre syndrome is an acute, immune-mediated demyelinating polyneuropathy typically following an antecedent _____.

Table 3.1: Comparing sensory, motor, and autonomic neuropathy patterns

Pattern	Typical symptom	Typical distribution
Sensory	Numbness, tingling, pain	Glove and stocking
Motor	Distal weakness, wasting	Length-dependent, distal first

Autonomic	Postural hypotension, dysmotility	Widespread, system-dependent

Pilot questions 3.2

1. Distinguish polyneuropathy from mononeuritis multiplex. (Linked to SLO 3.3)
2. Distinguish axonal from demyelinating neuropathy. (Linked to SLO 3.3)
3. Describe the typical presentation of sensory polyneuropathy. (Linked to SLO 3.3)
4. List common causes of peripheral neuropathy. (Linked to SLO 3.4)
5. Describe Guillain-Barre syndrome and why it requires urgent recognition. (Linked to SLO 3.4)

3.3 Neuromuscular Junction and Muscle Disease

3.3.1 myasthenia gravis: pathophysiology and presentation

An autoimmune attack on the connection between nerve and muscle

Myasthenia gravis, an autoimmune disorder resulting from antibodies directed against the acetylcholine receptor at the neuromuscular junction, disrupts normal transmission between nerve and muscle, producing the characteristic clinical hallmark of **fatigable weakness**, weakness that worsens with sustained or repeated activity and improves with rest, a genuinely distinctive pattern separating it clinically from most other causes of weakness.

Presentation frequently begins with ocular involvement, producing ptosis and diplopia that characteristically worsen through the day or with sustained upward gaze, and can progress to involve bulbar muscles, producing dysarthria and dysphagia, and limb muscles, with the specific pattern and severity of involvement varying considerably between affected individuals, ranging from purely ocular disease through to genuinely severe, generalised weakness.

Fill in the blank: The characteristic clinical hallmark of myasthenia gravis is fatigable weakness that worsens with sustained _____.

3.3.2 myasthenia gravis: diagnosis and management

Confirming the diagnosis, then restoring neuromuscular transmission

Diagnosis of myasthenia gravis relies on the characteristic clinical pattern of fatigable weakness, supported by **acetylcholine receptor antibody testing**, positive in the majority of patients, and

repetitive nerve stimulation or **single-fibre electromyography**, demonstrating the characteristic decremental response reflecting progressively impaired neuromuscular transmission with repeated stimulation, investigations that together confirm the diagnosis with genuine confidence.

Management includes **acetylcholinesterase inhibitor medication**, providing symptomatic benefit by increasing acetylcholine availability at the neuromuscular junction, alongside immunosuppressive treatment for more significant disease, and **thymectomy**, surgical removal of the thymus gland given its genuine pathophysiological role in many cases, and **myasthenic crisis**, severe weakness causing respiratory compromise, is a genuine medical emergency requiring urgent recognition and intensive supportive treatment.

Fill in the blank: Myasthenic crisis is severe weakness causing respiratory _____, a genuine medical emergency.

3.3.3 other neuromuscular junction disorders

Related conditions with genuinely distinct clinical patterns

Lambert-Eaton myasthenic syndrome, resulting from antibodies against presynaptic calcium channels rather than the postsynaptic acetylcholine receptor affected in myasthenia gravis, is frequently paraneoplastic, associated with an underlying malignancy, most commonly small cell lung cancer, and presents with proximal limb weakness that characteristically improves, rather than worsens, with sustained activity, a genuinely distinctive pattern helping distinguish it clinically from myasthenia gravis.

Botulism, resulting from botulinum toxin blocking acetylcholine release at the presynaptic terminal, produces a descending pattern of weakness beginning with cranial nerve involvement and progressing caudally, and, while genuinely rare, requires urgent recognition given its potential for severe respiratory compromise, and correctly distinguishing these related but pathophysiologically distinct neuromuscular junction disorders from one another and from myasthenia gravis is a genuinely important diagnostic skill this Part aims to build.

Fill in the blank: Lambert-Eaton myasthenic syndrome presents with weakness that characteristically improves with sustained _____.

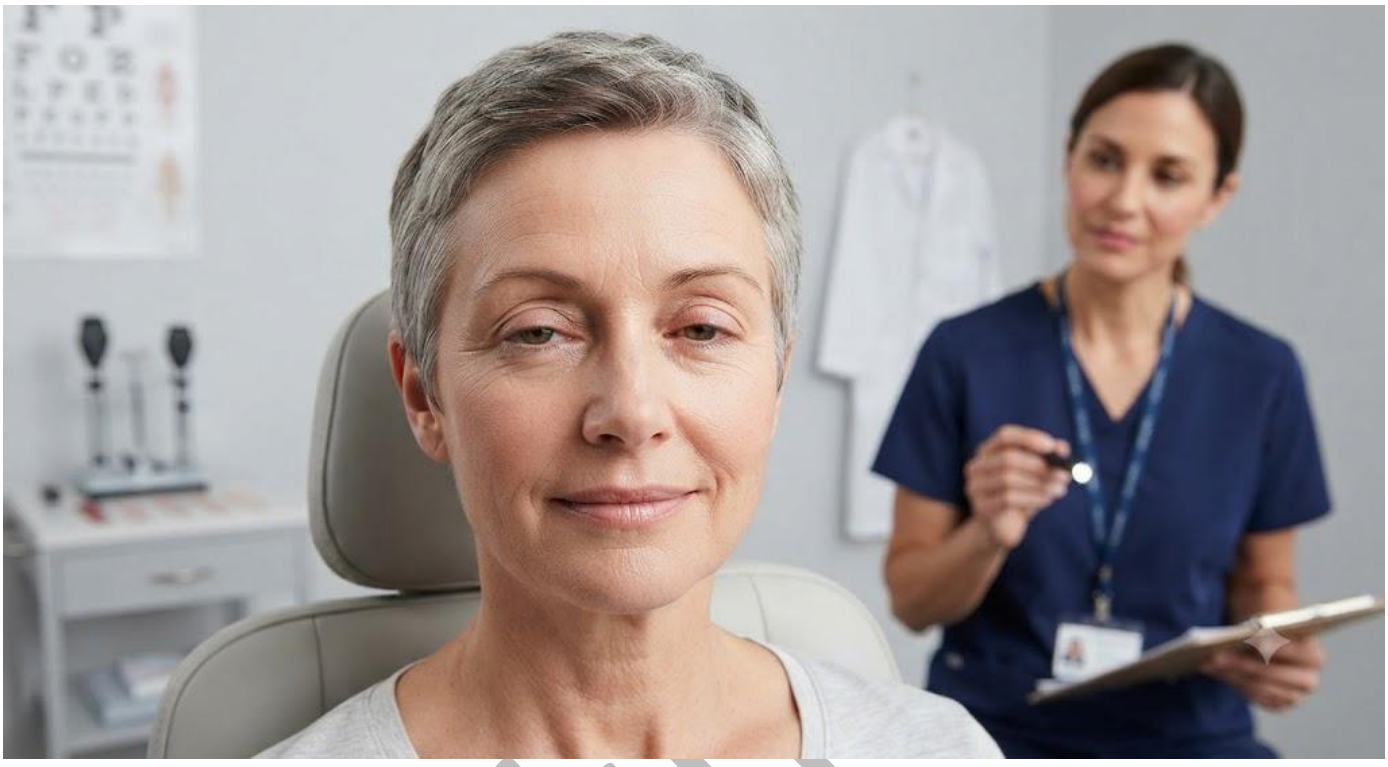

Figure 3.2: A patient with bilateral ptosis characteristic of myasthenia gravis.

Pilot questions 3.3

1. Describe the pathophysiology of myasthenia gravis. (Linked to SLO 3.5)
2. Describe fatigable weakness and its typical early presentation. (Linked to SLO 3.5)
3. List the investigations used to confirm myasthenia gravis. (Linked to SLO 3.5)
4. Describe myasthenic crisis. (Linked to SLO 3.5)
5. Distinguish Lambert-Eaton myasthenic syndrome from myasthenia gravis. (Linked to SLO 3.6)

3.4 Muscular Dystrophy and Myopathy

3.4.1 classification and approach to myopathy

Distinguishing muscle disease from other causes of weakness

Myopathy, disease primarily affecting muscle tissue itself, typically presents with **proximal weakness**, affecting shoulder and hip girdle muscles more than distal limb muscles, a pattern genuinely distinct from the distal predominance more characteristic of peripheral neuropathy,

and, unlike the fatigable weakness of myasthenia gravis, myopathic weakness is typically relatively constant rather than worsening specifically with sustained activity.

Myopathy is classified by underlying cause as **genetic**, including the muscular dystrophies covered in the following sub-Part, **inflammatory**, such as polymyositis and dermatomyositis, and **metabolic or toxic**, including drug-induced myopathy, and correctly distinguishing myopathy from other causes of weakness through this characteristic proximal, non-fatigable pattern, alongside supporting investigation including serum creatine kinase and, where indicated, muscle biopsy, forms the essential diagnostic foundation for this Part.

Fill in the blank: Myopathic weakness is typically relatively constant rather than worsening specifically with sustained _____.

3.4.2 duchenne and other muscular dystrophies

Progressive genetic muscle-wasting disorders

Duchenne muscular dystrophy, an X-linked recessive condition resulting from absence of the dystrophin protein essential for normal muscle membrane stability, is the most common and most severe childhood muscular dystrophy, presenting in early childhood with progressive proximal weakness, delayed walking, and a characteristic manoeuvre, Gower's sign, using the hands to climb up the legs when rising from the floor, reflecting significant proximal lower limb weakness.

Becker muscular dystrophy, resulting from a partial rather than complete dystrophin deficiency, produces a genuinely milder, more slowly progressive clinical course than Duchenne muscular dystrophy, and other muscular dystrophies, including facioscapulohumeral and myotonic dystrophy, present with genuinely distinct patterns of muscle involvement and inheritance, each worth recognising as part of the broader spectrum this category of genetic muscle disease encompasses.

Fill in the blank: Duchenne muscular dystrophy results from absence of the dystrophin protein essential for muscle membrane _____.

3.4.3 management of muscular dystrophy

Supportive, multidisciplinary care extending across a lifetime

Management of muscular dystrophy is genuinely multidisciplinary, combining physiotherapy to maintain function and prevent contracture, orthopaedic input for scoliosis and joint complications, respiratory support as respiratory muscle weakness progresses, and cardiac

monitoring given the genuine risk of cardiomyopathy in several muscular dystrophy subtypes, reflecting the genuinely progressive, multi-system nature of these conditions over time.

Corticosteroid therapy has been shown to meaningfully slow disease progression in Duchenne muscular dystrophy specifically, and newer, genuinely targeted molecular therapies, addressing the underlying genetic defect directly in appropriately selected patients, represent an area of active, ongoing therapeutic development, and coordinated, structured long-term follow-up, closing this Series on the same theme of comprehensive multidisciplinary care emphasised throughout this course, genuinely improves quality of life and functional outcome for affected patients and their families.

Fill in the blank: Corticosteroid therapy has been shown to meaningfully slow disease progression in Duchenne muscular _____.

Table 3.2: Comparing Duchenne and Becker muscular dystrophy

Feature	Duchenne muscular dystrophy	Becker muscular dystrophy
Dystrophin status	Absent	Partially present
Onset	Early childhood	Later childhood or adolescence
Course	Rapidly progressive	More slowly progressive

Pilot questions 3.4

1. Describe the typical pattern of weakness in myopathy. (Linked to SLO 3.7)
2. List the categories used to classify myopathy by cause. (Linked to SLO 3.7)
3. Describe Duchenne muscular dystrophy and Gower's sign. (Linked to SLO 3.7)
4. Distinguish Becker from Duchenne muscular dystrophy. (Linked to SLO 3.7)
5. Describe the multidisciplinary management approach to muscular dystrophy. (Linked to SLO 3.8)

Series Summary

This Series moved progressively down the neuraxis from the spinal cord through to muscle tissue itself. Part 3.1 covered the anatomy, localisation, and clinical syndromes of spinal cord lesions,

while Part 3.2 turned to the classification, presentation, and causes of peripheral neuropathy. Part 3.3 covered myasthenia gravis and other neuromuscular junction disorders, and Part 3.4 closed with myopathy and muscular dystrophy.

You should now be able to localise spinal cord lesions, classify and describe peripheral neuropathy, describe neuromuscular junction disorders, and describe myopathy and muscular dystrophy across all four Parts of this Series, meeting the outcomes set for this Series. Series 4 turns next to the cranial nerves, autonomic neuropathy, and coma.

Glossary of Terms

1. **Brown-Sequard syndrome:** a spinal cord hemisection syndrome with ipsilateral motor and contralateral sensory loss.
2. **Duchenne muscular dystrophy:** an X-linked muscular dystrophy from absent dystrophin, the most severe childhood form.
3. **Fatigable weakness:** weakness worsening with sustained activity and improving with rest, characteristic of myasthenia gravis.
4. **Gower's sign:** using the hands to climb up the legs when rising from the floor, reflecting proximal weakness.
5. **Guillain-Barre syndrome:** an acute, immune-mediated demyelinating polyneuropathy following infection.
6. **Lambert-Eaton myasthenic syndrome:** a paraneoplastic neuromuscular junction disorder improving with sustained activity.
7. **Myasthenia gravis:** an autoimmune disorder of the neuromuscular junction causing fatigable weakness.
8. **Myasthenic crisis:** severe myasthenic weakness causing respiratory compromise, a medical emergency.
9. **Myelopathy:** spinal cord dysfunction from any cause.
10. **Myopathy:** disease primarily affecting muscle tissue, presenting with proximal weakness.
11. **Polyneuropathy:** a symmetrical, length-dependent peripheral nerve disorder.
12. **Thymectomy:** surgical removal of the thymus gland, used in selected cases of myasthenia gravis.

Concept Check Questions [Series 3]

Objective questions

- The dorsal columns carry vibration and _____ sensation within the spinal cord. (Linked to SLO 3.1)
- Polyneuropathy is typically length-dependent, affecting the longest nerves first and beginning _____. (Linked to SLO 3.3)
- The characteristic clinical hallmark of myasthenia gravis is fatigable weakness that worsens with sustained _____. (Linked to SLO 3.5)
- Lambert-Eaton myasthenic syndrome presents with weakness that characteristically improves with sustained _____. (Linked to SLO 3.6)
- Duchenne muscular dystrophy results from absence of the dystrophin protein essential for muscle membrane _____. (Linked to SLO 3.7)

Short-answer questions

1. Describe Brown-Sequard syndrome. (Linked to SLO 3.2)
2. Distinguish axonal from demyelinating neuropathy. (Linked to SLO 3.3)
3. List the investigations used to confirm myasthenia gravis. (Linked to SLO 3.5)
4. Describe myasthenic crisis. (Linked to SLO 3.5)
5. Distinguish Becker from Duchenne muscular dystrophy. (Linked to SLO 3.7)

Long-answer questions

6. Discuss the anatomy, localisation, and clinical syndromes of spinal cord lesions. (Linked to SLO 3.1, 3.2)
7. Describe the classification, presentation, causes, and investigation of peripheral neuropathy. (Linked to SLO 3.3, 3.4)
8. Discuss the pathophysiology, presentation, diagnosis, and management of myasthenia gravis and other neuromuscular junction disorders. (Linked to SLO 3.5, 3.6)
9. Describe the classification and clinical features of myopathy and muscular dystrophy. (Linked to SLO 3.7)
10. Discuss the management of muscular dystrophy. (Linked to SLO 3.8)

Answers to Concept Check Questions [Series 3]

Objective questions

11. Proprioception.
12. Distally.
13. Activity.
14. Activity.
15. Stability.

Short-answer questions

16. Brown-Sequard syndrome produces ipsilateral motor and dorsal column loss with contralateral pain and temperature loss.
17. Axonal neuropathy damages the nerve fibre itself, while demyelinating neuropathy damages the surrounding myelin sheath.
18. Investigations include acetylcholine receptor antibody testing and repetitive nerve stimulation or single-fibre electromyography.
19. Myasthenic crisis is severe weakness causing respiratory compromise, a genuine medical emergency.
20. Becker muscular dystrophy has partial dystrophin deficiency and a milder, more slowly progressive course than Duchenne.

Long-answer questions

21. **Basic response:** The cord contains distinct tracts for motor, dorsal column, and spinothalamic function; localisation combines level and transverse extent; syndromes include Brown-Sequard, anterior cord, and central cord syndrome.

    Value-added response: A value-added response notes that myelopathy has a wide differential requiring prompt MRI given the risk of permanent deficit if treatment is delayed.

22. **Basic response:** Neuropathy is classified by distribution and fibre type; presentation reflects the affected fibre type; common causes include diabetes, alcohol, and vitamin B12 deficiency; investigation uses blood tests and nerve conduction studies.

    Value-added response: A value-added response notes that Guillain-Barre syndrome requires urgent recognition given its potential to affect respiratory muscles.

23. **Basic response:** Myasthenia gravis results from acetylcholine receptor antibodies causing fatigable weakness, diagnosed with antibody testing and nerve stimulation, managed with acetylcholinesterase inhibitors and immunosuppression; Lambert-Eaton syndrome and botulism present with distinct patterns.

    Value-added response: A value-added response notes that Lambert-Eaton syndrome is frequently paraneoplastic, associated with small cell lung cancer.

24. **Basic response:** Myopathy presents with proximal, non-fatigable weakness, classified as genetic, inflammatory, or metabolic; Duchenne muscular dystrophy is the most severe childhood form, with Gower's sign a characteristic feature.

Value-added response: A value-added response notes that Becker muscular dystrophy follows a milder course given partial rather than absent dystrophin.

25. **Basic response:** Management is multidisciplinary, combining physiotherapy, orthopaedic input, respiratory support, and cardiac monitoring, with corticosteroids slowing progression in Duchenne muscular dystrophy specifically.

    Value-added response: A value-added response notes that newer targeted molecular therapies represent an area of active, ongoing therapeutic development.

Answers to Pilot Questions [Series 3]

Part 3.1

1. The corticospinal tract carries motor signals, the dorsal columns carry vibration and proprioception, and the spinothalamic tract carries pain and temperature.
2. Localisation requires identifying both the level of the lesion and its transverse extent within the cord.
3. Brown-Sequard syndrome produces ipsilateral motor and dorsal column loss with contralateral pain and temperature loss.
4. Anterior cord syndrome spares dorsal column function, while central cord syndrome disproportionately affects the upper limbs.
5. Causes include compressive lesions, inflammatory disease, infection, and metabolic causes such as vitamin B12 deficiency.

Part 3.2

6. Polyneuropathy is symmetrical and length-dependent, while mononeuritis multiplex affects several individual nerves asynchronously.
7. Axonal neuropathy damages the nerve fibre itself, while demyelinating neuropathy damages the myelin sheath.
8. Sensory polyneuropathy presents with numbness, tingling, and pain in a glove and stocking distribution.
9. Common causes include diabetes, alcohol excess, vitamin B12 deficiency, and chronic kidney disease.
10. Guillain-Barre syndrome is an acute demyelinating polyneuropathy that can progress to affect respiratory muscles.

Part 3.3

11. Myasthenia gravis results from antibodies against the acetylcholine receptor disrupting neuromuscular transmission.
12. Fatigable weakness worsens with sustained activity, often first presenting as ptosis and diplopia.
13. Investigations include acetylcholine receptor antibody testing and repetitive nerve stimulation or single-fibre electromyography.
14. Myasthenic crisis is severe weakness causing respiratory compromise, requiring urgent intensive treatment.
15. Lambert-Eaton syndrome improves with sustained activity and is often paraneoplastic, unlike myasthenia gravis.

Part 3.4

1. Myopathy typically presents with proximal weakness affecting shoulder and hip girdle muscles more than distal muscles.
2. Categories include genetic, inflammatory, and metabolic or toxic causes.
3. Duchenne muscular dystrophy is absent dystrophin causing progressive proximal weakness; Gower's sign reflects lower limb weakness.
4. Becker muscular dystrophy has partial dystrophin deficiency and a milder, slower course than Duchenne.
5. Management combines physiotherapy, orthopaedic input, respiratory support, and cardiac monitoring.

References and Attributions [Series 3]

1. Ropper, A. H., Samuels, M. A., & Klein, J. P. (2019). Adams and Victor's Principles of Neurology (11th ed.). McGraw-Hill.
2. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
3. Bird, S. J., & Rich, M. M. (2021). Neuromuscular Disease: A Case-Based Approach. Cambridge University Press.
4. Douglas, G., Nicol, F., & Robertson, C. (2013). Macleod's Clinical Examination (13th ed.). Churchill Livingstone.

Figures, Plates, Tables, and Boxes

1. Figure 3.1: A labelled cross-sectional diagram of the spinal cord showing the major functional tracts. AI-generated using the prompt: "Create a clean, accurate labelled cross-sectional anatomical diagram titled Spinal Cord Cross-Section, showing the corticospinal tract, dorsal columns, and spinothalamic tract clearly labelled in their respective anatomical positions. Educational anatomical diagram style, no photographic content."
2. Table 3.1: Comparing sensory, motor, and autonomic neuropathy patterns. AI-generated using the prompt: "Create a clean reference table titled Comparing Sensory, Motor, and Autonomic Neuropathy Patterns, listing pattern, typical symptom, and typical distribution. Simple clinical reference table style with outside border."
3. Figure 3.2: A patient with bilateral ptosis characteristic of myasthenia gravis. AI-generated using the prompt: "Create a realistic clinical illustration titled Ptosis in Myasthenia Gravis, showing a patient's face with visible drooping of both upper eyelids, clinical examination setting, respectful and clinical in tone. Clean clinical illustration style, no text."
4. Table 3.2: Comparing Duchenne and Becker muscular dystrophy. AI-generated using the prompt: "Create a clean reference table titled Comparing Duchenne and Becker Muscular Dystrophy, listing feature, Duchenne muscular dystrophy, and Becker muscular dystrophy. Simple clinical reference table style with outside border."

Course code: MED 604

Course title: Neurology II

Course credit load: 2 Units

Series 4: The Cranial Nerves, Autonomic Neuropathy, and Coma

- **Part 4.1: The Cranial Nerves I**
    - Sub Part 4.1.1: Examination of the olfactory, optic, and oculomotor nerves
    - Sub Part 4.1.2: Examination of the trigeminal and facial nerves
    - Sub Part 4.1.3: Common cranial nerve palsies of nerves I to VII
- **Part 4.2: The Cranial Nerves II**
    - Sub Part 4.2.1: Examination of the vestibulocochlear, glossopharyngeal, and vagus nerves
    - Sub Part 4.2.2: Examination of the accessory and hypoglossal nerves
    - Sub Part 4.2.3: Bulbar and pseudobulbar palsy
- **Part 4.3: Autonomic Neuropathy**
    - Sub Part 4.3.1: Anatomy and physiology of the autonomic nervous system
    - Sub Part 4.3.2: Clinical presentation of autonomic neuropathy
    - Sub Part 4.3.3: Causes and management of autonomic neuropathy
- **Part 4.4: Coma**
    - Sub Part 4.4.1: Causes of coma
    - Sub Part 4.4.2: Assessment of the comatose patient
    - Sub Part 4.4.3: Management of coma

Introduction

This Series works through the twelve **cranial nerves**, the direct connections between the brain and the head and neck, before turning to the **autonomic nervous system**, operating largely outside conscious control, and closes with **coma**, the most profound disturbance of consciousness a clinician will encounter. Each topic demands the same disciplined, systematic examination technique that has run through this entire course, since a missed or misinterpreted finding in any of these three areas can genuinely change the whole direction of a diagnosis.

The aim of this Series is to equip you with the ability to examine cranial nerves I through XII and describe their common palsies, distinguish bulbar from pseudobulbar palsy, describe the anatomy, physiology, presentation, causes, and management of autonomic neuropathy, and describe the causes, assessment, and management of coma.

This Series opens with **the cranial nerves I**, moves through **the cranial nerves II** and **autonomic neuropathy**, and closes with **coma**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 4.1:** describe the examination of cranial nerves I to VII
- **SLO 4.2:** describe common cranial nerve palsies affecting nerves I to VII
- **SLO 4.3:** describe the examination of cranial nerves VIII to XII
- **SLO 4.4:** discuss bulbar and pseudobulbar palsy
- **SLO 4.5:** describe the anatomy, physiology, and clinical presentation of autonomic neuropathy
- **SLO 4.6:** discuss the causes and management of autonomic neuropathy
- **SLO 4.7:** describe the causes and assessment of coma
- **SLO 4.8:** discuss the management of coma

4.1 The Cranial Nerves I

4.1.1 examination of the olfactory, optic, and oculomotor nerves

Smell, sight, and the movement of the eye

The **olfactory nerve**, tested with a familiar, non-irritant smell in each nostril separately, is frequently overlooked in routine examination despite its genuine diagnostic value, particularly in head injury and certain neurodegenerative conditions, while the **optic nerve** is assessed through

visual acuity, visual fields, pupillary light reflex, and direct fundoscopic examination, a comprehensive assessment reflecting the nerve's genuinely complex visual pathway.

The **oculomotor**, **trochlear**, and **abducens nerves**, controlling eye movement, are assessed together by examining the range of eye movement in all directions and pupillary response, since these three nerves work in close coordination, and a specific pattern of restricted eye movement combined with pupillary findings provides genuinely valuable localising information about which specific nerve, or combination of nerves, is affected.

Fill in the blank: The olfactory nerve is tested with a familiar, non-irritant smell in each _____ separately.

4.1.2 examination of the trigeminal and facial nerves

Facial sensation and facial movement, examined together and apart

The **trigeminal nerve** is assessed through facial sensation across its three divisions, ophthalmic, maxillary, and mandibular, alongside motor testing of the muscles of mastication and the corneal reflex, a genuinely useful additional test since an absent corneal reflex can indicate trigeminal or facial nerve dysfunction, or a brainstem lesion affecting the reflex arc connecting the two nerves.

The **facial nerve** is assessed through observation of facial symmetry at rest and during voluntary movement, including raising the eyebrows, closing the eyes tightly, and smiling, and the specific pattern of weakness observed carries genuine localising value, since **upper motor neuron facial weakness** spares the forehead given its bilateral cortical innervation, while **lower motor neuron facial weakness** affects the entire side of the face including the forehead, a distinction genuinely important to establish confidently on examination.

Fill in the blank: Upper motor neuron facial weakness spares the forehead given its bilateral cortical _____.

4.1.3 common cranial nerve palsies of nerves i to vii

Recognisable patterns from damage to individual nerves

Third nerve palsy, producing ptosis, a dilated, poorly reactive pupil, and an eye deviated down and out, carries particular significance when the pupil is involved, since this pattern suggests external compression of the nerve, most classically from a posterior communicating artery aneurysm already discussed in an earlier course, while a pupil-sparing third nerve palsy more typically suggests an ischaemic cause, a genuinely important distinction with direct urgency implications.

Bell's palsy, an idiopathic, isolated lower motor neuron facial nerve palsy, is the most common cause of acute facial weakness, and correctly distinguishing it from an upper motor neuron facial weakness, which spares the forehead and typically points towards a central cause such as stroke, is a genuinely essential bedside skill given the very different urgency and management pathway each diagnosis demands.

Fill in the blank: A pupil-involving third nerve palsy suggests external _____ of the nerve, requiring urgent evaluation.

Figure 4.1: A clinician assessing eye movements during cranial nerve examination.

Pilot questions 4.1

- Describe the examination of the olfactory nerve. (Linked to SLO 4.1)
- List the components of oculomotor, trochlear, and abducens nerve examination. (Linked to SLO 4.1)
- Distinguish upper motor neuron from lower motor neuron facial weakness. (Linked to SLO 4.1)
- Describe third nerve palsy and the significance of pupil involvement. (Linked to SLO 4.2)
- Describe Bell's palsy and why distinguishing it from a central cause matters. (Linked to SLO 4.2)

4.2 The Cranial Nerves II

4.2.1 examination of the vestibulocochlear, glossopharyngeal, and vagus nerves

Hearing, balance, and the muscles of swallowing and voice

The **vestibulocochlear nerve** is assessed through hearing testing, including simple bedside tests distinguishing conductive from sensorineural hearing loss, and, where relevant, vestibular function testing given the nerve's role in balance alongside hearing, while the **glossopharyngeal** and **vagus nerves**, assessed together given their close functional relationship, are examined through palatal movement, the gag reflex, and voice quality.

Asymmetrical palatal elevation on phonation, with the uvula deviating towards the stronger, unaffected side, is a genuinely useful sign of unilateral glossopharyngeal or vagus nerve dysfunction, and, since these two nerves are examined together clinically given the practical difficulty of testing each in isolation, a combined deficit affecting both is considerably more commonly encountered in clinical practice than an isolated lesion of either nerve alone.

Fill in the blank: On phonation, the uvula deviates towards the stronger, _____ side in unilateral nerve dysfunction.

4.2.2 examination of the accessory and hypoglossal nerves

Shoulder shrug and tongue movement

The **accessory nerve** is assessed through testing shoulder shrug against resistance, evaluating trapezius muscle strength, and head rotation against resistance, evaluating sternocleidomastoid muscle strength, with weakness of either muscle indicating dysfunction of this specific nerve, which supplies both muscles despite their somewhat distant anatomical locations.

The **hypoglossal nerve** is assessed by observing the tongue at rest for wasting or fasciculation, and during protrusion, since a unilateral hypoglossal nerve lesion causes the protruded tongue to deviate towards the weaker, affected side, a specific and genuinely reliable finding reflecting the unopposed action of the stronger, unaffected side's tongue musculature pushing the tongue tip towards the weaker side during protrusion.

Fill in the blank: A unilateral hypoglossal nerve lesion causes the protruded tongue to deviate towards the weaker, affected _____.

4.2.3 bulbar and pseudobulbar palsy

Two genuinely distinct causes of swallowing and speech difficulty

Bulbar palsy, resulting from a lower motor neuron lesion affecting the cranial nerve nuclei or nerves themselves in the medulla, presents with a flaccid, wasted tongue with fasciculation, a quiet, nasal voice, and an absent gag reflex, while **pseudobulbar palsy**, resulting from bilateral upper motor neuron lesions affecting the corticobulbar tracts, presents with a spastic, contracted tongue without wasting, a strained, strangled voice, and an exaggerated, brisk gag reflex.

Correctly distinguishing bulbar from pseudobulbar palsy carries genuine clinical significance, since the two conditions point towards entirely different underlying pathology, bulbar palsy suggesting a lower brainstem or peripheral nerve process, and pseudobulbar palsy suggesting bilateral upper motor neuron pathology such as bilateral strokes or motor neuron disease, making this distinction genuinely essential when a patient presents with dysphagia and dysarthria.

Fill in the blank: Bulbar palsy presents with a flaccid, wasted tongue with _____, unlike the spastic tongue of pseudobulbar palsy.

Table 4.1: Comparing bulbar and pseudobulbar palsy

Feature	Bulbar palsy	Pseudobulbar palsy
Tongue appearance	Wasted, fasciculating	Spastic, no wasting
Voice character	Quiet, nasal	Strained, strangled
Gag reflex	Absent	Exaggerated

Pilot questions 4.2

1. Describe the examination of the glossopharyngeal and vagus nerves. (Linked to SLO 4.3)
2. Describe the examination of the accessory nerve. (Linked to SLO 4.3)
3. Describe the finding on tongue protrusion in a unilateral hypoglossal nerve lesion. (Linked to SLO 4.3)
4. Distinguish bulbar from pseudobulbar palsy by tongue appearance and gag reflex. (Linked to SLO 4.4)
5. Explain why distinguishing bulbar from pseudobulbar palsy carries genuine clinical significance. (Linked to SLO 4.4)

4.3 Autonomic Neuropathy

4.3.1 anatomy and physiology of the autonomic nervous system

The involuntary system governing internal organ function

The **autonomic nervous system**, comprising the **sympathetic** and **parasympathetic** divisions, regulates involuntary functions including heart rate, blood pressure, digestion, and bladder function, with the two divisions typically exerting opposing, balanced effects on a given target organ, a physiological arrangement allowing fine, continuous regulation of internal organ function without requiring conscious control.

Autonomic neuropathy, damage to the peripheral autonomic nerve fibres carrying these signals, disrupts this normal regulatory balance, and, since autonomic fibres are frequently affected alongside sensory and motor fibres in many of the peripheral neuropathies already covered in an earlier Part of this course, autonomic involvement should be actively considered in any patient with an established peripheral neuropathy rather than assumed absent simply because it was not the presenting complaint.

Fill in the blank: The sympathetic and parasympathetic divisions typically exert opposing, _____ effects on a given target organ.

4.3.2 clinical presentation of autonomic neuropathy

A genuinely wide-ranging pattern reflecting multiple affected organ systems

Cardiovascular autonomic neuropathy presents with **postural hypotension**, a significant fall in blood pressure on standing, producing dizziness or syncope, and a fixed heart rate that fails to respond appropriately to exertion or postural change, while **gastrointestinal autonomic neuropathy** presents with gastroparesis, producing early satiety, bloating, and vomiting, or, alternatively, diarrhoea or constipation depending on which specific gut function is predominantly affected.

Genitourinary autonomic neuropathy presents with bladder dysfunction, including incomplete emptying and recurrent urinary tract infection, and erectile dysfunction in men, and **sudomotor dysfunction**, affecting sweating regulation, can present with either reduced sweating, risking heat intolerance, or, less commonly, excessive sweating, together illustrating the genuinely wide-ranging, multi-system clinical impact autonomic neuropathy can produce.

Fill in the blank: Cardiovascular autonomic neuropathy produces a fixed heart rate that fails to respond appropriately to exertion or postural _____.

4.3.3 causes and management of autonomic neuropathy

A wide differential, with symptom-directed management

Diabetes mellitus remains the most common cause of autonomic neuropathy overall, alongside other recognised causes including amyloidosis, certain autoimmune conditions, and specific neurodegenerative disorders such as Parkinson's disease and multiple system atrophy, already discussed elsewhere in this course, meaning autonomic neuropathy is genuinely rarely an isolated diagnosis in itself but rather a manifestation of a broader underlying systemic or neurological condition.

Management is generally symptom-directed given the frequent absence of a specific disease-modifying treatment for the underlying autonomic nerve damage itself, including compression stockings and medication for postural hypotension, dietary modification and prokinetic medication for gastroparesis, and bladder management strategies for genitourinary dysfunction, a genuinely comprehensive, multidisciplinary approach reflecting the wide-ranging nature of the underlying condition.

Fill in the blank: Management of autonomic neuropathy is generally symptom-directed given the frequent absence of a specific _____ treatment.

Figure 4.2: A clinician assessing postural blood pressure change to test for autonomic dysfunction.

Pilot questions 4.3

1. Describe the sympathetic and parasympathetic divisions of the autonomic nervous system. (Linked to SLO 4.5)
2. Explain why autonomic involvement should be actively considered in peripheral neuropathy. (Linked to SLO 4.5)
3. Describe postural hypotension as a feature of cardiovascular autonomic neuropathy. (Linked to SLO 4.5)
4. List causes of autonomic neuropathy. (Linked to SLO 4.6)
5. Describe the general management approach to autonomic neuropathy. (Linked to SLO 4.6)

4.4 Coma

4.4.1 causes of coma

A genuinely wide differential requiring systematic exclusion

Coma, a state of profound unresponsiveness with no meaningful response to external stimuli, results from either **structural** causes, disrupting the reticular activating system directly through a mass lesion, haemorrhage, or significant brainstem injury, or **metabolic and toxic** causes, including hypoglycaemia, significant electrolyte disturbance, drug overdose, and hepatic or renal failure, each producing a diffuse disturbance of brain function without a discrete structural lesion.

Systematically working through this broad differential, rather than fixating prematurely on a single presumed cause, is genuinely essential, since several causes of coma are rapidly, completely reversible with prompt treatment, hypoglycaemia and opioid overdose among the most immediately treatable examples, making a structured, comprehensive approach to identifying the cause a genuine priority equal to the immediate supportive management of the comatose patient.

Fill in the blank: Several causes of coma, including hypoglycaemia and opioid overdose, are rapidly, completely _____ with prompt treatment.

4.4.2 assessment of the comatose patient

A structured examination despite the patient's inability to cooperate

Assessment of the comatose patient follows the same airway, breathing, circulation priorities already emphasised throughout this course, alongside the **Glasgow Coma Scale**, already introduced in an earlier course, to document conscious level objectively, and specific neurological examination adapted for the unresponsive patient, including pupillary response, eye movements, and motor response to painful stimulus, since standard cooperative testing is clearly not possible in this specific clinical scenario.

Brainstem reflexes, including pupillary light reflex, corneal reflex, and the oculocephalic reflex, testing eye movement in response to passive head turning, provide genuinely valuable information about brainstem function and help localise the level of dysfunction, and a structured, systematic approach to this specific examination, rather than an unstructured, incomplete assessment, is essential given the genuinely high clinical stakes involved in accurately assessing a comatose patient.

Fill in the blank: Brainstem reflexes provide genuinely valuable information about brainstem function and help _____ the level of dysfunction.

4.4.3 management of coma

Stabilise first, then work systematically towards the cause

Immediate management of the comatose patient prioritises airway protection, given the loss of normal protective airway reflexes at this level of unresponsiveness, alongside urgent correction of any immediately identifiable and reversible cause, such as administering glucose for suspected hypoglycaemia or naloxone for suspected opioid overdose, interventions that can be given empirically even before definitive confirmation given their low risk and potentially significant benefit.

Once immediately reversible causes have been addressed or excluded, further management proceeds according to the specific underlying cause identified through the systematic assessment already described, and ongoing supportive care, including careful attention to nutrition, pressure area care, and prevention of secondary complications, remains genuinely essential throughout the often prolonged course of coma from whatever underlying cause, closing this Series, and the entire course, on the theme of structured, systematic clinical assessment that has run throughout this whole programme of neurological study.

Fill in the blank: Immediate management prioritises airway protection given the loss of normal protective airway _____ at this level of unresponsiveness.

Table 4.2: Comparing structural and metabolic causes of coma

Category	Example cause	Typical clue
Structural	Intracranial haemorrhage	Focal neurological signs, asymmetry
Metabolic	Hypoglycaemia	Rapid reversal with correction
Toxic	Opioid overdose	Pinpoint pupils, response to naloxone

Pilot questions 4.4

1. Distinguish structural from metabolic and toxic causes of coma. (Linked to SLO 4.7)
2. List rapidly reversible causes of coma. (Linked to SLO 4.7)
3. Describe the components of assessing a comatose patient. (Linked to SLO 4.7)
4. Describe brainstem reflexes assessed in a comatose patient. (Linked to SLO 4.7)
5. Describe the immediate management priorities for a comatose patient. (Linked to SLO 4.8)

Series Summary

This Series covered the cranial nerves, autonomic neuropathy, and coma. Part 4.1 covered the examination and common palsies of cranial nerves I to VII, while Part 4.2 turned to cranial nerves VIII to XII and the distinction between bulbar and pseudobulbar palsy. Part 4.3 covered the anatomy, presentation, causes, and management of autonomic neuropathy, and Part 4.4 closed with the causes, assessment, and management of coma.

You should now be able to examine all twelve cranial nerves and describe their common palsies, describe autonomic neuropathy, and assess and manage the comatose patient across all four Parts of this Series, meeting the outcomes set for this Series. Series 5 turns next to CNS infections, demyelinating disease, and brain tumours.

Glossary of Terms

1. **Bell's palsy:** an idiopathic, isolated lower motor neuron facial nerve palsy.
2. **Bulbar palsy:** a lower motor neuron cause of dysarthria and dysphagia from medullary or nerve pathology.

3. **Coma:** a state of profound unresponsiveness with no meaningful response to external stimuli.
4. **Gastroparesis:** delayed gastric emptying, a feature of gastrointestinal autonomic neuropathy.
5. **Glasgow Coma Scale:** a standardised scale documenting conscious level objectively.
6. **Oculocephalic reflex:** a brainstem reflex testing eye movement in response to passive head turning.
7. **Postural hypotension:** a significant fall in blood pressure on standing, a feature of autonomic neuropathy.
8. **Pseudobulbar palsy:** a bilateral upper motor neuron cause of dysarthria and dysphagia.
9. **Reticular activating system:** the brainstem structure whose disruption produces coma.
10. **Sudomotor dysfunction:** abnormal sweating regulation from autonomic neuropathy.
11. **Third nerve palsy:** oculomotor nerve dysfunction producing ptosis and eye deviation, with or without pupil involvement.
12. **Upper motor neuron facial weakness:** facial weakness sparing the forehead given bilateral cortical innervation.

Concept Check Questions [Series 4]

Objective questions

- Upper motor neuron facial weakness spares the forehead given its bilateral cortical _____. (Linked to SLO 4.1)
- A unilateral hypoglossal nerve lesion causes the protruded tongue to deviate towards the weaker, affected _____. (Linked to SLO 4.3)
- Bulbar palsy presents with a flaccid, wasted tongue with _____, unlike the spastic tongue of pseudobulbar palsy. (Linked to SLO 4.4)
- The sympathetic and parasympathetic divisions typically exert opposing, _____ effects on a given target organ. (Linked to SLO 4.5)
- Several causes of coma, including hypoglycaemia and opioid overdose, are rapidly, completely _____ with prompt treatment. (Linked to SLO 4.7)

Short-answer questions

1. Describe third nerve palsy and the significance of pupil involvement. (Linked to SLO 4.2)
2. Distinguish bulbar from pseudobulbar palsy by tongue appearance and gag reflex. (Linked to SLO 4.4)

3. Describe postural hypotension as a feature of cardiovascular autonomic neuropathy. (Linked to SLO 4.5)
4. List causes of autonomic neuropathy. (Linked to SLO 4.6)
5. List rapidly reversible causes of coma. (Linked to SLO 4.7)

Long-answer questions

6. Discuss the examination and common palsies of cranial nerves I to VII. (Linked to SLO 4.1, 4.2)
7. Describe the examination of cranial nerves VIII to XII and distinguish bulbar from pseudobulbar palsy. (Linked to SLO 4.3, 4.4)
8. Discuss the anatomy, presentation, causes, and management of autonomic neuropathy. (Linked to SLO 4.5, 4.6)
9. Describe the causes and assessment of coma. (Linked to SLO 4.7)
10. Discuss the management of coma. (Linked to SLO 4.8)

Answers to Concept Check Questions [Series 4]

Objective questions

11. Innervation.
12. Side.
13. Fasciculation.
14. Balanced.
15. Reversible.

Short-answer questions

16. Third nerve palsy produces ptosis, a dilated pupil, and eye deviation; pupil involvement suggests external compression requiring urgent evaluation.
17. Bulbar palsy shows a wasted, fasciculating tongue with absent gag; pseudobulbar palsy shows a spastic tongue with exaggerated gag.
18. Postural hypotension is a significant blood pressure fall on standing, causing dizziness or syncope.
19. Causes include diabetes, amyloidosis, autoimmune conditions, and neurodegenerative disorders such as Parkinson's disease.
20. Rapidly reversible causes include hypoglycaemia and opioid overdose.

Long-answer questions

21. **Basic response:** Examination covers smell, vision, eye movement, facial sensation, and facial movement; common palsies include third nerve palsy and Bell's palsy, each with distinctive, distinguishable patterns.

    Value-added response: A value-added response notes that upper versus lower motor neuron facial weakness is distinguished by whether the forehead is spared.

22. **Basic response:** Examination covers hearing, palatal movement, gag reflex, shoulder shrug, and tongue protrusion; bulbar palsy shows lower motor neuron signs with a wasted tongue and absent gag, while pseudobulbar palsy shows upper motor neuron signs with a spastic tongue and exaggerated gag.

    Value-added response: A value-added response notes that this distinction points towards entirely different underlying pathology, such as motor neuron disease versus bilateral stroke.

23. **Basic response:** The autonomic nervous system comprises sympathetic and parasympathetic divisions; autonomic neuropathy presents with cardiovascular, gastrointestinal, genitourinary, and sudomotor symptoms; diabetes is the most common cause; management is generally symptom-directed.

    Value-added response: A value-added response notes that autonomic involvement should be actively considered in any patient with established peripheral neuropathy.

24. **Basic response:** Coma results from structural or metabolic and toxic causes; assessment follows airway, breathing, circulation priorities alongside the Glasgow Coma Scale and brainstem reflex testing.

    Value-added response: A value-added response notes that several causes of coma are rapidly, completely reversible, making a systematic differential essential.

25. **Basic response:** Management prioritises airway protection and empirical correction of immediately reversible causes such as hypoglycaemia or opioid overdose, followed by cause-directed treatment and ongoing supportive care.

    Value-added response: A value-added response notes that ongoing supportive care, including nutrition and pressure area care, remains essential throughout a prolonged coma course.

Answers to Pilot Questions [Series 4]

Part 4.1

1. The olfactory nerve is tested with a familiar, non-irritant smell presented to each nostril separately.
2. Examination includes assessing eye movement in all directions and pupillary response.
3. Upper motor neuron weakness spares the forehead, while lower motor neuron weakness affects the whole side including the forehead.
4. Third nerve palsy produces ptosis, a dilated pupil, and eye deviation; pupil involvement suggests external compression.
5. Bell's palsy is idiopathic lower motor neuron facial weakness; distinguishing it from central weakness matters given different urgency.

Part 4.2

6. Examination assesses palatal movement, gag reflex, and voice quality.
7. The accessory nerve is assessed through shoulder shrug and head rotation against resistance.
8. The tongue deviates towards the weaker, affected side on protrusion.
9. Bulbar palsy shows a wasted, fasciculating tongue with absent gag; pseudobulbar palsy shows a spastic tongue with exaggerated gag.
10. This distinction points towards entirely different underlying pathology requiring different management approaches.

Part 4.3

11. The sympathetic and parasympathetic divisions exert opposing, balanced effects on target organs.
12. Autonomic fibres are frequently affected alongside sensory and motor fibres in many peripheral neuropathies.
13. Postural hypotension is a significant blood pressure fall on standing, causing dizziness or syncope.
14. Causes include diabetes, amyloidosis, autoimmune conditions, and neurodegenerative disorders.
15. Management is generally symptom-directed given the frequent absence of a specific disease-modifying treatment.

Part 4.4

1. Structural causes disrupt the reticular activating system directly, while metabolic and toxic causes produce diffuse dysfunction.

2. Rapidly reversible causes include hypoglycaemia and opioid overdose.
3. Assessment follows airway, breathing, circulation priorities alongside the Glasgow Coma Scale and neurological examination.
4. Brainstem reflexes include pupillary light reflex, corneal reflex, and the oculocephalic reflex.
5. Immediate priorities include airway protection and empirical correction of immediately reversible causes.

References and Attributions [Series 4]

1. Ropper, A. H., Samuels, M. A., & Klein, J. P. (2019). Adams and Victor's Principles of Neurology (11th ed.). McGraw-Hill.
2. Douglas, G., Nicol, F., & Robertson, C. (2013). Macleod's Clinical Examination (13th ed.). Churchill Livingstone.
3. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
4. Plum, F., & Posner, J. B. (2007). Plum and Posner's Diagnosis of Stupor and Coma (4th ed.). Oxford University Press.

Figures, Plates, Tables, and Boxes

1. Figure 4.1: A clinician assessing eye movements during cranial nerve examination. AI-generated using the prompt: "Create a realistic clinical illustration titled Eye Movement Examination, showing a clinician holding a small target and asking a seated patient to follow it with their eyes across different directions of gaze, consultation room setting. Clean clinical illustration style, no text."
2. Table 4.1: Comparing bulbar and pseudobulbar palsy. AI-generated using the prompt: "Create a clean reference table titled Comparing Bulbar and Pseudobulbar Palsy, listing feature, bulbar palsy, and pseudobulbar palsy. Simple clinical reference table style with outside border."
3. Figure 4.2: A clinician assessing postural blood pressure change to test for autonomic dysfunction. AI-generated using the prompt: "Create a realistic clinical illustration titled Postural Blood Pressure Assessment, showing a clinician measuring a patient's blood pressure with a cuff while the patient stands, examination room setting. Clean clinical illustration style, no text."

4. Table 4.2: Comparing structural and metabolic causes of coma. AI-generated using the prompt: "Create a clean reference table titled Comparing Structural and Metabolic Causes of Coma, listing category, example cause, and typical clue. Simple clinical reference table style with outside border."

Course code: MED 604

Course title: Neurology II

Course credit load: 2 Units

Series 5: CNS Infections, Demyelinating Disease, and Brain Tumours

- **Part 5.1: CNS Infections I: Meningitis**
  - Sub Part 5.1.1: Bacterial meningitis: pathogens and presentation
  - Sub Part 5.1.2: Tuberculous meningitis
  - Sub Part 5.1.3: Investigation and management of meningitis
- **Part 5.2: CNS Infections II: Viral Infections and Abscess**
  - Sub Part 5.2.1: Viral encephalitis
  - Sub Part 5.2.2: Other viral CNS infections
  - Sub Part 5.2.3: Cerebral abscess
- **Part 5.3: Demyelinating Disease and Subacute Combined Degeneration**
  - Sub Part 5.3.1: Multiple sclerosis: pathophysiology and presentation
  - Sub Part 5.3.2: Diagnosis and management of multiple sclerosis
  - Sub Part 5.3.3: Subacute combined degeneration of the cord
- **Part 5.4: Cord Compression and Brain Tumours**
  - Sub Part 5.4.1: Spinal cord compression: causes and presentation
  - Sub Part 5.4.2: Management of spinal cord compression
  - Sub Part 5.4.3: Brain tumours

Introduction

This final Series closes the course with three genuinely urgent categories of neurological disease, **infections of the central nervous system**, where delayed recognition can prove rapidly fatal, **demyelinating disease and subacute combined degeneration**, both disrupting the normal insulation and metabolic support of nerve fibres, and **cord compression and brain tumours**, space-occupying processes demanding prompt recognition to prevent permanent neurological injury.

The aim of this Series is to equip you with the ability to describe bacterial and tuberculous meningitis and their investigation and management, describe viral encephalitis, other viral CNS infections, and cerebral abscess, describe the pathophysiology, presentation, diagnosis, and management of multiple sclerosis and subacute combined degeneration, and describe the causes, presentation, and management of spinal cord compression and brain tumours.

This Series opens with **CNS infections I: meningitis**, moves through **CNS infections II: viral infections and abscess** and **demyelinating disease and subacute combined degeneration**, and closes with **cord compression and brain tumours**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 5.1:** describe the pathogens and presentation of bacterial meningitis
- **SLO 5.2:** describe tuberculous meningitis
- **SLO 5.3:** discuss the investigation and management of meningitis
- **SLO 5.4:** describe viral encephalitis and other viral CNS infections
- **SLO 5.5:** describe cerebral abscess
- **SLO 5.6:** describe the pathophysiology, presentation, diagnosis, and management of multiple sclerosis and subacute combined degeneration
- **SLO 5.7:** describe the causes, presentation, and management of spinal cord compression
- **SLO 5.8:** describe brain tumours

5.1 CNS Infections I: Meningitis

5.1.1 bacterial meningitis: pathogens and presentation

A genuine neurological emergency requiring immediate recognition

Bacterial meningitis, infection and inflammation of the meninges, most commonly results from **Neisseria meningitidis**, cerebrospinal meningitis given its epidemic potential across certain regions, **Streptococcus pneumoniae**, and, particularly in unvaccinated populations, **Haemophilus influenzae**, each producing a broadly similar clinical picture though with somewhat differing epidemiology, severity, and specific complication patterns.

Classic presentation includes fever, severe headache, neck stiffness, and photophobia, alongside, in meningococcal disease specifically, a characteristic non-blanching petechial or purpuric rash reflecting the associated septicaemia that frequently accompanies this particular organism, and, given the genuinely rapid clinical deterioration bacterial meningitis can produce, prompt recognition and immediate treatment, covered further in the third sub-Part of this Part, is a genuine clinical priority overriding almost any other consideration.

Fill in the blank: A characteristic non-blanching petechial or purpuric rash reflects the septicaemia accompanying meningococcal _____.

5.1.2 tuberculous meningitis

A more insidious, but equally serious, infectious presentation

Tuberculous meningitis, resulting from Mycobacterium tuberculosis infection of the meninges, typically presents more insidiously than acute bacterial meningitis, with symptoms developing gradually over days to weeks, including low-grade fever, headache, and personality change, before progressing to more overt meningitic features, a pattern that can genuinely delay diagnosis given its less immediately dramatic initial presentation compared with acute bacterial meningitis.

Diagnosis is genuinely challenging, since cerebrospinal fluid microscopy and culture for tuberculosis carry relatively low sensitivity and culture results take considerable time to become available, meaning treatment is frequently initiated empirically based on clinical suspicion and supportive cerebrospinal fluid findings rather than awaiting definitive microbiological confirmation, given the genuine risk of significant, potentially permanent neurological damage from delayed treatment of this specific, more indolent infection.

Fill in the blank: Tuberculous meningitis treatment is frequently initiated empirically given the genuine risk of delayed treatment causing permanent neurological _____.

5.1.3 investigation and management of meningitis

Confirming the diagnosis while never delaying treatment

Lumbar puncture, obtaining cerebrospinal fluid for microscopy, culture, and biochemical analysis, is the key investigation confirming meningitis and helping distinguish bacterial, tuberculous, and viral causes based on the specific pattern of white cell count, protein, and glucose obtained, though it should be deferred in any patient with signs suggesting significantly raised intracranial pressure given the genuine risk of causing brainstem herniation in this specific clinical scenario.

Empirical antibiotic therapy for suspected bacterial meningitis should be administered immediately once the diagnosis is suspected, genuinely without waiting for lumbar puncture or other confirmatory investigation if this would meaningfully delay treatment, since every hour of delay in appropriate antibiotic treatment for bacterial meningitis measurably worsens outcome, making this one of the clearest examples in the whole of medicine where treatment genuinely should never wait for complete diagnostic confirmation.

Fill in the blank: Empirical antibiotic therapy for suspected bacterial meningitis should never meaningfully _____ for confirmatory investigation.

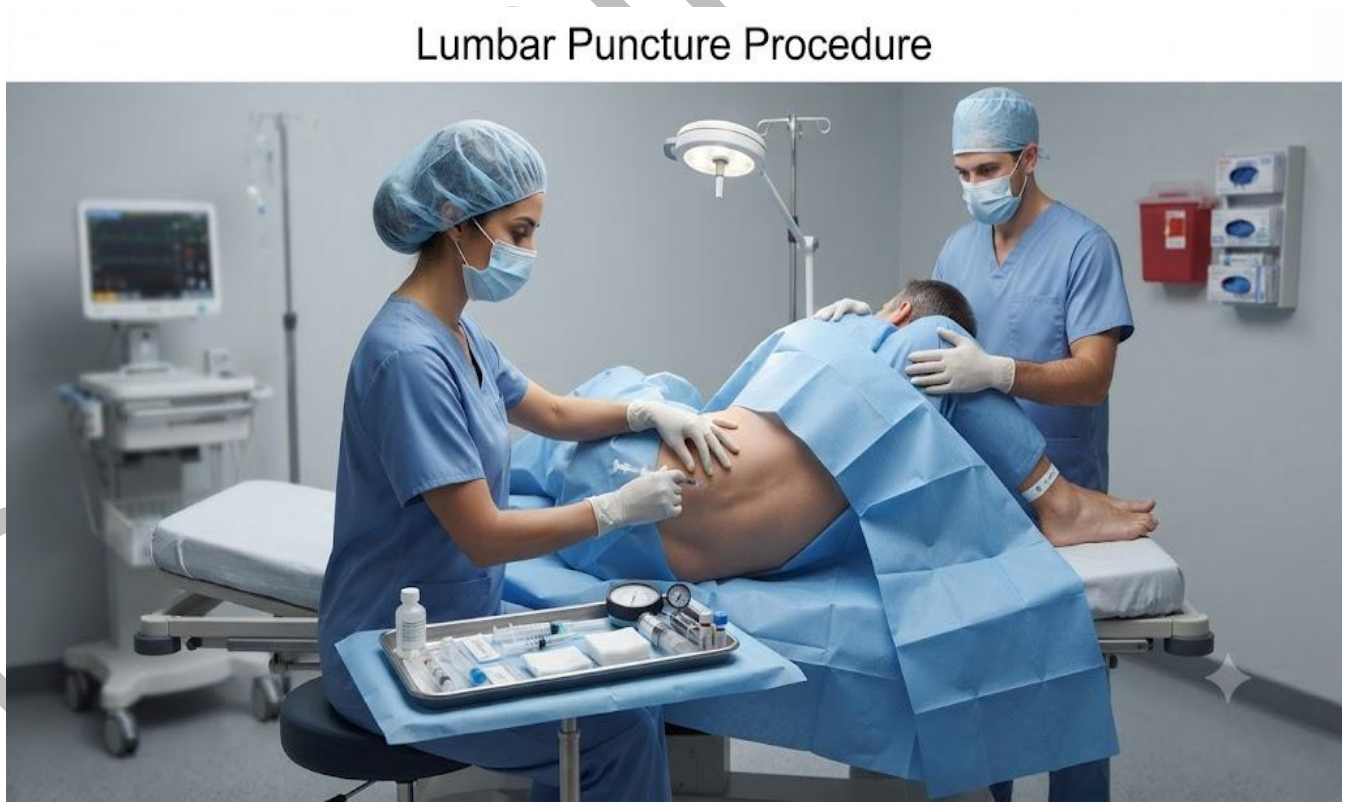

Figure 5.1: A clinician performing a lumbar puncture to obtain cerebrospinal fluid.

Pilot questions 5.1

- List the common pathogens causing bacterial meningitis. (Linked to SLO 5.1)
- Describe the classic presentation of bacterial meningitis. (Linked to SLO 5.1)
- Describe the typical presentation of tuberculous meningitis. (Linked to SLO 5.2)
- Explain why treatment of tuberculous meningitis is often started empirically. (Linked to SLO 5.2)
- Explain why antibiotic treatment for suspected bacterial meningitis should not wait for confirmatory investigation. (Linked to SLO 5.3)

5.2 CNS Infections II: Viral Infections and Abscess

5.2.1 viral encephalitis

Infection and inflammation of the brain parenchyma itself

Viral encephalitis, infection and inflammation of the brain parenchyma itself rather than the meninges alone, presents with fever, headache, altered consciousness, seizures, and focal neurological deficit, a pattern that distinguishes it clinically from meningitis, where consciousness is typically preserved unless the disease has progressed to a genuinely severe stage, though the two conditions can genuinely coexist as meningoencephalitis in a proportion of cases.

Herpes simplex virus is the most common identifiable cause of sporadic viral encephalitis and characteristically affects the temporal lobes, and, given its genuinely significant associated morbidity and mortality if untreated, empirical antiviral treatment with aciclovir should be initiated promptly in any patient with suspected encephalitis while awaiting confirmatory investigation, mirroring the same principle of not delaying treatment for confirmation already emphasised for bacterial meningitis in the previous Part.

Fill in the blank: Herpes simplex virus encephalitis characteristically affects the _____ lobes.

5.2.2 other viral cns infections

A genuinely wide range of viral pathogens and presentations

Beyond herpes simplex virus, a genuinely wide range of other viruses can cause central nervous system infection, including enteroviruses, most commonly causing a comparatively milder viral meningitis rather than encephalitis, and, in specific regional or epidemiological contexts, arboviruses transmitted by mosquito vectors, each carrying somewhat differing epidemiology, severity, and specific investigation approach relevant to the local clinical setting.

Polymerase chain reaction testing of cerebrospinal fluid has genuinely transformed the diagnostic approach to viral central nervous system infection, allowing rapid, sensitive identification of the specific causative virus in many cases, and this diagnostic advance, combined with the availability of specific antiviral treatment for certain viral causes, particularly herpes simplex virus, underlines why accurate identification of the specific causative organism genuinely matters clinically rather than simply confirming a viral cause in general terms alone.

Fill in the blank: Polymerase chain reaction testing of cerebrospinal fluid allows rapid, sensitive identification of the specific causative _____.

5.2.3 cerebral abscess

A focal collection requiring drainage and antimicrobial therapy

Cerebral abscess, already discussed in an earlier course of this programme in the context of neurosurgery, presents with headache, fever, and focal neurological deficit, though the classical triad is present together in only a minority of cases, and can arise from direct spread from adjacent infected structures, haematogenous spread from a distant infection, or, less commonly, penetrating trauma or neurosurgical procedures.

Management combines surgical drainage of any significant collection with prolonged, targeted antimicrobial therapy guided by culture of aspirated material where possible, and, given the genuine overlap between neurology and neurosurgery in managing this specific condition, close collaboration between the two specialties is genuinely essential to achieving a good outcome, a theme worth remembering as this Series draws this programme's coverage of central nervous system infection to a close.

Fill in the blank: Management of cerebral abscess combines surgical drainage with prolonged, targeted antimicrobial _____.

Table 5.1: Comparing meningitis, encephalitis, and cerebral abscess

Condition	Primary site affected	Consciousness typically
Meningitis	Meninges	Preserved unless severe
Encephalitis	Brain parenchyma	Frequently altered
Cerebral abscess	Focal brain lesion	Depends on lesion size and location

Pilot questions 5.2

1. Distinguish viral encephalitis from meningitis by typical presentation. (Linked to SLO 5.4)
2. Explain why empirical aciclovir is started promptly in suspected encephalitis. (Linked to SLO 5.4)
3. Describe the role of polymerase chain reaction testing in viral CNS infection. (Linked to SLO 5.4)
4. List the routes by which a cerebral abscess can develop. (Linked to SLO 5.5)
5. Describe the management approach to cerebral abscess. (Linked to SLO 5.5)

5.3 Demyelinating Disease and Subacute Combined Degeneration

5.3.1 multiple sclerosis: pathophysiology and presentation

An autoimmune process disrupting the nervous system's insulation

Multiple sclerosis, an autoimmune, inflammatory demyelinating disease of the central nervous system, results from immune-mediated damage to the myelin sheath insulating nerve fibres, producing lesions, termed **plaques**, that can occur anywhere within the central nervous system, and, given this genuinely wide potential distribution, multiple sclerosis can present with a genuinely diverse range of neurological symptoms depending on the specific location of the affected plaques.

Common presentations include optic neuritis, producing painful visual loss, sensory disturbance, limb weakness, and cerebellar dysfunction producing incoordination, and the classic disease course, **relapsing-remitting multiple sclerosis**, involves discrete episodes of new or worsening neurological symptoms separated by periods of partial or complete recovery, a pattern that,

alongside the specific criterion of lesions occurring at different times and in different locations within the central nervous system, forms the essential basis of diagnosis covered in the following sub-Part.

Fill in the blank: Multiple sclerosis produces lesions, termed _____, that can occur anywhere within the central nervous system.

5.3.2 diagnosis and management of multiple sclerosis

Confirming dissemination in time and space

Diagnosis of multiple sclerosis relies on demonstrating **dissemination in time and space**, meaning evidence of at least two separate neurological episodes affecting different areas of the central nervous system, supported by **magnetic resonance imaging**, identifying characteristic demyelinating lesions, and, where the clinical picture remains genuinely uncertain, cerebrospinal fluid analysis for **oligoclonal bands**, a specific finding reflecting intrathecal immune activity supporting the diagnosis.

Management includes **acute relapse treatment** with corticosteroids to hasten recovery from a significant relapse, and **disease-modifying therapy**, a genuinely expanding range of medications reducing relapse frequency and slowing long-term disability accumulation in appropriately selected patients, alongside symptomatic management of specific symptoms such as spasticity and fatigue, reflecting the genuinely comprehensive, long-term approach multiple sclerosis management requires.

Fill in the blank: Diagnosis of multiple sclerosis relies on demonstrating dissemination in time and _____.

5.3.3 subacute combined degeneration of the cord

A genuinely treatable cause of combined spinal cord dysfunction

Subacute combined degeneration, resulting from vitamin B12 deficiency, produces combined dysfunction of the dorsal columns, causing loss of vibration and proprioception, and the corticospinal tracts, causing spastic weakness, alongside, frequently, a coexisting peripheral neuropathy producing additional distal sensory symptoms, a genuinely distinctive combination of upper and lower motor neuron and dorsal column findings worth recognising confidently.

Prompt recognition and treatment with **vitamin B12 replacement** is genuinely essential, since this is one of the relatively few causes of significant spinal cord dysfunction that is genuinely, completely reversible if treated sufficiently early, though established, longstanding neurological

deficit may not fully recover even with appropriate treatment, underlining why active consideration of this diagnosis in any patient presenting with a combined dorsal column and corticospinal pattern of deficit genuinely matters.

Fill in the blank: Subacute combined degeneration results from vitamin _____ deficiency.

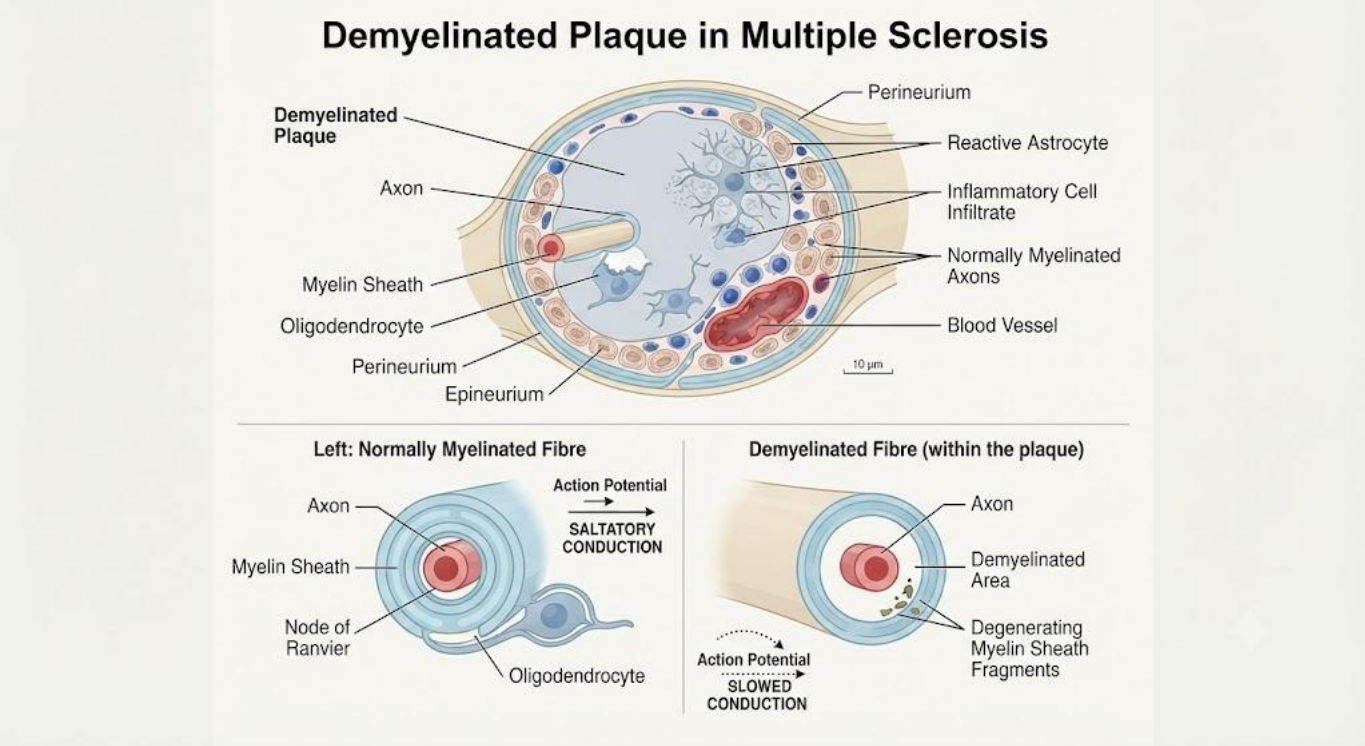

Figure 5.2: A labelled diagram of a demyelinated plaque within cerebral white matter.

Pilot questions 5.3

1. Describe the pathophysiology of multiple sclerosis. (Linked to SLO 5.6)
2. Describe relapsing-remitting multiple sclerosis. (Linked to SLO 5.6)
3. Describe how multiple sclerosis is diagnosed. (Linked to SLO 5.6)
4. Describe subacute combined degeneration and its underlying cause. (Linked to SLO 5.6)
5. Explain why prompt treatment of subacute combined degeneration genuinely matters. (Linked to SLO 5.6)

5.4 Cord Compression and Brain Tumours

5.4.1 spinal cord compression: causes and presentation

A genuine neurological emergency demanding rapid recognition

Spinal cord compression, external pressure on the spinal cord from a mass lesion, most commonly results from metastatic malignancy, already discussed in an earlier course of this programme, though it can also result from disc disease, infection, or haematoma, and presents with progressive back pain, weakness, sensory disturbance, and, in more advanced cases, bladder and bowel dysfunction, a presentation with genuine overlap with the myelopathy pattern already covered earlier in this Series.

Recognising spinal cord compression promptly is genuinely essential, since delayed treatment risks permanent, irreversible neurological deficit, and any patient presenting with progressive back pain accompanied by neurological symptoms should be actively and urgently evaluated for this specific diagnosis rather than being managed as simple mechanical back pain, given the genuinely narrow window during which treatment can still meaningfully prevent permanent disability.

Fill in the blank: Delayed treatment of spinal cord compression risks permanent, _____ neurological deficit.

5.4.2 management of spinal cord compression

Urgent imaging, then decompression or targeted treatment

Urgent magnetic resonance imaging of the whole spine, given the genuine possibility of multiple levels of involvement, confirms the diagnosis and characterises the specific level and extent of compression, and immediate management includes **corticosteroid therapy** to reduce cord oedema while definitive treatment is arranged, a genuinely important bridging intervention that can meaningfully preserve neurological function in the short term.

Definitive treatment, whether surgical decompression, radiotherapy, or a combination of both, depends on the specific underlying cause, the patient's overall prognosis, and the urgency of the clinical presentation, and coordinated, rapid multidisciplinary decision-making, drawing on oncology, neurosurgery, and radiotherapy input where malignancy is the underlying cause, is genuinely essential given how directly treatment timing affects the eventual functional outcome for the patient.

Fill in the blank: Corticosteroid therapy reduces cord oedema while definitive treatment for spinal cord compression is _____.

5.4.3 brain tumours

Closing the course with a familiar, genuinely important topic

Brain tumours, already discussed in detail in an earlier course of this programme covering neurosurgery, present with headache, seizures, focal neurological deficit, or symptoms of raised intracranial pressure, depending on tumour location, size, and growth rate, and, from a neurological rather than purely surgical perspective, the emphasis genuinely shifts towards recognising the presentation promptly and coordinating appropriate onward referral for definitive neurosurgical and oncological management.

Neurological involvement in brain tumour care extends beyond initial diagnosis to include management of tumour-related seizures with anticonvulsant medication, and ongoing surveillance for both tumour progression and treatment-related neurological complications, and this genuinely collaborative approach between neurology, neurosurgery, and oncology, closing this Series and the entire course, reflects the coordinated, multidisciplinary theme that has run consistently throughout this whole programme of neurological study.

Fill in the blank: Neurological involvement in brain tumour care includes management of tumour-related _____ with anticonvulsant medication.

Table 5.2: Common causes of spinal cord compression by category

Category	Example	Typical urgency
Malignant	Metastatic spinal disease	Urgent, time-critical
Degenerative	Large disc prolapse	Urgent if progressive deficit
Infective	Spinal epidural abscess	Urgent, genuine emergency

Pilot questions 5.4

1. List the causes of spinal cord compression. (Linked to SLO 5.7)
2. Describe the presentation of spinal cord compression. (Linked to SLO 5.7)
3. Describe the immediate management of suspected spinal cord compression. (Linked to SLO 5.7)
4. Describe the presentation of brain tumours from a neurological perspective. (Linked to SLO 5.8)
5. Describe the neurologist's ongoing role in brain tumour care. (Linked to SLO 5.8)

Series Summary

This final Series closed the course with CNS infections, demyelinating disease, and space-occupying neurological conditions. Part 5.1 covered bacterial and tuberculous meningitis and their investigation and management, while Part 5.2 turned to viral encephalitis, other viral CNS infections, and cerebral abscess. Part 5.3 covered multiple sclerosis and subacute combined degeneration, and Part 5.4 closed with spinal cord compression and brain tumours.

You should now be able to describe CNS infections, demyelinating disease, subacute combined degeneration, spinal cord compression, and brain tumours across all four Parts of this Series, meeting the outcomes set for this Series. Across the full course, Series 1 covered higher cerebral function, dementia, and stroke, Series 2 covered epilepsy, sleep disorders, Parkinson's disease, and headache, Series 3 covered spinal cord lesions, peripheral neuropathy, and neuromuscular diseases, Series 4 covered the cranial nerves, autonomic neuropathy, and coma, and this Series has closed the course with CNS infections, demyelinating disease, and brain tumours, together forming a comprehensive foundation in Neurology II.

Glossary of Terms

1. **Cerebral abscess:** a localised collection of pus within the brain parenchyma.
2. **Dissemination in time and space:** the diagnostic criterion for multiple sclerosis requiring separate episodes in different locations.
3. **Herpes simplex encephalitis:** the most common identifiable cause of sporadic viral encephalitis.
4. **Lumbar puncture:** a procedure obtaining cerebrospinal fluid for diagnostic analysis.
5. **Multiple sclerosis:** an autoimmune, inflammatory demyelinating disease of the central nervous system.
6. **Oligoclonal bands:** a cerebrospinal fluid finding reflecting intrathecal immune activity, supporting multiple sclerosis.
7. **Plaque:** a focal area of demyelination in multiple sclerosis.
8. **Relapsing-remitting multiple sclerosis:** the classic multiple sclerosis course of discrete relapses and recovery.
9. **Spinal cord compression:** external pressure on the spinal cord from a mass lesion, a genuine emergency.
10. **Subacute combined degeneration:** combined dorsal column and corticospinal tract dysfunction from vitamin B12 deficiency.
11. **Tuberculous meningitis:** meningitis from Mycobacterium tuberculosis, typically presenting insidiously.

12. **Viral encephalitis:** infection and inflammation of the brain parenchyma, most commonly from herpes simplex virus.

Concept Check Questions [Series 5]

Objective questions

- A characteristic non-blanching petechial or purpuric rash reflects the septicaemia accompanying meningococcal _____. (Linked to SLO 5.1)
- Herpes simplex virus encephalitis characteristically affects the _____ lobes. (Linked to SLO 5.4)
- Multiple sclerosis produces lesions, termed _____, that can occur anywhere within the central nervous system. (Linked to SLO 5.6)
- Subacute combined degeneration results from vitamin _____ deficiency. (Linked to SLO 5.6)
- Delayed treatment of spinal cord compression risks permanent, _____ neurological deficit. (Linked to SLO 5.7)

Short-answer questions

1. Describe the typical presentation of tuberculous meningitis. (Linked to SLO 5.2)
2. Distinguish viral encephalitis from meningitis by typical presentation. (Linked to SLO 5.4)
3. Describe relapsing-remitting multiple sclerosis. (Linked to SLO 5.6)
4. Describe subacute combined degeneration and its underlying cause. (Linked to SLO 5.6)
5. List the causes of spinal cord compression. (Linked to SLO 5.7)

Long-answer questions

6. Discuss bacterial and tuberculous meningitis and their investigation and management. (Linked to SLO 5.1, 5.2, 5.3)
7. Describe viral encephalitis, other viral CNS infections, and cerebral abscess. (Linked to SLO 5.4, 5.5)
8. Discuss the pathophysiology, presentation, diagnosis, and management of multiple sclerosis and subacute combined degeneration. (Linked to SLO 5.6)
9. Describe the causes, presentation, and management of spinal cord compression. (Linked to SLO 5.7)
10. Discuss brain tumours from a neurological perspective. (Linked to SLO 5.8)

Answers to Concept Check Questions [Series 5]

Objective questions

11. Disease.
12. Temporal.
13. Plaques.
14. B12.
15. Irreversible.

Short-answer questions

16. Tuberculous meningitis presents insidiously over days to weeks with low-grade fever, headache, and personality change.
17. Encephalitis presents with altered consciousness and focal deficit, while meningitis typically preserves consciousness unless severe.
18. Relapsing-remitting multiple sclerosis involves discrete episodes of new symptoms separated by periods of recovery.
19. Subacute combined degeneration is combined dorsal column and corticospinal dysfunction from vitamin B12 deficiency.
20. Causes include metastatic malignancy, disc disease, infection, and haematoma.

Long-answer questions

21. **Basic response:** Bacterial meningitis presents acutely with fever, headache, and neck stiffness, requiring immediate empirical antibiotics; tuberculous meningitis presents insidiously, often treated empirically given diagnostic difficulty; lumbar puncture confirms diagnosis but should not delay treatment.

    Value-added response: A value-added response notes that lumbar puncture should be deferred if signs suggest significantly raised intracranial pressure, given herniation risk.

22. **Basic response:** Viral encephalitis, most commonly from herpes simplex virus, presents with altered consciousness and focal deficit, treated empirically with aciclovir; other viruses cause milder meningitis; cerebral abscess presents with headache, fever, and deficit, managed with drainage and antibiotics.

    Value-added response: A value-added response notes that polymerase chain reaction testing has transformed rapid identification of the specific causative virus.

23. **Basic response:** Multiple sclerosis is autoimmune demyelination producing plaques anywhere in the CNS, diagnosed by dissemination in time and space; subacute combined

degeneration is vitamin B12 deficiency causing combined dorsal column and corticospinal dysfunction, treated with B12 replacement.

Value-added response: A value-added response notes that subacute combined degeneration is genuinely reversible if treated early, unlike many other causes of spinal cord dysfunction.

24. **Basic response:** Causes include metastatic malignancy, disc disease, infection, and haematoma; presentation includes progressive pain, weakness, and sensory disturbance; management involves urgent MRI, corticosteroids, and definitive surgical or radiotherapy treatment.

    Value-added response: A value-added response notes that coordinated multidisciplinary decision-making is essential given how directly treatment timing affects functional outcome.

25. **Basic response:** Brain tumours present with headache, seizures, focal deficit, or raised intracranial pressure symptoms; neurological involvement includes prompt recognition, seizure management, and ongoing surveillance alongside neurosurgical and oncological care.

    Value-added response: A value-added response notes that this collaborative, multidisciplinary approach reflects the coordinated care theme running throughout the whole course.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Common pathogens include Neisseria meningitidis, Streptococcus pneumoniae, and Haemophilus influenzae.
2. Classic presentation includes fever, severe headache, neck stiffness, and photophobia.
3. Tuberculous meningitis presents insidiously with low-grade fever, headache, and personality change over days to weeks.
4. Treatment is often empirical given low sensitivity and slow culture results, with genuine risk of delayed treatment.
5. Every hour of delay in antibiotic treatment for bacterial meningitis measurably worsens outcome.

Part 5.2

6. Encephalitis presents with altered consciousness and focal deficit, while meningitis typically preserves consciousness.
7. Aciclovir is started promptly given significant morbidity and mortality if herpes simplex encephalitis is untreated.
8. PCR testing allows rapid, sensitive identification of the specific causative virus.
9. Routes include direct spread, haematogenous spread, and penetrating trauma or neurosurgical procedures.
10. Management combines surgical drainage of any collection with prolonged, targeted antimicrobial therapy.

Part 5.3

11. Multiple sclerosis results from autoimmune, immune-mediated demyelination of the central nervous system.
12. Relapsing-remitting multiple sclerosis involves discrete relapses separated by periods of partial or complete recovery.
13. Diagnosis relies on demonstrating dissemination in time and space, supported by MRI and cerebrospinal fluid findings.
14. Subacute combined degeneration is combined dorsal column and corticospinal dysfunction from vitamin B12 deficiency.
15. It is one of the few genuinely reversible causes of spinal cord dysfunction if treated sufficiently early.

Part 5.4

1. Causes include metastatic malignancy, disc disease, infection, and haematoma.
2. Presentation includes progressive back pain, weakness, sensory disturbance, and bladder or bowel dysfunction.
3. Immediate management includes urgent MRI and corticosteroid therapy to reduce cord oedema.
4. Brain tumours present with headache, seizures, focal deficit, or symptoms of raised intracranial pressure.
5. The neurologist coordinates seizure management and ongoing surveillance alongside neurosurgical and oncological care.

References and Attributions [Series 5]

1. Ropper, A. H., Samuels, M. A., & Klein, J. P. (2019). Adams and Victor's Principles of Neurology (11th ed.). McGraw-Hill.
2. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
3. National Institute for Health and Care Excellence (2016). Meningitis (Bacterial) and Meningococcal Septicaemia in Under 16s (CG102). NICE.
4. Compston, A., & Coles, A. (2008). Multiple Sclerosis. The Lancet.

Figures, Plates, Tables, and Boxes

1. Figure 5.1: A clinician performing a lumbar puncture to obtain cerebrospinal fluid. AI-generated using the prompt: "Create a realistic clinical illustration titled Lumbar Puncture Procedure, showing a clinician performing a lumbar puncture on a patient positioned on their side with knees drawn up, sterile drapes and equipment visible, clinical procedure room setting. Clean clinical illustration style, no text."
2. Table 5.1: Comparing meningitis, encephalitis, and cerebral abscess. AI-generated using the prompt: "Create a clean reference table titled Comparing Meningitis, Encephalitis, and Cerebral Abscess, listing condition, primary site affected, and consciousness typically. Simple clinical reference table style with outside border."
3. Figure 5.2: A labelled diagram of a demyelinated plaque within cerebral white matter. AI-generated using the prompt: "Create a clean, accurate labelled anatomical diagram titled Demyelinated Plaque in Multiple Sclerosis, showing a cross-section of a nerve fibre bundle with a labelled area of myelin loss surrounding an axon compared with an adjacent normally myelinated fibre. Educational anatomical diagram style, no photographic content."
4. Table 5.2: Common causes of spinal cord compression by category. AI-generated using the prompt: "Create a clean reference table titled Common Causes of Spinal Cord Compression by Category, listing category, example, and typical urgency. Simple clinical reference table style with outside border."