



PHYSIOCITY

Multiple Sclerosis

Complete Study Notes for Competitive Physiotherapy Examinations

**Anatomy • Physiology • Pathology • Musculoskeletal & Neuromuscular Changes
PT Assessment • PT Intervention • Recent Research • Case Study**

With Topic-wise MCQs for AIIMS • CUET PG • MPT Entrance • PGIMER • RUHS

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MODULE

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MODULE 1

INTRODUCTION & EPIDEMIOLOGY

Definition • Burden of disease • Disease-course classification

Definition

Multiple sclerosis (MS) is a chronic, immune-mediated, inflammatory demyelinating and neurodegenerative disease of the central nervous system (CNS), characterized by multifocal areas of demyelination (“plaques”) that are disseminated in time and space. It is the leading cause of non-traumatic neurological disability in young adults.

Epidemiology

- Peak age of onset: 20–40 years.
- Female : Male ratio $\approx$ 2–3 : 1.
- Prevalence rises with increasing distance from the equator (latitude gradient).
- Global prevalence is rising — in Australia, the number of people living with MS increased by 13.3% between 2021 and 2024, with annual disease-related cost exceeding AU\$3 billion (Learmonth, *J Physiother* 2026).
- MS is the leading cause of non-traumatic neurological disability in young adults, particularly women.

Classification of Disease Course (Lublin Phenotypes)

Course	Key Feature
Clinically Isolated Syndrome (CIS)	First clinical episode suggestive of demyelination; may or may not evolve to MS
Relapsing-Remitting MS (RRMS)	Most common ($\approx$ 85%); discrete relapses with full/partial recovery, stable between attacks
Secondary Progressive MS (SPMS)	Follows an initial relapsing course; gradual worsening with or without relapses
Primary Progressive MS (PPMS)	Steady disability progression from onset, without distinct relapses ($\approx$ 10–15%)
Progressive-Relapsing MS (PRMS)	Rare; progressive disease from onset with occasional superimposed relapses

MODULE 1 — MCQs (Q1–Q5)

1 Multiple sclerosis is best defined as a disease that is:

- A. Peripheral, degenerative, and hereditary B. ✓ Central, immune-mediated, and demyelinating
C. Muscular, metabolic, and progressive D. Central, infectious, and self-limiting

Answer: B. Central, immune-mediated, and demyelinating

Explanation

MS is a chronic immune-mediated inflammatory demyelinating and neurodegenerative disease confined to the CNS.



2 MS is the leading cause of non-traumatic neurological disability in which population?

- A. Elderly men
- B. ✓ Young adults, particularly women
- C. Neonates
- D. Adolescent males

Answer: B. Young adults, particularly women

Explanation

MS typically has peak onset between 20–40 years and disproportionately affects women, making it the leading cause of non-traumatic neurological disability in this group.

3 Which disease course accounts for the majority of MS cases at onset?

- A. Primary progressive MS
- B. Secondary progressive MS
- C. ✓ Relapsing-remitting MS
- D. Progressive-relapsing MS

Answer: C. Relapsing-remitting MS

Explanation

Approximately 85% of patients initially present with relapsing-remitting MS (RRMS).

4 A first clinical demyelinating episode that has not yet fulfilled criteria for MS is termed:

- A. Radiologically isolated syndrome
- B. ✓ Clinically isolated syndrome (CIS)
- C. Transverse myelitis
- D. Optic neuritis syndrome

Answer: B. Clinically isolated syndrome (CIS)

Explanation

CIS refers to a single clinical episode suggestive of CNS demyelination; it may or may not progress to definite MS depending on subsequent clinical and radiological findings.

5 Which epidemiological pattern is well established for MS?

- A. Prevalence decreases with increasing latitude
- B. ✓ Prevalence increases with increasing latitude
- C. Prevalence is unrelated to geography
- D. MS is more common near the equator

Answer: B. Prevalence increases with increasing latitude

Explanation

MS shows a well-documented latitude gradient, with higher prevalence further from the equator, attributed partly to reduced sunlight exposure and vitamin D levels.

MODULE 2

APPLIED ANATOMY

CNS myelin • Common lesion sites • Tracts & cranial nerves

Myelin in the CNS

CNS myelin is produced by **oligodendrocytes** (versus Schwann cells in the peripheral nervous system). A single oligodendrocyte myelinates multiple axonal segments, unlike a Schwann cell which myelinates only one internodal segment of a single axon. This is why MS — an oligodendrocyte/CNS-myelin-directed disease — spares the peripheral nerves.

Common Sites of Demyelinating Plaques

- **Periventricular white matter** — classic site; lesions oriented perpendicular to the ventricles (“Dawson’s fingers”), following the path of medullary veins
- **Juxtacortical / cortical** — at the grey-white matter junction
- **Infratentorial** — brainstem and cerebellum — produce cranial nerve and cerebellar signs
- **Optic nerve** — optic neuritis, often an early presenting feature
- **Spinal cord** — cervical cord commonly involved — produces UMN limb signs and sphincter disturbance
- **Corpus callosum** — callosal lesions contribute to cognitive slowing

Key Tracts and Structures Implicated in Clinical Signs

Structure	Resulting Clinical Feature
Corticospinal tract	Spastic weakness, hyperreflexia, Babinski sign, clonus
Spinothalamic tract	Impaired pain and temperature sensation
Dorsal columns / medial lemniscus	Loss of vibration and proprioception, sensory ataxia, positive Romberg
Medial longitudinal fasciculus (MLF)	Internuclear ophthalmoplegia (impaired adduction with contralateral abducting nystagmus)
Cerebellum & peduncles	Intention tremor, dysmetria, ataxia, scanning speech (Charcot's triad)
Optic nerve (CN II)	Optic neuritis — painful monocular vision loss, RAPD, reduced colour vision
Trigeminal nerve (CN V)	Trigeminal neuralgia (may be bilateral — atypical for idiopathic cases)

MODULE 2 — MCQs (Q6–Q10)

6 Which cell type produces myelin in the CNS, the primary target in MS?

- A.** Schwann cell **B. ✓ Oligodendrocyte**
C. Astrocyte **D.** Microglia

Answer: B. Oligodendrocyte

Explanation

Oligodendrocytes myelinate multiple CNS axons and are the cells destroyed in MS; Schwann cells myelinate peripheral nerves and are unaffected.



7 Periventricular demyelinating lesions oriented perpendicular to the ventricles are called:

- A. Onion-bulb lesions
- B. ✓ Dawson's fingers
- C. Negri bodies
- D. Lewy bodies

Answer: B. Dawson's fingers

Explanation

These lesions follow the course of medullary veins radiating from the ventricles and are a classic MRI finding in MS.

8 A lesion in the medial longitudinal fasciculus (MLF) in MS characteristically produces:

- A. Trigeminal neuralgia
- B. ✓ Internuclear ophthalmoplegia
- C. Bell's palsy
- D. Horner's syndrome

Answer: B. Internuclear ophthalmoplegia

Explanation

INO presents with impaired adduction of the affected eye and nystagmus of the contralateral abducting eye; it is a highly characteristic (though not exclusive) sign of MS.

9 Loss of vibration sense and a positive Romberg sign in an MS patient indicates involvement of the:

- A. Spinothalamic tract
- B. ✓ Dorsal columns / medial lemniscus
- C. Corticospinal tract
- D. Reticulospinal tract

Answer: B. Dorsal columns / medial lemniscus

Explanation

The dorsal column–medial lemniscus pathway carries vibration and proprioceptive information; its involvement produces sensory ataxia and a positive Romberg sign.

10 Which cranial nerve is most frequently affected as an early presenting feature of MS?

- A. Facial nerve (CN VII)
- B. ✓ Optic nerve (CN II)
- C. Hypoglossal nerve (CN XII)
- D. Accessory nerve (CN XI)

Answer: B. Optic nerve (CN II)

Explanation

Optic neuritis is one of the earliest and most common presenting manifestations of MS.



MODULE 3

PHYSIOLOGY OF DEMYELINATION

Saltatory conduction • Conduction block • Uhthoff's phenomenon

Normal Saltatory Conduction

In myelinated axons, the action potential “jumps” from one node of Ranvier to the next (saltatory conduction), which is far faster and more energy-efficient than continuous conduction. Voltage-gated sodium channels are concentrated at the nodes, while the internodal axon is electrically insulated by myelin.

Effect of Demyelination on Conduction

- Loss of myelin exposes internodal axolemma, which lacks a high density of sodium channels, slowing or blocking conduction.
- Mild demyelination → slowed conduction velocity (e.g., delayed VEP P100 latency in optic neuritis).
- Severe demyelination → conduction block — the impulse fails to propagate altogether, producing clinical deficit.
- Chronic demyelination increases the axon's metabolic (ATP) demand to maintain conduction, contributing to central fatigue.

Uhthoff's Phenomenon — High-Yield

- Temporary worsening of neurological symptoms with a rise in body temperature (hot bath, fever, exercise, hot weather).
- Physiological basis: heat reduces the safety factor for conduction across demyelinated segments by further inactivating sodium channels, pushing marginal fibres into conduction block.
- Symptoms typically reverse with cooling — forms the rationale for pre-cooling strategies in PT practice.

Remyelination and Neuroplasticity

- Oligodendrocyte precursor cells (OPCs) can migrate to demyelinated plaques and attempt repair, forming thinner, shorter 'shadow plaques'.
- Remyelination capacity declines with lesion chronicity and age, and is markedly reduced in progressive MS.
- Rehabilitation exploits activity-dependent neuroplasticity (cortical reorganisation, use-dependent strengthening of residual pathways) to optimise function despite fixed structural damage.

MODULE 3 — MCQs (Q11–Q15)

11 Saltatory conduction refers to impulse propagation that:

- | | |
|---|---|
| A. Travels continuously along the axon membrane | B. ✓ Jumps from node to node of Ranvier |
| C. Occurs only in unmyelinated fibres | D. Is independent of sodium channels |

Answer: B. Jumps from node to node of Ranvier

Explanation

In myelinated axons, voltage-gated sodium channels are concentrated at the nodes of Ranvier, allowing the action potential to jump between nodes — a fast, energy-efficient mode of conduction.



12 Complete failure of impulse propagation across a demyelinated axon segment is termed:

- A. Saltatory conduction
- B. ✓ **Conduction block**
- C. Wallerian degeneration
- D. Temporal summation

Answer: B. Conduction block

Explanation

Severe demyelination exposes internodal axolemma with insufficient sodium channel density, causing the action potential to fail to propagate — producing clinical neurological deficit.

13 The physiological basis of Uhthoff's phenomenon is:

- A. Cold-induced axonal hyperexcitability
- B. ✓ **Heat-induced reduction in the safety factor for conduction**
- C. Increased myelin thickness with heat
- D. Improved sodium channel function with heat

Answer: B. Heat-induced reduction in the safety factor for conduction

Explanation

Elevated temperature further impairs conduction across already-compromised demyelinated segments, temporarily worsening symptoms; this reverses with cooling.

14 Which finding on visual evoked potential testing reflects demyelination-related conduction slowing?

- A. Absent P100 wave only
- B. ✓ **Prolonged P100 latency**
- C. Shortened P100 latency
- D. Increased P100 amplitude only

Answer: B. Prolonged P100 latency

Explanation

Demyelination slows conduction along the optic pathway, producing a delayed (prolonged) P100 latency, even when vision is subjectively normal.

15 Thinly remyelinated plaques with shorter internodes are referred to as:

- A. Dawson's fingers
- B. ✓ **Shadow plaques**
- C. Negri bodies
- D. Lewy plaques

Answer: B. Shadow plaques

Explanation

Oligodendrocyte precursor cells can partially remyelinate lesions, producing thinner, shorter segments of myelin known as shadow plaques; this capacity declines with chronicity and in progressive disease.



MODULE 4

PATHOLOGY, ETIOLOGY & DIAGNOSIS

Immunopathogenesis • Risk factors • MRI/CSF • McDonald criteria

Immunopathogenesis

- Autoreactive CD4+ T-helper (Th1/Th17) lymphocytes cross a compromised blood–brain barrier and target CNS myelin antigens.
- B lymphocytes and macrophages/microglia amplify inflammation and contribute to demyelination and axonal injury.
- Repeated inflammatory attacks cause plaque formation, gliosis (astrocytic scarring), and progressive axonal loss — the latter correlates most closely with permanent disability.

Risk Factors

Category	Factor
Genetic	HLA-DRB1*15:01 (strongest known genetic susceptibility allele)
Infectious	Epstein–Barr virus (EBV) infection — strongly implicated as a near-necessary trigger
Nutritional	Vitamin D deficiency
Lifestyle	Smoking, adolescent obesity
Geographic	Higher latitude / reduced sunlight exposure
Demographic	Female sex, family history

Diagnostic Work-up

- **MRI (investigation of choice)** — demyelinating plaques — periventricular, juxtacortical, infratentorial, spinal cord; Gadolinium-enhancing lesions indicate active inflammation
- **CSF analysis** — oligoclonal IgG bands not present in matched serum, indicating intrathecal antibody synthesis
- **Visual evoked potentials (VEP)** — prolonged P100 latency detects subclinical optic nerve demyelination
- **McDonald Criteria** — combines clinical attacks, MRI lesions, and CSF findings to demonstrate dissemination in space (multiple CNS sites) and dissemination in time (lesions of different ages / new attacks)

Diagnostic Concept — High Yield

- Dissemination in SPACE = lesions in ≥ 2 of 4 characteristic CNS regions (periventricular, juxtacortical/cortical, infratentorial, spinal cord).
- Dissemination in TIME = simultaneous presence of enhancing and non-enhancing lesions on a single MRI, OR a new lesion on follow-up MRI, OR a new clinical attack.

MODULE 4 — MCQs (Q16–Q20)



16 Which immune cell subset is central to initiating the autoimmune attack on CNS myelin in MS?

- A. Neutrophils
- B. ✓ CD4+ T-helper (Th1/Th17) lymphocytes
- C. Eosinophils
- D. Platelets

Answer: B. CD4+ T-helper (Th1/Th17) lymphocytes

Explanation

Autoreactive CD4+ T cells cross the blood-brain barrier and initiate the inflammatory attack on myelin; B cells and macrophages/microglia further amplify the process.

17 Which factor correlates most closely with permanent, irreversible disability in MS?

- A. Number of relapses
- B. ✓ Axonal loss
- C. Degree of spasticity
- D. CSF protein level

Answer: B. Axonal loss

Explanation

While demyelination itself may be reversible via remyelination, cumulative axonal loss is the pathological substrate most closely linked to permanent disability.

18 The strongest known genetic risk allele for MS is located within which gene region?

- A. HLA-B27
- B. ✓ HLA-DRB1*15:01
- C. HLA-A3
- D. HLA-B51

Answer: B. HLA-DRB1*15:01

Explanation

HLA-DRB1*15:01, part of the HLA-DR2 haplotype, is the strongest established genetic susceptibility factor for MS.

19 Which viral infection is most strongly implicated as an environmental trigger for MS?

- A. ✓ Epstein–Barr virus
- B. Herpes zoster virus
- C. Hepatitis B virus
- D. Cytomegalovirus

Answer: A. Epstein–Barr virus

Explanation

Epidemiological and immunological evidence strongly links EBV infection to MS risk, positioning it as a near-necessary environmental trigger; this has spurred research into antiviral and vaccine strategies.

20 The McDonald Criteria establish a diagnosis of MS by demonstrating:

- A. ✓ Dissemination in time and space
- B. Presence of muscle fasciculations only
- C. Elevated serum creatine kinase
- D. Peripheral nerve conduction block

Answer: A. Dissemination in time and space

Explanation

The criteria combine clinical, MRI, and CSF evidence to confirm that demyelinating lesions have occurred in different CNS locations (space) and at different time points (time).



MODULE 5

MUSCULOSKELETAL CHANGES

Spasticity • Contractures • Bone health • Postural deformity

Spasticity

- Velocity-dependent increase in resistance to passive stretch, resulting from corticospinal (UMN) tract damage.
- Affects up to 80% of people with MS during the disease course, most often the lower limbs (hip adductors, knee extensors/flexors, ankle plantarflexors).
- Can be a source of functional advantage (e.g., supporting stance in weak limbs) as well as pain, impaired hygiene, and reduced function — must always be assessed in the context of overall function, not treated in isolation.

Secondary Musculoskeletal Consequences

- **Contractures** — prolonged spasticity and immobility predispose to soft-tissue shortening, especially ankle plantarflexion and hip/knee flexion contractures
- **Disuse muscle weakness/atrophy** — compounds primary neurological weakness, particularly in advanced disability
- **Osteoporosis/osteopenia** — reduced weight-bearing activity, vitamin D deficiency, and corticosteroid use increase fracture risk
- **Postural deformity** — asymmetric weakness and tone lead to pelvic obliquity, scoliosis, and compensatory kyphosis
- **Falls and fractures** — combination of weakness, ataxia, sensory loss, and osteoporosis substantially raises fall-related fracture risk

Clinical Pearl

- Always differentiate true muscle weakness from fatigue-related and spasticity-masked weakness before prescribing strengthening programs — overtreatment of spasticity without addressing underlying weakness can destabilise function (e.g., removing extensor spasticity that was assisting standing).

MODULE 5 — MCQs (Q21–Q25)

21 Spasticity in MS is best defined as:

- A. A fixed, non-reducible joint contracture
- B. ✓ A velocity-dependent increase in resistance to passive stretch
- C. Loss of deep tendon reflexes
- D. Reduced muscle tone at rest

Answer: B. A velocity-dependent increase in resistance to passive stretch

Explanation

Spasticity is a UMN sign resulting from corticospinal tract damage, characterised by increased resistance to passive stretch that varies with the speed of the movement.



22 Which muscle groups are most commonly affected by spasticity in MS?

- A. ✓ **Hip adductors, knee flexors/extensors, ankle plantarflexors**
B. Intrinsic hand muscles only
C. Extraocular muscles
D. Diaphragm

Answer: A. Hip adductors, knee flexors/extensors, ankle plantarflexors

Explanation

Lower limb muscle groups — particularly hip adductors, knee flexors/extensors, and ankle plantarflexors — are most frequently affected, impacting gait and transfers.

23 Which factor increases fracture risk in people with MS?

- A. Increased weight-bearing activity
B. ✓ **Osteoporosis secondary to reduced mobility and corticosteroid use**
C. Excess vitamin D
D. Increased muscle bulk

Answer: B. Osteoporosis secondary to reduced mobility and corticosteroid use

Explanation

Reduced weight-bearing activity, vitamin D deficiency, and repeated corticosteroid courses for relapses all contribute to osteoporosis and increased fracture risk.

24 A patient with MS relies partly on extensor spasticity in the lower limb to maintain standing. The MOST appropriate clinical reasoning is to:

- A. Aggressively reduce all spasticity regardless of function
B. ✓ **Assess the functional role of spasticity before intervening**
C. Immediately prescribe botulinum toxin
D. Ignore spasticity entirely

Answer: B. Assess the functional role of spasticity before intervening

Explanation

Spasticity can be functionally useful (e.g., assisting stance in a weak limb); management decisions must weigh functional benefit against pain, hygiene, and mobility limitations on a case-by-case basis.

25 Prolonged plantarflexor spasticity with immobility in MS most commonly predisposes to which contracture?

- A. Wrist flexion contracture
B. ✓ **Ankle plantarflexion (equinus) contracture**
C. Elbow extension contracture
D. Cervical extension contracture

Answer: B. Ankle plantarflexion (equinus) contracture

Explanation

Sustained plantarflexor tone and reduced ambulation commonly lead to shortening of the gastrocnemius–soleus complex, producing an equinus contracture that impairs gait.



MODULE 6

NEUROMUSCULAR & CLINICAL FEATURES

UMN & cerebellar signs • Sensory changes • Fatigue • Cognition • Autonomic

Motor Features

- Upper motor neuron (UMN) signs: hyperreflexia, spasticity, positive Babinski sign, clonus, spastic weakness (predominantly CNS pathology — significant LMN wasting is atypical).
- Cerebellar signs: intention tremor, dysmetria, dysdiadochokinesia, ataxia, nystagmus, scanning speech — classically described as Charcot's neurological triad.
- Gait pattern: characteristic spastic-ataxic gait from combined corticospinal and cerebellar involvement.

Sensory & Paroxysmal Features

- **Lhermitte's sign** — electric-shock sensation down the spine on neck flexion, from cervical cord demyelination
- **Paraesthesia / numbness** — common early symptom from posterior column or spinothalamic involvement
- **Trigeminal neuralgia** — paroxysmal facial pain; bilateral presentation should raise suspicion for MS
- **Tonic spasms** — brief, painful, paroxysmal muscle spasms

Non-Motor and Systemic Features

- **Fatigue** — one of the most common and disabling symptoms; arises from increased metabolic demand of impaired conduction (primary/central fatigue) plus deconditioning, poor sleep, and depression (secondary fatigue)
- **Cognitive impairment** — affects roughly 40–65% of patients — slowed information processing speed, impaired working memory and executive function; often under-recognised
- **Autonomic dysfunction** — bladder urgency/retention, constipation, sexual dysfunction from spinal cord involvement
- **Mood disorders** — depression and anxiety are common comorbidities and influence rehabilitation engagement

MODULE 6 — MCQs (Q26–Q30)

26 Charcot's neurological triad in MS consists of:

- A. Rigidity, bradykinesia, resting tremor
- B. ✓ Nystagmus, intention tremor, scanning speech
- C. Ataxia, aphasia, apraxia
- D. Ptosis, diplopia, dysarthria

Answer: B. Nystagmus, intention tremor, scanning speech

Explanation

This classic triad reflects cerebellar and cerebellar-pathway involvement in MS.

27 Electric shock-like sensation radiating down the spine on neck flexion is known as:

- A. Uhthoff's phenomenon
- B. ✓ Lhermitte's sign
- C. Romberg's sign
- D. Babinski sign

Answer: B. Lhermitte's sign

Explanation

Lhermitte's sign results from demyelination of the cervical spinal cord and is elicited by neck flexion.



28 Fatigue in MS is best explained physiologically by:

- A. ✓ Increased metabolic demand of impaired axonal conduction plus secondary factors like poor sleep** B. Primary muscle fibre necrosis
C. Peripheral nerve compression D. Excess dopamine release

Answer: A. Increased metabolic demand of impaired axonal conduction plus secondary factors like poor sleep

Explanation

Central (primary) fatigue arises from the higher energy cost of conducting impulses through demyelinated axons; secondary fatigue is compounded by deconditioning, disturbed sleep, and depression.

29 Approximately what proportion of people with MS experience cognitive impairment?

- A. <5% B. 10–15%
C. ✓ 40–65% D. Nearly 100%

Answer: C. 40–65%

Explanation

Cognitive dysfunction — particularly slowed processing speed and impaired working memory — affects roughly 40–65% of people with MS and is frequently under-recognised in clinical practice.

30 Bilateral trigeminal neuralgia in a young adult should raise clinical suspicion for:

- A. Bell's palsy **B. ✓ Multiple sclerosis**
C. Migraine D. Tension headache

Answer: B. Multiple sclerosis

Explanation

While trigeminal neuralgia is usually idiopathic and unilateral, bilateral presentation — especially in a young adult — is atypical and warrants investigation for an underlying demyelinating cause such as MS.



MODULE 7

PHYSICAL THERAPY ASSESSMENT

Outcome measures • Systems review • Examination framework

Standardised Outcome Measures

Domain	Tool
Overall disability	Expanded Disability Status Scale (EDSS) — Kurtzke; heavily weighted towards ambulation
Composite function	Multiple Sclerosis Functional Composite (MSFC): Timed 25-Foot Walk (T25FW), 9-Hole Peg Test (9HPT), PASAT/SDMT (cognition)
Balance	Berg Balance Scale; Dynamic Gait Index; Activities-specific Balance Confidence (ABC) Scale
Tone / spasticity	Modified Ashworth Scale (MAS)
Fatigue	Modified Fatigue Impact Scale (MFIS); Fatigue Severity Scale (FSS)
Walking ability (self-report)	12-Item MS Walking Scale (MSWS-12)
Walking capacity/endurance	6-Minute Walk Test (6MWT); Timed 25-Foot Walk
Gait/fall risk	Tinetti Performance-Oriented Mobility Assessment (POMA)
Manual dexterity	9-Hole Peg Test (9HPT)
Cognition (screen)	Symbol Digit Modalities Test (SDMT)

Systems Review / Examination Framework

- **History** — disease course/subtype, relapse pattern, current DMT, fatigue pattern, heat sensitivity, bladder/bowel history, fall history
- **Tone & reflexes** — Modified Ashworth Scale, deep tendon reflexes, clonus, Babinski sign
- **Strength** — manual muscle testing — interpret cautiously in the context of fatigue and effort-dependence
- **Sensation** — light touch, pin-prick, vibration, proprioception; test for Lhermitte's sign
- **Coordination** — finger-to-nose, heel-to-shin, rapid alternating movements, dysmetria
- **Balance & gait** — static/dynamic balance, gait pattern and deviations, assistive device needs, fall risk
- **Fatigue & cognition** — screen using MFIS/FSS and SDMT; assess impact on therapy tolerance and pacing needs
- **Cardiorespiratory fitness** — important given deconditioning risk and heat sensitivity during aerobic testing
- **Skin & pressure areas** — especially in advanced disability with reduced mobility

Assessment Pearl

- Schedule strength and endurance testing earlier in the day and in a cool environment to avoid Uhthoff-related pseudo-worsening of performance, which can otherwise be mistaken for a true relapse or decline.

MODULE 7 — MCQs (Q31–Q35)



31 Which scale is the standard tool for quantifying overall disability in MS and is heavily weighted towards ambulation?

- A. Berg Balance Scale
- B. ✓ Expanded Disability Status Scale (EDSS)
- C. Modified Ashworth Scale
- D. Fatigue Severity Scale

Answer: B. Expanded Disability Status Scale (EDSS)

Explanation

The Kurtzke EDSS is the most widely used disability scale in MS, assessing functional systems with a strong emphasis on walking ability.

32 The Multiple Sclerosis Functional Composite (MSFC) includes all of the following EXCEPT:

- A. Timed 25-Foot Walk
- B. 9-Hole Peg Test
- C. PASAT/SDMT
- D. ✓ Modified Ashworth Scale

Answer: D. Modified Ashworth Scale

Explanation

The MSFC is a composite of ambulation (T25FW), upper limb dexterity (9HPT), and cognitive processing speed (PASAT/SDMT). The Modified Ashworth Scale separately assesses spasticity, not composite function.

33 Which scale specifically measures self-perceived walking ability in people with MS?

- A. ✓ MSWS-12
- B. Berg Balance Scale
- C. EDSS
- D. MFIS

Answer: A. MSWS-12

Explanation

The 12-Item MS Walking Scale (MSWS-12) is a patient-reported outcome measure of perceived walking ability and its impact on daily life.

34 Which tool is used to assess spasticity severity in clinical practice?

- A. Berg Balance Scale
- B. ✓ Modified Ashworth Scale
- C. 6-Minute Walk Test
- D. Tinetti POMA

Answer: B. Modified Ashworth Scale

Explanation

The Modified Ashworth Scale grades resistance to passive stretch and is the standard clinical tool for quantifying spasticity.



35

Best practice when assessing exercise tolerance in a person with MS who is heat-sensitive is to:

- A. Test in a warm room to simulate real-world conditions
- B. ✓ Test earlier in the day in a cool environment
- C. Avoid testing altogether
- D. Test only after a hot bath

Answer: B. Test earlier in the day in a cool environment

Explanation

Testing in a cool environment and earlier in the day minimises Uhthoff-related pseudo-worsening, yielding a more accurate assessment of true functional capacity.



MODULE 8

PHYSICAL THERAPY INTERVENTION

Exercise therapy • Spasticity & fatigue management • Technology-assisted rehab

Exercise Therapy

- **Aerobic exercise** — moderate-intensity training improves fatigue, cardiovascular fitness, mood, and mobility; recommended throughout the disease course per Kalb et al. exercise guidelines
- **Progressive resistance training** — improves strength and function; dosage should be individualised, monitoring for fatigue and heat sensitivity
- **Balance & gait training** — task-specific practice, dual-task training, and functional gait training reduce fall risk
- **Stretching / ROM** — maintains soft-tissue length and reduces contracture risk in spastic muscle groups

Symptom-Specific Management

- **Spasticity management** — positioning, stretching, splinting; combined with medical management (e.g., baclofen, botulinum toxin) as indicated; avoid over-treating functionally useful spasticity
- **Fatigue management** — energy conservation education, activity pacing, graded exercise, cooling garments/pre-cooling strategies to counter Uhthoff's phenomenon
- **Aquatic therapy** — buoyancy-assisted exercise in cool water supports strengthening and gait training while minimising heat-related symptom exacerbation
- **Fall prevention** — environmental modification, assistive devices, balance/strength training, education
- **Bladder/bowel & pelvic floor** — referral/collaboration with continence physiotherapy as indicated
- **Respiratory & bulbar care** — breathing exercises and dysphagia management in advanced disease

Technology-Assisted & Emerging Rehabilitation Approaches

- Robot-assisted / body-weight-supported treadmill training (BWSTT) for gait retraining.
- Functional electrical stimulation (FES) for foot-drop and muscle activation.
- Virtual reality (VR) rehabilitation and exergaming to improve balance, engagement, and adherence.
- Wearable-guided therapy and telerehabilitation — hybrid in-person plus remote models show promising long-term outcomes.
- Dual-task and cognitive-motor training to address the interaction between cognitive and motor deficits.

Guiding Framework

- The Rehabilitation in Multiple Sclerosis (RiMS) network endorses the WHO Rehabilitation 2030 call to action, positioning rehabilitation as an essential, integrated component of MS care across the disease course — not solely for advanced disability.

MODULE 8 — MCQs (Q36–Q40)



36 According to current exercise guidelines, aerobic exercise for people with MS should be recommended:

- A. Only in the terminal stage of disease
- B. ✓ Throughout the disease course, with individualisation
- C. Only prior to diagnosis
- D. Never, due to risk of relapse

Answer: B. Throughout the disease course, with individualisation

Explanation

Current lifestyle physical activity guidelines (e.g., Kalb et al.) recommend exercise across the disease course, tailored to the individual's symptoms, disability level, and heat sensitivity.

37 Which intervention specifically helps counter Uhthoff-related symptom exacerbation during exercise?

- A. Performing exercise in a heated pool
- B. ✓ Pre-cooling strategies / cooling garments
- C. Exercising during peak afternoon heat
- D. Avoiding all aerobic activity

Answer: B. Pre-cooling strategies / cooling garments

Explanation

Cooling strategies lower core body temperature, reducing the conduction-related symptom worsening seen with Uhthoff's phenomenon, and allow better exercise tolerance.

38 Which technology-assisted intervention is used specifically to address foot-drop in MS?

- A. ✓ Functional electrical stimulation (FES)
- B. Body-weight-supported treadmill training only
- C. Virtual reality gaming only
- D. Cooling garments

Answer: A. Functional electrical stimulation (FES)

Explanation

FES stimulates the common peroneal nerve/ankle dorsiflexors during the swing phase of gait to correct foot-drop, a common gait deviation in MS.

39 A scoping review of physiotherapy advances in MS (2014–2025) found that rehabilitation has generally shifted toward:

- A. Purely compensatory, bed-rest-based care
- B. ✓ Technology-integrated, neurorestorative rehabilitation
- C. Complete avoidance of exercise
- D. Isolated pharmacological management only

Answer: B. Technology-integrated, neurorestorative rehabilitation

Explanation

The review found that physiotherapy has moved from compensatory approaches toward neurorestorative and technology-integrated models (VR, exergaming, telerehabilitation, wearables), often matching or exceeding outcomes of conventional therapy alone.



40 The RiMS network's endorsement of the WHO Rehabilitation 2030 call to action emphasises that rehabilitation in MS should be:

- A. Reserved only for wheelchair-dependent patients
- B. ✓ An integrated component of care across the disease course
- C. Delivered exclusively via telehealth
- D. Optional and secondary to medication

Answer: B. An integrated component of care across the disease course

Explanation

This framework positions rehabilitation as essential throughout the MS disease trajectory, not only in advanced disability, aligning with global calls to strengthen rehabilitation services.



MODULE 9

RECENT RESEARCH & ADVANCES

2025–2026 rehabilitation and remyelination research

Rehabilitation Research (2025–2026)

- **Learmonth Y, J Physiother 2026;72(1):11–22** — a comprehensive evidence review of physiotherapy management of MS, summarising common impairments and the evidence base for interventions used in physiotherapy practice, funded by an MS Australia Fellowship
- **Scoping review (Int J Res Med Sci, 2025)** — reviewed 18 studies (2014–2025) and found technology-assisted rehabilitation — high-intensity interval training, virtual reality, exergaming, telerehabilitation, wearable-guided therapy, and cognitive-motor dual-task training — achieved outcomes comparable or superior to conventional therapy for mobility, balance, and fatigue; hybrid in-person plus remote models were most effective long-term
- **Technology-based balance rehabilitation meta-analysis (Arch Phys Med Rehabil, 2025)** — systematic review/meta-analysis confirmed technology-based balance interventions are comparable to, and in some contexts more effective than, conventional physical therapy
- **RiMS Network / WHO Rehabilitation 2030 (Int J MS Care, 2025)** — positioned rehabilitation as key to the WHO's global call to action for people with MS, advocating for stronger integration of rehabilitation services into standard MS care

Disease-Modifying Therapy & Remyelination Research

- **High-efficacy anti-CD20 therapies** — extension trials of ocrelizumab and ofatumumab show near-complete suppression of relapse activity and new MRI lesion formation, with some patients experiencing fewer than one relapse per 20–30 years on continuous high-efficacy therapy
- **BTK inhibitors (Bruton's tyrosine kinase)** — agents such as fenebrutinib and remibrutinib act on B cells, macrophages, and microglia within the CNS, offering a potential mechanism to address disability progression independent of relapse activity — an unmet need in progressive MS
- **Remyelination research** — a 2025 systematic review (De Keersmaecker et al., *Eur J Neurol*) evaluated the remyelinating properties of currently approved DMTs, informing future combination immunomodulatory + regenerative strategies
- **Clemastine fumarate trials** — this generic antihistamine (M1 muscarinic receptor antagonist) remains the most-studied candidate remyelinating agent, with trials using advanced MRI techniques (e.g., ultrashort echo time imaging) to quantify myelin repair in chronic lesions

Exam Takeaway

- Current rehabilitation research supports individualised, technology-integrated, and hybrid (in-person + remote) exercise-based physiotherapy as central to MS management across the disease course — shifting away from a purely compensatory model. On the pharmacological side, research focus has expanded beyond relapse suppression toward remyelination and progression independent of relapse activity (PIRA).

MODULE 9 — MCQs (Q41–Q45)



41 Recent scoping-review evidence (2025) indicates that technology-assisted rehabilitation in MS, compared to conventional therapy, generally shows:

- A. Consistently inferior outcomes
- B. ✓ Outcomes comparable to or better than conventional therapy for mobility, balance, and fatigue
- C. No measurable effect on any outcome
- D. Benefit only for cognitive outcomes

Answer: B. Outcomes comparable to or better than conventional therapy for mobility, balance, and fatigue

Explanation

The 2025 scoping review found technology-assisted approaches such as VR, exergaming, and telerehabilitation achieved comparable or superior outcomes to conventional therapy across mobility, balance, and fatigue domains.

42 Which care model was identified as most effective for long-term physiotherapy management of MS in recent research?

- A. Purely in-person care only
- B. Purely remote/telehealth care only
- C. ✓ Hybrid in-person plus remote care
- D. No structured care model

Answer: C. Hybrid in-person plus remote care

Explanation

Recent evidence suggests hybrid models combining in-person and remote (tele-) rehabilitation appear most effective for long-term management of MS.

43 BTK (Bruton's tyrosine kinase) inhibitors under investigation in MS are of particular interest because they may target:

- A. Only peripheral nerve regeneration
- B. ✓ Disability progression independent of relapse activity
- C. Bone density loss exclusively
- D. Only visual symptoms

Answer: B. Disability progression independent of relapse activity

Explanation

BTK inhibitors act on B cells, macrophages, and microglia within the CNS, offering a potential mechanism to address disability progression that occurs independent of clinical relapses — a major unmet need in progressive MS.

44 Clemastine fumarate is being investigated in MS research primarily for its potential as a:

- A. Anti-spasticity agent
- B. ✓ Remyelinating agent
- C. Analgesic for trigeminal neuralgia
- D. Antibiotic

Answer: B. Remyelinating agent

Explanation

Clemastine, an antihistamine with M1 muscarinic receptor antagonism, was identified through drug screening as a candidate agent capable of stimulating myelin repair and remains the most-studied remyelinating compound in clinical trials.



45 The RiMS network's alignment with the WHO Rehabilitation 2030 initiative primarily advocates for:

- A. Reducing physiotherapy access for people with MS
- B. ✓ Strengthening integration of rehabilitation as a core component of global MS care
- C. Replacing physiotherapy with medication alone
- D. Limiting rehabilitation research funding

Answer: B. Strengthening integration of rehabilitation as a core component of global MS care

Explanation

This initiative calls for rehabilitation to be recognised and resourced as an essential, integrated part of MS care internationally, in line with the WHO's broader rehabilitation 2030 call to action.



MODULE 10

CASE STUDY

Clinical scenario • Assessment • Goals • Intervention • Outcome

Clinical Scenario

Ms. R, 32 years, diagnosed with Relapsing-Remitting MS 4 years ago, is referred to physiotherapy following a recent relapse involving the cervical spinal cord and cerebellar pathways. She reports worsening imbalance, right lower limb stiffness, and profound fatigue that worsens in the afternoon and with heat exposure. She is independently ambulant with a single-point stick for community distances but reports two falls in the past month.

Physiotherapy Assessment Findings

Domain	Finding
Tone (MAS)	Grade 2 spasticity, right ankle plantarflexors and knee extensors
Strength	4/5 right hip flexors/abductors; fatigable weakness on repeated testing
Coordination	Mild dysmetria on right heel-to-shin; intention tremor, right upper limb
Sensation	Reduced vibration sense, bilateral lower limbs; positive Lhermitte's sign
Balance	Berg Balance Scale: 42/56 (moderate fall risk)
Gait	Spastic-ataxic pattern, reduced right stance-phase stability, foot-drop tendency
Walking capacity	6MWT: 320 m (reduced for age); MSWS-12 indicates high perceived walking impact
Fatigue	MFIS score indicating significant fatigue impact on daily activity
EDSS	Estimated 4.0 — significant disability, self-sufficient, up and about ≈12 hours/day

Problem List

- Impaired dynamic balance with increased fall risk.
- Right lower limb spasticity contributing to gait deviation and foot-drop tendency.
- Cerebellar ataxia/dysmetria affecting fine motor and gait control.
- Significant fatigue limiting activity tolerance, exacerbated by heat.
- Reduced walking capacity and community mobility confidence.

Goals

- **Short-term (2–4 weeks)** — reduce fall frequency to zero; improve Berg Balance Scale by ≥ 4 points; patient education on energy conservation and heat management
- **Long-term (8–12 weeks)** — improve 6MWT distance; reduce reliance on single-point stick for indoor mobility where safe; improve MFIS score and self-reported walking confidence (MSWS-12)

Intervention Plan

- **Balance & gait training** — task-specific balance exercises, dual-task training, gait re-education targeting right stance stability; consider FES trial for foot-drop
- **Spasticity management** — stretching and positioning program for right plantarflexors/knee extensors, in liaison with the medical team regarding pharmacological options
- **Cerebellar/coordination retraining** — Frenkel-type coordination exercises, proximal stability work
- **Aerobic & resistance exercise** — individualised moderate-intensity aerobic program in a cool environment, progressive resistance training for right hip musculature



- **Fatigue management** — energy conservation education, activity pacing, cooling strategies prior to exercise sessions
- **Falls prevention & education** — home environment review, assistive device reassessment, patient/family education on relapse vs Uhthoff-related fluctuation

Outcome (Re-assessment at 8 Weeks)

Berg Balance Scale improved to 48/56; no further falls reported; 6MWT distance improved by 28 metres; MFIS score reduced, reflecting better-managed fatigue; patient reports improved confidence in community ambulation. Continued follow-up planned with periodic outcome-measure reassessment and liaison with the neurology team regarding disease-modifying therapy review.

CASE-BASED MCQ (Q46)

46 In the case above, the **MOST** appropriate initial physiotherapy priority given a recent history of two falls is:

- | | |
|---|---|
| A. Aggressive resistance training to fatigue | B. ✓ Balance and fall-risk-focused intervention with environmental/device review |
| C. Complete rest until the next relapse resolves | D. Isolated cognitive training only |

Answer: B. Balance and fall-risk-focused intervention with environmental/device review

Explanation

Given the recent falls and moderate fall-risk Berg Balance Scale score, addressing balance, gait stability, and environmental/assistive-device factors is the most immediate safety priority before progressing intensive strengthening.



MODULE 11

HIGH-YIELD REVISION SUMMARY

One-page rapid revision for exam day

Rapid Revision — Read Before the Exam

- Autoimmune, CNS-restricted, demyelinating disease targeting oligodendrocytes; strongest genetic risk = HLA-DRB1*15:01.
- Peak onset 20–40 years; Female:Male $\approx$ 2–3:1; prevalence rising globally with latitude gradient.
- Most common course: Relapsing-remitting MS ($\approx$ 85%); diagnosis requires dissemination in time AND space (McDonald Criteria).
- Classic MRI: Dawson's fingers (periventricular); classic CSF: oligoclonal IgG bands; classic VEP: delayed P100.
- Most common presenting symptom: optic neuritis; classic ocular sign: internuclear ophthalmoplegia (MLF lesion).
- Charcot's triad (cerebellar): nystagmus, intention tremor, scanning speech; classic gait: spastic-ataxic.
- Uhthoff's phenomenon = heat-induced temporary conduction failure/worsening; Lhermitte's sign = cervical cord demyelination.
- Spasticity, contractures, disuse weakness, and osteoporosis are the principal musculoskeletal consequences.
- Fatigue and cognitive impairment (40–65%) are highly prevalent, disabling, and often under-recognised.
- Key outcome measures: EDSS, MSFC (T25FW/9HPT/SDMT), Berg Balance Scale, Modified Ashworth Scale, MFIS/FSS, MSWS-12, 6MWT.
- PT management: individualised aerobic + resistance exercise, balance/gait training, spasticity and fatigue management, cooling strategies, and technology-assisted rehabilitation (VR, FES, telerehab).
- Acute relapse: high-dose IV methylprednisolone; DMTs: interferon-beta, glatiramer acetate, fingolimod, natalizumab, ocrelizumab; emerging BTK inhibitors target progression independent of relapse.
- Recent research (2025–2026) favours technology-integrated, neurorestorative, hybrid rehabilitation models and continues to pursue remyelination strategies (e.g., clemastine fumarate).

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