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CHAPTER 1

Fundamentals: Neurons & Motor Pathways

1.1 The Neuron — Structure & Function

A neuron consists of a cell body (soma), dendrites, and an axon. Motor neurons carry efferent signals from the CNS to effector organs (muscles/glands). Sensory neurons carry afferent signals from periphery to CNS. Interneurons connect neurons within the CNS.

- Cell body (soma): Contains nucleus, Nissl bodies (rough ER), mitochondria
- Axon: Conducts impulses away from soma; myelinated or unmyelinated
- Myelin sheath: Formed by Schwann cells (PNS) or oligodendrocytes (CNS)
- Nodes of Ranvier: Gaps in myelin; saltatory conduction increases velocity
- Synapse: Junction between neurons; uses neurotransmitters (ACh, DA, GABA)

1.2 Motor Pathways — Pyramidal & Extrapyramidal

Pyramidal (Corticospinal) Tract: Primary voluntary motor pathway.

- Origin: Primary motor cortex (area 4), premotor (area 6), SMA
- Course: Through corona radiata → internal capsule (posterior limb, genu) → cerebral peduncle → pons (corticobulbar) → medullary pyramids
- Decussation: 85-90% cross at pyramidal decussation (lower medulla)
- Termination: Synapse on anterior horn cells (alpha motor neurons) at spinal cord
- Function: Fine voluntary movements, especially distal limb muscles

Extrapyramidal System: Modulates movement via basal ganglia, cerebellum, thalamus.

- Includes: Rubrospinal, reticulospinal, vestibulospinal, tectospinal tracts
- Function: Postural control, tone regulation, automatic movements, coordination

1.3 Alpha and Gamma Motor Neurons

Feature	Alpha Motor Neuron	Gamma Motor Neuron
Location	Anterior horn of spinal cord	Anterior horn of spinal cord
Fiber type	A-alpha (Ia)	A-gamma
Size	Large (70-120 μ m)	Small (15-30 μ m)
Target	Extrafusal muscle fibers	Intrafusal fibers (muscle spindle)
Function	Voluntary movement	Regulates spindle sensitivity
Role in tone	Direct muscle contraction	Maintains reflex arc sensitivity

1.4 Stretch Reflex Arc

Muscle stretch → Ia afferents from muscle spindle → posterior horn → synapse on alpha motor neuron → muscle contraction. This monosynaptic reflex is the basis of deep tendon reflexes (DTR).

Key Concept

- UMN controls LMNS (removes inhibition) → UMN lesion = disinhibition = hyperreflexia
- LMN IS the final common pathway → LMN lesion = interrupts reflex arc = hyporeflexia/areflexia

- Babinski sign: Extensor plantar response — pathognomonic of UMN lesion

CHAPTER 2

UMN vs LMN — Master Comparison Table

Feature	UMN Lesion	LMN Lesion
Location of lesion	Brain/spinal cord (CNS)	Anterior horn cell/nerve root/peripheral nerve/NMJ/muscle
Tone	Hypertonia (spasticity)	Hypotonia/flaccidity
Reflexes (DTR)	Hyperreflexia	Hyporeflexia/Areflexia
Plantar reflex	Extensor (Babinski +ve)	Flexor (normal) or absent
Clonus	Present	Absent
Muscle wasting	Disuse atrophy (late, mild)	Early, severe neurogenic atrophy
Fasciculations	Absent	Present (pathognomonic)
Fibrillations (EMG)	Absent	Present
Weakness pattern	Groups of muscles (pyramidal distribution)	Individual muscles (myotomal distribution)
Upper limb weakness	Extensors > Flexors (flexors spared longer)	Depends on nerve involved
Lower limb weakness	Flexors > Extensors (extensors spared longer)	Depends on nerve involved
Coordination	Impaired (if cerebellar)	Not primarily affected
Bladder	Spastic (urgency, frequency)	Flaccid (overflow incontinence)
Trophic changes	Minimal	Significant (skin, nail changes)
EMG	Normal/minimal changes	Denervation potentials (fibrillations, +ve sharp waves)
NCV	Normal	Reduced (if demyelinating)
Recovery	Slower, incomplete	Better if axon regenerates (1 mm/day)
Cause examples	Stroke, SCI, MS, brain tumors	GBS, polio, peripheral neuropathy, Bell palsy

CHAPTER 3

Upper Motor Neuron (UMN) Lesions — In Detail

3.1 Anatomy of UMN

The UMN cell body lies in Layer V (Betz cells) of the primary motor cortex (Brodmann area 4). The axon descends through internal capsule → cerebral peduncle → pons → medullary pyramid → spinal cord (lateral corticospinal tract after decussation). The UMN exerts an **inhibitory influence** on spinal interneurons and LMN circuits.

3.2 Pathophysiology of UMN Lesion

Loss of UMN inhibition on spinal cord interneurons leads to:

- Release of spinal stretch reflexes → hyperreflexia
- Disinhibition of gamma motor neurons → increased spindle sensitivity → spasticity
- Abnormal activation of flexor withdrawal circuits → spasms
- Loss of fine fractionated movement → weakness in pyramidal distribution
- Babinski sign: Dorsal reticulospinal tract (normally inhibits extensor plantar response) is lost

3.3 Spasticity — Pathophysiology & Clinical Features

Spasticity = velocity-dependent increase in tonic stretch reflexes. Lance (1980) definition: "motor disorder characterized by velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks."

Modified Ashworth Scale (MAS) — Grading Spasticity

- 0 — No increase in muscle tone
- 1 — Slight increase: catch and release or minimal resistance at end of ROM
- 1+ — Slight increase: catch followed by minimal resistance throughout remainder (<50%) of ROM
- 2 — More marked increase in muscle tone through most of ROM, but parts easily moved
- 3 — Considerable increase in tone; passive movement difficult
- 4 — Affected part(s) rigid in flexion or extension

3.4 Clinical Features of UMN Lesion (Detailed)

POSITIVE FEATURES (Release phenomena)	NEGATIVE FEATURES (Loss of function)
Spasticity (velocity-dependent hypertonia)	Weakness/paresis (pyramidal distribution)
Hyperreflexia (exaggerated DTRs)	Loss of fine motor control
Babinski sign / extensor plantar	Fatigue
Clonus (rhythmic involuntary contractions)	Loss of dexterity
Spasms (flexor or extensor)	Impaired coordination (if cerebellar involved)
Clasp-knife phenomenon	Decreased proprioception
Synkinesis (associated reactions)	

3.5 Patterns of Weakness in UMN

Upper Limb: Flexors are relatively spared; extensors are weak → characteristic posture: shoulder adduction/IR, elbow flexion, wrist flexion, finger flexion ("hemiplegic posture").

Lower Limb: Extensors relatively spared; flexors are weak → hip/knee extension preserved, foot drop (weak dorsiflexors), circumduction gait.

Pyramidal Distribution Mnemonic: WEST-FLEX

- W — Wrist extensors (weak in UE)
- E — Elbow extensors (weak in UE)
- S — Shoulder abductors (weak in UE)
- T — Triceps (weak in UE)
- F — Foot dorsiflexors (weak in LE)
- L — Leg flexors/hamstrings (weak in LE)
- E — Evertors of foot (weak in LE)
- X — (Extensors of UE + Flexors of LE are relatively preserved)

3.6 Important UMN Signs in Detail

Sign	Method	Significance
Babinski Sign	Stroke sole of foot lateral to medial; +ve = great toe dorsiflexion + fanning of other toes	Indicates UMN lesion; absent in infants <12 months (UMN not fully myelinated)
Clonus	Sustained rapid stretching of muscle; +ve = rhythmic oscillations >3 beats (sustained clonus)	Ankle clonus most common; indicates UMN lesion with severe spasticity
Hoffman Sign	Flick terminal phalanx of middle finger; +ve = flexion of thumb and index finger	Indicates cervical myelopathy or bilateral UMN lesion
Tromner Sign	Tap volar aspect of middle finger; same significance as Hoffman	UMN lesion above C5
Lhermitte Sign	Neck flexion → electric shock-like sensation down spine	Indicates cervical cord demyelination (MS)
Jaw Jerk	Tap chin with mouth slightly open; +ve = exaggerated jaw closure	Lesion above pons (pseudobulbar palsy)
Clasp-knife	Resistance then sudden give on passive muscle stretch	Hyperactive inverse stretch reflex (Golgi tendon organ)

CHAPTER 4

Lower Motor Neuron (LMN) Lesions — In Detail

4.1 Components of LMN

Component	Location	Disease Examples
Anterior horn cell (AHC)	Spinal cord gray matter	Poliomyelitis, SMA, ALS
Nerve root	Exit from spinal cord	Disc prolapse, herpes zoster
Peripheral nerve	Mixed motor/sensory	Peripheral neuropathy, mononeuropathy
Neuromuscular junction (NMJ)	Synapse between nerve and muscle	Myasthenia gravis, Lambert-Eaton
Muscle	Effector organ	Muscular dystrophy, myopathy

4.2 Pathophysiology of LMN Lesion

LMN is the FINAL COMMON PATHWAY for all motor commands. Disruption at any point in the LMN circuit causes denervation of muscle fibers.

- Wallerian degeneration: Axon distal to injury degenerates within 3-7 days
- Loss of trophic influences from nerve → muscle atrophy begins within 2-3 weeks
- Surviving motor neurons attempt sprouting (collateral reinnervation)
- Fibrillations: Spontaneous action potentials from individual muscle fibers due to ACh receptor hypersensitivity (Cannon's law of denervation)
- Fasciculations: Spontaneous firing of entire motor units (pathological in LMN disease)

4.3 Denervation Changes & Timeline

Timeline of LMN Lesion Changes

- Day 0-3: Wallerian degeneration begins distally; NCV still normal
- Day 3-7: NCV reduces distal to lesion; EMG shows decreased MUAP recruitment
- Week 1-3: Fibrillation potentials and positive sharp waves appear on EMG
- Week 2-4: Visible muscle wasting begins; fasciculations may appear
- Month 1-3: Collateral sprouting by surviving axons (enlarged MUAP on EMG)
- Month 3+: Reinnervation MUAPs (large amplitude, polyphasic, long duration) if recovery occurs
- Year 1-2: If no reinnervation → irreversible fibrosis of muscle

4.4 Sensory Features in LMN Disease

Since peripheral nerves carry both motor and sensory fibers, LMN lesions often have associated sensory loss:

- Glove-and-stocking pattern: Length-dependent polyneuropathy (diabetic, GBS)
- Dermatomal pattern: Nerve root lesion (disc prolapse)
- Single nerve territory: Mononeuropathy (carpal tunnel, ulnar nerve)
- NO sensory loss: Anterior horn cell disease (polio, SMA) — pure motor

4.5 Classification of LMN Disorders by Site

Site	Diseases	Distinguishing Feature
Anterior Horn Cell	SMA (Type I-IV), Poliomyelitis, ALS (combined UMN+LMN), Kennedy disease	Fasciculations + atrophy; NO sensory loss
Nerve Root (Radiculopathy)	Disc prolapse, spondylosis, foraminal stenosis	Dermatomal pattern; positive Spurling/SLR
Plexus (Plexopathy)	Brachial neuritis (Parsonage-Turner), trauma, radiation	Multiple root territories; complex pattern
Peripheral Nerve (Neuropathy)	GBS, CMT, diabetic neuropathy, leprosy	Distal > proximal; length-dependent
NMJ	Myasthenia gravis, Lambert-Eaton, botulism	Fatigability; normal reflexes initially
Muscle (Myopathy)	Duchenne/Becker MD, inflammatory myopathies	Proximal > distal; CK elevated; reflexes preserved till late

CHAPTER 5

Specific UMN Conditions — Pathophysiology & Features

5.1 Stroke (Cerebrovascular Accident)

Definition: Sudden onset focal neurological deficit due to vascular cause (ischemia or hemorrhage), lasting >24 hours.

- Ischemic stroke (80%): Thrombotic or embolic occlusion of cerebral artery
- Hemorrhagic stroke (20%): Intraparenchymal or subarachnoid hemorrhage
- TACI: Total anterior circulation infarct — MCA + ACA territory
- PACI: Partial anterior circulation infarct
- LACI: Lacunar infarct — small vessel disease, internal capsule, basal ganglia
- POCI: Posterior circulation infarct — brainstem, cerebellum

Clinical Features by Territory:

Territory	Clinical Features
MCA (most common)	Contralateral hemiplegia (arm > leg), hemisensory loss, homonymous hemianopia, aphasia (dominant), neglect (non-dominant)
ACA	Contralateral leg > arm weakness, urinary incontinence, abulia, grasp reflex
PCA	Contralateral hemianopia, thalamic pain syndrome, Weber syndrome (ipsilateral CN III palsy + contralateral hemiplegia)
Basilar artery	Locked-in syndrome, quadriplegia, bulbar palsy, coma
PICA	Wallenberg (lateral medullary) syndrome: ipsilateral facial sensory loss, Horner, cerebellar ataxia; contralateral body sensory loss
Internal capsule (lacunar)	Pure motor hemiplegia (posterior limb), pure sensory stroke (thalamus), ataxic hemiparesis

5.2 Spinal Cord Injury (SCI)

Classification: By ASIA Impairment Scale (AIS)

ASIA Grade	Description
A (Complete)	No motor or sensory function below the level of injury, including S4-S5
B (Sensory)	Sensory but NOT motor function preserved below level, including S4-S5
C (Motor)	Motor function preserved below level; >50% key muscles below level have grade <3
D (Motor)	Motor function preserved below level; >50% key muscles below level have grade ≥3
E (Normal)	Sensory and motor function normal

Key SCI Syndromes:

Syndrome	Features	Notes
Complete transection	All modalities lost below level; UMN below + LMN at level	Paraplegia or quadriplegia
Anterior Cord Syndrome	Loss of motor (CST) + pain/temp (STT); proprioception PRESERVED	Worst prognosis; hyperflexion injury
Central Cord Syndrome	Arms > legs weakness; bladder dysfunction; variable sensory	Most common; older patients with cervical spondylosis + hyperextension

Brown-Sequard Syndrome	Ipsilateral motor (CST) + proprioception loss; Contralateral pain/temp loss	Hemisecution; best prognosis
Posterior Cord Syndrome	Loss of proprioception/vibration; motor PRESERVED	Sensory ataxia; rare; posterior column affected
Cauda Equina Syndrome	LMN pattern ALL; saddle anaesthesia; bladder/bowel LMN	Emergency; surgical decompression required
Conus Medullaris Syndrome	Mixed UMN+LMN; bladder/bowel at L1-L2 level	Bulbocavernous reflex absent then returns

5.3 Multiple Sclerosis (MS)

Pathophysiology: Autoimmune demyelination of CNS white matter → conduction block and axonal loss. Multiple lesions disseminated in space and time. Oligoclonal bands in CSF.

- Types: RRMS (85%), PPMS, SPMS, PRMS
- Common symptoms: Optic neuritis, Uhthoff phenomenon (worsening with heat), Lhermitte sign, spasticity, fatigue, ataxia, bladder dysfunction
- McDonald Criteria 2017: Dissemination in space + time on MRI for diagnosis
- Charcot Triad: Nystagmus + Intention tremor + Scanning dysarthria (cerebellar MS)
- EDSS scale: 0-10 for disability assessment in MS

5.4 Cerebral Palsy (CP)

Definition: Group of permanent disorders of movement/posture due to non-progressive disturbance in developing fetal or infant brain.

Type	Features	% of CP
Spastic (UMN)	Most common; hypertonia, hyperreflexia, Babinski +ve; diplegia/hemiplegia/quadruplegia	70-80%
Dyskinetic/Athetoid	Extrapyramidal; athetosis (writhing), dystonia, choreoathetosis	10-15%
Ataxic	Cerebellar; hypotonia, dysmetria, poor balance	5-10%
Mixed	Combination of above types	Variable

CHAPTER 6

Specific LMN Conditions — In Detail

6.1 Guillain-Barré Syndrome (GBS)

Pathophysiology: Acute inflammatory demyelinating polyradiculoneuropathy (AIDP). Molecular mimicry: Antibodies against *Campylobacter jejuni* cross-react with gangliosides (GM1, GQ1b) on peripheral nerve myelin. T-cell mediated attack on myelin and axons.

- Triggers: *Campylobacter jejuni* (most common), CMV, EBV, Zika virus, COVID-19 vaccination (rare)
- Onset: Ascending flaccid paralysis, areflexia, albuminocytologic dissociation in CSF (high protein, normal cells)
- Brighton Criteria: Bilateral flaccid limb weakness + decreased/absent DTRs + onset <4 weeks
- Variants: AIDP (demyelinating), AMSAN/AMAN (axonal), MFS (Miller Fisher: ophthalmoplegia, ataxia, areflexia)
- Treatment: IVIG or plasmapheresis; NOT steroids; supportive care, ventilatory support

Albuminocytologic Dissociation

- CSF protein: Elevated (>45 mg/dL, often >100 mg/dL) — peaks at 4-6 weeks
- CSF cells: Normal (<10 cells/mm³)
- This dissociation is PATHOGNOMONIC of GBS
- NCV: Reduced conduction velocity + prolonged F-wave latency (early finding)

6.2 Poliomyelitis

Pathophysiology: Poliovirus (enterovirus) infects anterior horn cells (AHC) of spinal cord and brainstem motor neurons. Preferentially destroys AHCs → irreversible LMN lesion.

- Tropism: Virus attaches to CD155 receptor on motor neurons
- Clinical: Asymmetric flaccid paralysis with fever, NO sensory loss, NO cognitive involvement
- Post-polio syndrome: New weakness, fatigue 30-40 years later due to motor unit exhaustion
- Bulbar polio: CN IX, X, XII nuclei → dysphagia, respiratory failure
- Orthopaedic complications: Foot drop, scoliosis, limb length discrepancy, joint deformity

6.3 Bell's Palsy (Facial Nerve Palsy)

Pathophysiology: Idiopathic peripheral facial nerve (CN VII) palsy. Viral reactivation (HSV-1) → edema → compression in narrow facial canal → LMN facial palsy.

LMN vs UMN Facial Palsy Distinction (HIGH YIELD):

Feature	LMN Facial Palsy (Bell's)	UMN Facial Palsy (Stroke)
Forehead sparing	NO — forehead affected (wrinkles absent)	YES — forehead SPARED (bilateral cortical representation)
Eye closure	Cannot close eye (Bell's phenomenon)	Can close eye (weakly)
Taste	Loss of taste (ant. 2/3 tongue) if chorda tympani involved	Usually intact
Lacrimation	May be affected	Usually intact
Grading	House-Brackmann scale (I-VI)	Not applicable
Cause	Bell's palsy, parotid tumor, Ramsay Hunt	Stroke, brain tumor

6.4 Peripheral Nerve Injuries

Seddon Classification:

Type	Pathology	Prognosis
Neuropraxia	Focal demyelination; axon intact	Conduction block; NCV reduced across lesion; full recovery expected 6-12 weeks
Axonotmesis	Axon disrupted; endoneurium intact	Wallerian degeneration; recovery possible at 1mm/day via intact sheath
Neurotmesis	Complete nerve disruption	No spontaneous recovery; needs surgical repair; worst prognosis

Sunderland Classification (5 grades): I=neuropraxia, II=axonotmesis (endoneurium intact), III=endoneurium disrupted (perineurium intact), IV=perineurium disrupted (epineurium intact), V=neurotmesis

6.5 Common Peripheral Nerve Injuries

Nerve	Features	Key Notes
Radial nerve (C5-T1)	Wrist drop (extensor carpi radialis brevis), finger drop; loss of thumb abduction/extension	Posterior interosseous nerve (PIN) syndrome
Ulnar nerve (C8-T1)	Claw hand (ring/little finger), hypothenar wasting, interossei wasting; Froment sign	Cubital tunnel, Guyon canal
Median nerve (C6-T1)	Ape hand (thenar atrophy), loss of opposition/pinch; carpal tunnel (sensory loss 3.5 fingers)	Carpal tunnel syndrome most common
Common peroneal (L4-S1)	Foot drop (dorsiflexion/eversion lost); sensory loss dorsum foot; steppage gait	Fibular head injury
Femoral nerve (L2-L4)	Quadriceps weakness, knee extension lost; sensory loss medial leg	Psoas hematoma, hip arthroplasty
Sciatic nerve (L4-S3)	Combined peroneal + tibial features; complete foot drop + plantar flexion loss	Hip dislocation, injection injury
Long thoracic nerve (C5-C7)	Winging of scapula (serratus anterior palsy)	Surgical trauma, backpack palsy
Axillary nerve (C5-C6)	Deltoid palsy; loss of shoulder abduction; sensory loss badge area	Shoulder dislocation, surgical neck fracture of humerus

CHAPTER 7

Neurological Assessment Techniques

7.1 Systematic Neurological Examination Approach

ORDER: Higher functions → Cranial nerves → Motor → Sensory → Coordination → Gait → Autonomic

7.2 Motor Assessment

MRC Grading of Muscle Power:

Grade	Description	Clinical Meaning
0	No contraction	Complete paralysis
1	Flicker/trace contraction visible or palpable	No movement
2	Active movement with gravity eliminated	Movement in horizontal plane
3	Active movement against gravity	Minimum functional level
4-	Active movement against slight resistance	Impaired strength
4	Active movement against moderate resistance	Still weakness
4+	Active movement against strong resistance	Near normal
5	Normal power	Full strength against full resistance

7.3 Tone Assessment

- Pendulum test: Passive elevation and release of limb; abnormal oscillation in spasticity
- Modified Ashworth Scale (MAS): Grades 0-4 for spasticity
- Tardieu Scale: Tests at 3 velocities (V1 slow, V2 gravity, V3 fast); more specific than MAS
- Hypertonia Assessment Tool (HAT): Distinguishes spasticity vs dystonia vs rigidity

Distinguishing Tone Abnormalities:

Type	Characteristics	Cause
Spasticity	Velocity-dependent; clasp-knife; pyramidal distribution	Stroke, SCI, MS, CP — UMN
Rigidity	Velocity-independent; cogwheel (Parkinson) or lead-pipe; affects flexors+extensors equally	Parkinson's disease; extrapyramidal
Dystonia	Sustained muscle contractions; abnormal postures; not velocity-dependent	Extrapyramidal; may be focal or generalized
Paratonia (Gegenhalten)	Variable resistance that increases with speed of movement; diffuse frontal lobe	Frontal lobe dementia, metabolic encephalopathy

7.4 Reflexes Assessment

Grading: 0=absent, 1+=hyporeflexia, 2+=normal, 3+=hyperreflexia, 4+=clonus

Superficial Reflexes (Absent in UMN Disease)

- Abdominal reflex: T7-T9 (upper), T10-T12 (lower) — absent in UMN lesion and MS
- Cremasteric reflex (male): L1,L2 — absent in UMN above L1 or LMN L1,L2

- Plantar reflex: S1,S2 — Babinski +ve in UMN; Flexor in normal adults
- Bulbocavernous reflex: S3,S4 — absent in sacral cord/conus lesion

7.5 Sensory Assessment

- Touch: Cotton wool; assess dermatomal patterns
- Pain/Temperature: Pin prick / hot-cold tubes; spinothalamic tract
- Vibration: 128 Hz tuning fork; posterior column
- Proprioception (JPS): Passive movement of fingers/toes; posterior column
- Two-point discrimination: Cortical sensation; primary somatosensory cortex
- Stereognosis: Identifying objects by touch; parietal lobe
- Graphesthesia: Number/letter on palm; parietal lobe

7.6 Coordination Tests

Test	Method	Significance
Finger-Nose Test	Ask to touch nose then examiner's finger repeatedly	Dysmetria, intention tremor → cerebellar
Diadochokinesis	Rapid alternating supination/pronation of hand	Dysdiadochokinesia → cerebellar/ipsilateral
Heel-Shin Test	Run heel along shin from knee to ankle	Cerebellar ataxia of lower limb
Romberg Test	Stand feet together, eyes open then closed; positive = falls with eyes closed	Positive = posterior column / vestibular (NOT cerebellar)
Tandem Walking	Walk heel-to-toe in straight line	Cerebellar vermis pathology
Fukuda Stepping Test	March on spot eyes closed; >45° rotation indicates labyrinthine dysfunction	Vestibular lesion

7.7 Gait Analysis

Gait Pattern	Cause	Characteristics
Hemiplegic/Circumduction gait	Stroke (UMN hemiplegia)	Leg circumduction; arm held in flexion; foot drop
Steppage gait	LMN: foot drop (peroneal nerve, radiculopathy)	High stepping to clear toes; unable to heel walk
Scissor gait	Bilateral spastic CP, paraparesis	Legs cross midline; hip adductor spasticity
Waddling gait	Hip abductor weakness (proximal myopathy, DDH)	Trunk sways toward stance leg; Trendelenburg +ve
Festinant gait	Parkinson's disease	Short steps, shuffling, reduced arm swing, forward lean, freezing
Ataxic/Broad-based gait	Cerebellar lesion	Wide-based, staggering, veering to side of lesion
Antalgic gait	Pain (hip/knee)	Short stance phase on affected side
Trendelenburg gait	Gluteus medius weakness	Pelvis drops to opposite side during stance

CHAPTER 8

Physiotherapy Interventions — UMN Conditions

8.1 Goals of Physiotherapy in UMN Lesions

- Reduce spasticity and prevent complications (contractures, pressure sores)
- Facilitate voluntary movement and motor recovery
- Improve functional independence (ADL, transfers, mobility)
- Restore normal movement patterns and prevent abnormal synergies
- Strengthen weak muscles in pyramidal distribution
- Improve balance, coordination, and gait
- Prevent DVT, respiratory complications, pain

8.2 Management of Spasticity

Physiotherapy Modalities for Spasticity:

Modality	Method	Mechanism
Positioning & Splinting	Maintain muscles at lengthened position; anti-spastic positioning; serial casting; resting splints	Prevents contractures; maintains ROM; reduces reflex activity
Passive Stretching	Slow, sustained stretch 20-30 seconds; 3-5 reps; 2x daily	Reflexive relaxation; temporary spasticity reduction
Cryotherapy	Ice packs/massage; cold water immersion; Clinically: wrap limb in towel soaked in ice water for 10-15 min before treatment	Reduces gamma motor neuron activity; beneficial for 20-45 min post application
Heat (CAUTION)	Superficial heat in some cases; Uhthoff phenomenon in MS means AVOID heat	Use with care; monitor for increased spasticity
TENS/FES	TENS for reciprocal inhibition; FES for voluntary movement facilitation	Electrical stimulation of antagonists to inhibit spastic agonists via reciprocal inhibition
Vibration	Tonic vibration reflex (TVR); apply to antagonist muscle	Inhibits spastic muscle via reciprocal Ia inhibition
Biofeedback	EMG biofeedback to reduce spastic muscle activity	Self-regulation; improves motor control
Hydrotherapy	Warm water (33-36°C) for MS patients only if Uhthoff not a problem; reduces gravitational load	Buoyancy assists movement; reduces weight-bearing stress

8.3 Task-Specific Training for Stroke

- Constraint-Induced Movement Therapy (CIMT): Restrain unaffected arm; intensive practice of affected arm 6 hrs/day x 2 weeks; evidence level IA
- Repetitive Task Training: Repetitive practice of functional tasks (reaching, grasping); drives neuroplasticity
- Mental Practice/Motor Imagery: Imagining movement activates same cortical areas as actual movement; adjunct to physical training
- Mirror Therapy: Mirror placed sagittally; reflects unaffected arm movement; tricks brain into "seeing" affected arm move; useful for hand function and CRPS
- Treadmill Training (with/without body weight support): Repetitive stepping pattern; activates CPG; evidence for gait improvement post stroke
- Robotics/Technology-Assisted Therapy: Robot-assisted upper and lower limb training; allows high repetitions with reduced therapist effort

8.4 Balance and Postural Training

- Static balance: Sitting on unstable surface (foam, wobble board); quiet standing
- Dynamic balance: Perturbations; reaching tasks; sitting reach test; functional reach test
- BBS (Berg Balance Scale): 14 items; max 56; <45 = fall risk; widely used
- Proprioceptive training: Eyes closed standing; single leg stance; proprioceptive taping
- Vestibular rehabilitation: Cawthorne-Cooksey exercises; gaze stabilization
- Core stabilization: Improve trunk control; prerequisite for limb function

CHAPTER 9

Physiotherapy Interventions — LMN Conditions

9.1 Goals in LMN Lesions

- Prevent denervation atrophy and maintain muscle bulk
- Maintain joint ROM and prevent contractures
- Prevent trophic changes (skin, nail care)
- Facilitate nerve regeneration and reinnervation
- Strengthening during reinnervation phase
- Functional rehabilitation and orthotic management
- Manage pain (neuropathic, musculoskeletal)

9.2 Electrotherapy in LMN Denervation

Electrical Stimulation (ES) for Denervated Muscle:

Chronaxie and Rheobase

- Rheobase: Minimum current to cause contraction with infinitely long pulse duration
- Chronaxie: Duration of pulse at TWICE the rheobase intensity needed to cause contraction
- Normal muscle: Chronaxie = 0.05-1.0 ms; Denervated muscle: Chronaxie >1 ms (up to 100 ms)
- Strength-Duration (SD) Curve: Plot of intensity vs pulse duration; deviation to right = denervation
- Galvanic stimulation: Long pulse duration (>10ms) required for denervated muscle (faradic stimulation is ineffective)

- Faradic current: Short pulse duration (0.1-1 ms); only stimulates INNERVATED muscle
- Galvanic/Direct current: Long pulse duration (>10 ms); stimulates DENERVATED muscle
- Interrupted DC (IDC): For maintaining denervated muscle; prevents atrophy; 10-20 mA; 0-1 Hz
- TENS: For pain management in peripheral neuropathy; conventional TENS 80-100 Hz, 50-200 μ s
- FES: Once reinnervation begins; facilitates functional movement

9.3 Progressive Resistance Exercise in LMN Recovery

As nerve regeneration occurs (1 mm/day), progressive strengthening program:

- Stage 0-1 (MRC 0-1): Passive movements, electrical stimulation, positioning, AROM assisted by gravity eliminated
- Stage 2 (MRC 2): Active gravity-eliminated exercises; hydrotherapy; AROM assisted
- Stage 3 (MRC 3): Active against gravity; closed kinetic chain exercises; aquatic therapy
- Stage 4 (MRC 4): Progressive resistance training; 3 sets of 10; increase 10% per week (DeLorme or DAPRE protocols)
- Stage 5 (MRC 5): Sport-specific/occupation-specific training; endurance training; functional return

9.4 Orthotic Management in LMN

Condition	Orthosis	Notes
Foot drop (peroneal palsy)	AFO (Ankle Foot Orthosis) — solid or articulated; drop foot splint	Cock-up splint with foot drop correction
Wrist drop (radial nerve)	Cock-up splint; wrist extension orthosis	Thomas splint (plaster of Paris for acute phase)

GBS / polio (acute)	Full body positioning; pressure relief; splints to prevent deformity	Thoracolumbosacral orthosis (TLSO) for trunk support
Claw hand (ulnar)	Reverse knuckle bender splint; anti-claw splint	Maintains MCP flexion while IPs can extend
Facial nerve palsy	Tape for eye closure; facial sling	Protect cornea; ptosis prop

CHAPTER 10

Exercise Therapy in Detail

10.1 Bobath/Neurodevelopmental Therapy (NDT)

Principles: Facilitate normal movement; inhibit abnormal patterns; use handling and key points of control.

- Key Points of Control (KPC): Proximal (shoulder, hip, trunk) for gross movement; distal (hand, foot) for fine
- Reflex Inhibiting Postures (RIP): Positions that inhibit spasticity and abnormal reflexes
- Facilitatory Techniques: Tapping, stretch, approximation to facilitate weak muscles
- Inhibitory Techniques: Slow rotation, passive elongation, weight bearing on affected limb

10.2 Proprioceptive Neuromuscular Facilitation (PNF)

Diagonal Patterns: D1 and D2 for upper and lower limbs; work in diagonal planes.

Pattern	Components	Functional Example
UL D1 Flexion	Shoulder: Flex, Adduct, ER; Elbow: Flex; Wrist/Fingers: Flex, Radial deviation	Feeding, combing hair
UL D1 Extension	Shoulder: Ext, Abduct, IR; Elbow: Ext; Wrist/Fingers: Ext, Ulnar deviation	Pushing away from face
UL D2 Flexion	Shoulder: Flex, Abduct, ER; Elbow: Ext; Wrist/Fingers: Ext, Radial deviation	Tennis backhand
UL D2 Extension	Shoulder: Ext, Adduct, IR; Elbow: Flex; Wrist/Fingers: Flex, Ulnar deviation	Golf swing
LL D1 Flexion	Hip: Flex, Adduct, ER; Knee: Flex; Ankle: DF, Inversion	Kicking soccer ball
LL D2 Flexion	Hip: Flex, Abduct, IR; Knee: Flex; Ankle: DF, Eversion	Hiking motion

PNF Techniques:

Technique	Description	Indication
Rhythmic Initiation (RI)	Passive → active assist → active; builds motivation and coordination	Initiating movement in rigid/apraxic patients
Repeated Contractions (RC)	Repeated stretch and resistance through range; stretch at point of weakness	Strengthening throughout range
Slow Reversal (SR)	Alternating agonist-antagonist; isotonic concentric; promotes reciprocal movement	Gait, trunk rotation
Slow Reversal Hold (SRH)	SR + isometric hold at end range	Improving stability at end range
Contract-Relax (CR)	Isometric contraction of tight muscle → relaxation → stretch; for ROM improvement	Hamstring tightness, spasticity
Hold-Relax (HR)	Isometric contraction at barrier then relax then passive stretch	Pain-limited range; preferred when active movement painful
Rhythmic Stabilization (RS)	Simultaneous isometric contraction of agonist+antagonist; no movement; improves stability	Trunk stability, proximal stability
Combination of Isotonics (CI)	Concentric → eccentric → isometric in sequence without relaxing	Functional strengthening

10.3 Motor Relearning Programme (MRP) — Carr & Shepherd

- Based on motor learning theory; task-oriented approach
- 4 Steps: (1) Analysis of task and missing components → (2) Practice of missing components → (3) Practice of full task → (4) Transfer of training
- Emphasizes: Specific practice, feedback, mental practice, self-monitoring
- 7 functional tasks: Up from supine → Sitting balance → Sit to stand → Standing up → Walking → Upper limb function → Orofacial function

10.4 Core Stabilization Exercises

Critical for trunk control as prerequisite for limb function in both UMN and LMN conditions.

Exercise	Method	Purpose
Transversus abdominis (TrA) activation	Draw-in maneuver; "Hollow your belly"; 10 sec hold x 10 reps	Activates deepest core; reduces lumbar instability
Multifidus activation	Co-contraction with TrA; prone on elbows; bird dog (alternate arm-leg)	Segmental spinal stabilization
Dead bug exercise	Supine; arms/legs raised; alternate arm-leg lowering	Dissociation; core stability with limb movement
Plank (modified)	Forearm plank; progress to full plank; side plank	Global core endurance
Bridging	Supine hook lying; lift pelvis; progress to single-leg bridge	Gluteal + core activation; functional for transfers
Sit to Stand	Sit at edge; shift forward; stand without arm support	Highly functional; key in stroke rehabilitation

CHAPTER 11

Neuro-Rehabilitation Techniques

11.1 Constraint-Induced Movement Therapy (CIMT)

- Indication: Chronic stroke with minimal arm function (MRC ≥ 3 wrist ext, $\geq 10^\circ$ finger ext)
- Protocol: Mitt/sling on unaffected arm for 90% waking hours x 2-3 weeks; 6 hours/day shaping & task practice of affected arm
- Mechanism: Overcomes "learned non-use" (Taub); promotes cortical reorganization
- Evidence: Level IA for upper limb function in chronic stroke; EXCITE trial
- Modified CIMT (mCIMT): Constraint 5 hrs/day + 30 min therapy; better compliance

11.2 Virtual Reality (VR) and Gaming Rehabilitation

- Mechanisms: Augmented feedback, repetition, engagement, motivation
- Evidence: Nintendo Wii, Xbox Kinect, specialized VR systems; moderate evidence for upper limb
- Advantages: High repetition, adjustable difficulty, performance feedback, motivating
- Applications: Balance training, upper limb function, gait, cognitive-motor dual task

11.3 Body Weight Supported Treadmill Training (BWSTT)

- Harness supports 0-40% body weight over treadmill; manual facilitation of limb movement
- Activates spinal Central Pattern Generators (CPGs) for locomotion
- Evidence: SCIRE systematic review — moderate evidence for SCI; moderate for stroke
- Progression: Reduce body weight support; increase treadmill speed; progress to over-ground
- Robotic-assisted: Lokomat (exoskeleton); allows high repetitions with precise gait patterns

11.4 Functional Electrical Stimulation (FES)

- Definition: ES to elicit functional movements; contractions during task performance
- Neuroprosthetics: FES systems for foot drop (Bioness L300), hand function (Bioness H200)
- Mechanism: Strengthens weak muscles; provides sensory feedback; reduces spasticity via reciprocal inhibition
- FES cycling: Electrically stimulates leg muscles for pedaling; cardiovascular + motor benefits
- Therapeutic FES: Short-term treatment benefit; vs Orthotic FES: Permanent use as orthosis

11.5 Electromyographic Biofeedback (EMG-BF)

- Surface EMG signal amplified and fed back to patient as audio/visual signal
- Enables voluntary control of muscle activity; particularly useful for partial reinnervation
- Applications: Reducing spastic muscle activity (antagonist biofeedback) or facilitating weak muscles
- Evidence: Moderate evidence for ankle dorsiflexion in stroke; Grade B

11.6 Transcranial Magnetic Stimulation (TMS) & tDCS

- TMS: Magnetic pulses to motor cortex; rTMS (repetitive TMS) modulates cortical excitability
- Inhibitory rTMS (1Hz) to contralesional hemisphere or excitatory (10Hz) to lesional hemisphere → both improve motor function
- tDCS (transcranial DC stimulation): Low-level DC current; anodal (excitatory) or cathodal (inhibitory)
- Evidence: Emerging; adjunct to conventional therapy; Grade C recommendation

11.7 Proprioceptive Training Techniques

Technique	Method	Indication
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Joint Position Sense (JPS) retraining	Active/passive reproduction tasks; eyes open then closed	Posterior column / peripheral nerve lesion
Frenkel's Exercises	Graduated coordination exercises for ataxia; lying → sitting → standing → walking (15 exercises)	Posterior column ataxia (tabes dorsalis, MS)
Cawthorne-Cooksey exercises	Eye, head, body movements; in bed → sitting → standing; for vestibular rehabilitation	Vestibular hypofunction, labyrinthitis
Balance training on unstable surfaces	Foam pads, BOSU, wobble board; eyes open then closed	Vestibular, proprioceptive, cerebellar training
Rocker board exercises	Weight shifts; single leg stance; perturbation	Ankle proprioception, LMN recovery

11.8 Brunnstrom Stages of Recovery (Post Stroke)

Stage	Features	PT Goal
Stage 1	Flaccidity; no voluntary movement; areflexia	Flaccid; passive movements; positioning
Stage 2	Spasticity developing; synergy patterns emerge; no voluntary control	Prevent contractures; stimulate movement
Stage 3	Voluntary movement in synergy only; spasticity peaks	Facilitation within synergies; positioning
Stage 4	Some movement outside synergy; spasticity decreasing	Begin breaking out of synergy; task practice
Stage 5	Movement more independent of synergy; spasticity minimal	Task-specific training; selective movements
Stage 6	Individual joint movement; coordination near normal; minimal spasticity	Advanced ADL; fine motor; speed training
Stage 7	Normal movement	Sport/work-specific; maintenance

CHAPTER 12

MCQ Bank — 100+ Questions with Answers & Explanations

12.1 Anatomy & Physiology

Q1. Which of the following is the level of decussation of the corticospinal tract?

- (A) Pons
- (B) Upper medulla
- ✓ (C) Lower medulla (pyramidal decussation)
- (D) Mid-cervical spinal cord

Explanation: The pyramidal decussation occurs at the junction of medulla and spinal cord (lower medulla). 85-90% fibers cross here.

Q2. The posterior limb of the internal capsule carries which fibers?

- ✓ (A) Corticospinal motor fibers
- (B) Corticobulbar fibers to upper cranial nerves
- (C) Frontopontine fibers
- (D) Thalamocortical sensory fibers

Explanation: Posterior limb carries CST (motor fibers). Genu carries corticobulbar fibers. Anterior limb carries frontopontine fibers.

Q3. Alpha motor neurons are located in which region of the spinal cord?

- (A) Posterior horn
- (B) Intermediate zone
- ✓ (C) Anterior horn
- (D) Lateral horn

Explanation: Alpha motor neurons (large) and gamma motor neurons (small) are both in the anterior horn (anterior gray matter).

Q4. Chronaxie of a denervated muscle is:

- (A) Less than 1 ms
- (B) 0.5-1 ms
- ✓ (C) 1-100 ms (increased)
- (D) Unchanged

Explanation: Denervated muscle has increased chronaxie (>1 ms) because without neural input, membrane excitability decreases and longer pulse durations are needed.

Q5. The Muscle spindle is innervated by which efferent fibers?

- (A) Alpha motor neurons
- ✓ (B) Gamma motor neurons
- (C) Beta motor neurons
- (D) Ia afferents

Explanation: Gamma motor neurons (fusimotor) innervate intrafusal fibers of muscle spindle, maintaining its sensitivity during muscle contraction.

Q6. Which tract is responsible for pain and temperature sensation?

- (A) Dorsal columns (fasciculus gracilis/cuneatus)
- (B) Lateral corticospinal tract
- ✓ (C) Spinothalamic tract
- (D) Spinocerebellar tract

Explanation: Anterior spinothalamic: light touch; Lateral spinothalamic: pain and temperature. Both cross at spinal cord level.

Q7. The clasp-knife phenomenon seen in UMN lesion is due to:

- (A) Hyperactive Ia stretch reflex
- ✓ (B) Hyperactive Golgi tendon organ (inverse stretch reflex)
- (C) Increased gamma drive
- (D) Increased alpha motor neuron excitability

Explanation: Clasp-knife = initial high resistance then sudden release = hyperactive inverse stretch reflex (Ib afferents from GTOs).

Q8. Wallerian degeneration refers to:

- ✓ (A) Anterograde degeneration of axon distal to injury

(B) Retrograde degeneration of axon proximal to injury

(C) Demyelination only without axon loss

(D) Selective loss of anterior horn cells

Explanation: Wallerian degeneration = anterograde (distal) degeneration of axon and myelin after injury. Begins 3-7 days post-injury.

12.2 UMN Signs & Diagnosis

Q9. Babinski sign is characterized by:

(A) Plantar flexion of great toe + toe fanning

✓ (B) Dorsiflexion of great toe + fanning of other toes

(C) Knee jerks absent

(D) Ankle clonus only

Explanation: Babinski = extension (dorsiflexion) of great toe + fanning (abduction) of other toes = positive = UMN lesion. Normal = toe flexion.

Q10. Which of the following is NOT a feature of UMN lesion?

(A) Spasticity

(B) Hyperreflexia

✓ (C) Fasciculations

(D) Babinski sign +ve

Explanation: Fasciculations are pathognomonic of LMN disease (spontaneous firing of entire motor units). NOT seen in UMN lesion.

Q11. Lhermitte sign indicates involvement of:

(A) Posterior cranial fossa

✓ (B) Cervical spinal cord posterior columns

(C) Lumbar cord

(D) Peripheral nerves

Explanation: Lhermitte sign (electric shock on neck flexion) = demyelination of cervical cord posterior columns. Classic in MS.

Q12. In a patient with stroke, forehead is spared in facial palsy because:

(A) Facial nerve has two divisions

✓ (B) Frontalis receives bilateral cortical input (ipsi + contralateral)

(C) Forehead is supplied by CN V

(D) The lesion is in the pons

Explanation: Upper face (frontalis/orbicularis oculi) receives bilateral cortical representation; so contralateral stroke spares forehead. Lower face is only contralaterally controlled.

Q13. Which of the following best describes central cord syndrome?

✓ (A) Arm weakness > leg weakness; bladder dysfunction

(B) Leg weakness > arm weakness; proprioception lost

(C) Ipsilateral motor, contralateral sensory loss

(D) Complete loss of all modalities below injury

Explanation: Central cord: Cervical cord center more affected → arm fibers medial in CST → arm > leg weakness. Also: bladder dysfunction, saddle sensory disturbance.

Q14. Modified Ashworth Scale grade "1+" indicates:

(A) No increase in tone

(B) Slight catch throughout full ROM

✓ (C) Catch then minimal resistance in less than 50% of ROM

(D) Rigid limb

Explanation: MAS 1+ = catch followed by minimal resistance through less than half the range. Grade 1 = catch and release only.

Q15. Brown-Sequard syndrome is due to:

(A) Anterior cord infarction

(B) Complete cord transection

✓ (C) Hemisection of spinal cord

(D) Central cord lesion

Explanation: Brown-Sequard = spinal cord hemisection → ipsilateral CST (motor) + dorsal column loss; contralateral spinothalamic (pain/temp) loss.

Q16. Pseudobulbar palsy is a UMN lesion of:

(A) CN IX, X, XI nuclei (bilateral LMN)

✓ (B) Bilateral UMN lesion above the pons affecting bulbar motor nuclei

(C) Unilateral cortical lesion

(D) Peripheral cranial nerves IX, X, XII

Explanation: Pseudobulbar = bilateral UMN lesion above pons → dysarthria, dysphagia, emotional lability, brisk jaw jerk. Contrasted with bulbar palsy (LMN of CN IX, X, XII).

12.3 LMN Signs & Nerve Injuries

Q17. Albuminocytologic dissociation in CSF is characteristic of:

- (A) Bacterial meningitis
- (B) Viral meningitis
- ✓ (C) **Guillain-Barré Syndrome**
- (D) Multiple sclerosis

Explanation: GBS: High CSF protein (due to inflamed nerve roots) + normal cells = albuminocytologic dissociation. Bacterial meningitis has high cells + high protein.

Q18. Froment sign tests which nerve?

- (A) Radial nerve
- (B) Median nerve
- ✓ (C) **Ulnar nerve**
- (D) Musculocutaneous nerve

Explanation: Froment sign = weakness of thumb adduction (adductor pollicis = ulnar nerve). Patient flexes IP joint of thumb to compensate when gripping paper (median nerve FPL takes over).

Q19. Which is the earliest EMG finding in peripheral nerve injury?

- (A) Fibrillation potentials
- (B) Positive sharp waves
- ✓ (C) **Decreased MUAP recruitment**
- (D) Polyphasic MUAPs

Explanation: Decreased MUAP recruitment (fewer firing motor units) is earliest EMG finding. Fibrillations/PSW appear 1-3 weeks after denervation.

Q20. Foot drop due to common peroneal nerve palsy shows weakness of:

- (A) Plantarflexion and inversion
- ✓ (B) **Dorsiflexion and eversion**
- (C) Dorsiflexion and inversion
- (D) Only inversion

Explanation: Common peroneal = deep peroneal (dorsiflexion + inversion) + superficial peroneal (eversion). Net: Dorsiflexion + Eversion lost = foot drop with inversion (tibialis anterior partially spared).

Q21. Winging of scapula is due to palsy of which nerve?

- (A) Axillary nerve
- (B) Suprascapular nerve
- ✓ (C) **Long thoracic nerve**
- (D) Accessory nerve

Explanation: Long thoracic nerve (C5,6,7) innervates serratus anterior. Palsy → serratus weakness → medial scapular border protrudes ("winged scapula").

Q22. In Neuropraxia, which of the following is CORRECT?

- (A) Axon is disrupted; myelin is intact
- ✓ (B) **Only focal demyelination; axon intact; full recovery expected**
- (C) Endoneurium disrupted; poor recovery
- (D) Complete nerve transection

Explanation: Neuropraxia (Seddon) = Sunderland Grade I = focal demyelination, axon intact. Conduction block; NCV reduced across lesion but normal distal. Full recovery in 6-12 weeks.

Q23. Which of the following is NOT a feature of myasthenia gravis?

- (A) Fatigability
- (B) Improvement with rest
- ✓ (C) **Fasciculations**
- (D) Diplopia and ptosis

Explanation: MG is a NMJ disorder (antibodies against ACh receptors). Features = fatigability, diplopia, ptosis, bulbar symptoms. Fasciculations indicate LMN (anterior horn) disease, NOT NMJ.

Q24. Galvanic stimulation is preferred over faradic in:

- (A) Innervated muscle strengthening
- ✓ (B) **Denervated muscle maintenance**
- (C) Spasticity reduction

(D) Pain relief

Explanation: Faradic (short pulse) stimulates innervated muscle; Galvanic/IDC (long pulse >10ms) is required for denervated muscle because it has slower membrane response.

12.4 Physiotherapy Techniques (HIGH YIELD)

Q25. Brunnstrom stage 3 is characterized by:

- (A) Flaccidity with no voluntary movement
- ✓ (B) Voluntary movement only within synergy
- (C) Movement outside synergy possible
- (D) Normal coordinated movement

Explanation: Stage 3 = spasticity peaks + voluntary movement only possible IN synergy. Stage 4 = movement outside synergy begins. This is a critical stage for PT intervention.

Q26. Which PNF technique is BEST for range of motion improvement?

- (A) Rhythmic Initiation
- (B) Repeated Contractions
- ✓ (C) Contract-Relax
- (D) Rhythmic Stabilization

Explanation: Contract-Relax (CR) and Hold-Relax (HR) are PNF stretching techniques that use autogenic inhibition (post-isometric relaxation) to improve ROM.

Q27. CIMT is indicated in stroke patients with:

- (A) Complete arm paralysis (MRC 0)
- ✓ (B) Minimum wrist extension of 10° and finger extension of 10° (MRC ≥3)
- (C) Flaccid stage
- (D) Only within 6 months of stroke

Explanation: CIMT requires minimal voluntary movement to train (wrist ext ≥10°, finger ext ≥10°). Targets "learned non-use" phenomenon. Evidence Level IA.

Q28. Frenkel exercises are used for:

- (A) Spasticity reduction
- (B) Muscle strengthening
- ✓ (C) Sensory ataxia (posterior column)
- (D) Flaccid paralysis

Explanation: Frenkel's exercises: 15 graded coordination exercises for posterior column ataxia (e.g., tabes dorsalis, MS posterior column involvement). Use visual compensation.

Q29. The goal of tonic vibration reflex (TVR) in physiotherapy is:

- ✓ (A) Facilitate the vibrated muscle (autogenic facilitation)
- (B) Inhibit the vibrated muscle via Ia inhibition
- (C) Reduce pain by gate control
- (D) Improve proprioception only

Explanation: TVR: Vibration of muscle tendon → activates Ia afferents → facilitates that muscle (agonist). Conversely, vibrating antagonist inhibits the spastic agonist. Used in both facilitation and inhibition.

Q30. Which of the following is a sign of LMN lesion at the level of the conus medullaris?

- (A) Hyperreflexia at knee and ankle
- (B) Extensor plantar response
- ✓ (C) Areflexia at ankle with saddle anaesthesia
- (D) Spastic bladder

Explanation: Conus (L1-2): Mixes UMN and LMN signs. Classically: Areflexia (LMN) at ankle, saddle anaesthesia (S3-5), flaccid bladder/bowel. Contrasted with cauda equina (pure LMN, below L2).

Q31. The "D2 flexion" PNF diagonal for upper limb consists of:

- (A) Shoulder flexion, adduction, ER; wrist/fingers flexion
- ✓ (B) Shoulder flexion, abduction, ER; elbow extension; wrist/fingers extension
- (C) Shoulder extension, abduction, IR; wrist/fingers extension
- (D) Shoulder flexion, adduction, IR

Explanation: D2 Flexion UL: Shoulder = Flexion + Abduction + External Rotation; Elbow = Extension; Wrist/Fingers = Extension + Radial deviation. Think "Tennis forehand follow through."

Q32. Modified Ashworth Scale (MAS) was designed to measure:

- (A) Muscle strength
- ✓ (B) Spasticity (velocity-dependent hypertonia)

- (C) Coordination
- (D) Proprioception

Explanation: MAS grades resistance to passive movement (proxy for spasticity). Range 0-4. Important clinical tool for spasticity monitoring post-stroke, SCI, MS, CP.

Q33. Berg Balance Scale score below which indicates HIGH fall risk:

- (A) Below 56
- ✓ (B) Below 45
- (C) Below 36
- (D) Below 28

Explanation: BBS: 14 items; max 56 points. Score <45 associated with significantly increased fall risk (sensitivity ~53%, specificity ~90% for community ambulators). Score <20 = wheelchair bound.

Q34. Central Pattern Generator (CPG) for locomotion is located in:

- (A) Cerebral cortex
- (B) Cerebellum
- ✓ (C) Lumbar spinal cord
- (D) Basal ganglia

Explanation: CPG for locomotion = lumbar spinal cord (L1-L2 region). Generates rhythmic stepping without cortical input. Basis for treadmill training in SCI patients.

Q35. In GBS, which treatment is contraindicated?

- (A) IVIG
- (B) Plasmapheresis
- ✓ (C) Corticosteroids
- (D) Mechanical ventilation when FVC <20ml/kg

Explanation: Corticosteroids are CONTRAINDICATED in GBS (may worsen outcome). Treatment = IVIG or Plasmapheresis. Ventilate when FVC <20ml/kg or declining rapidly.

12.5 Assessment & Special Tests

Q36. Straight Leg Raise (SLR) test is positive in:

- (A) Sacroiliac joint dysfunction
- ✓ (B) L4-L5 or L5-S1 disc prolapse (sciatic nerve root)
- (C) Femoral nerve lesion
- (D) Hip flexor tightness

Explanation: SLR: Passive hip flexion with knee extended → stretches sciatic nerve → positive (radicular pain in leg) at <60° suggests L4-S1 nerve root compression.

Q37. Which nerve root is tested by the Spurling test?

- (A) Thoracic nerve roots
- ✓ (B) Cervical nerve roots (C4-C7)
- (C) Lumbar nerve roots
- (D) Sacral nerve roots

Explanation: Spurling test = cervical compression test. Neck extension + rotation + axial compression → positive if radicular pain in arm. Tests cervical foraminal stenosis/disc prolapse.

Q38. ASIA Grade B means:

- (A) No function below injury level
- ✓ (B) Sensory but NOT motor function preserved below level
- (C) Motor function preserved; most key muscles grade <3
- (D) Normal function

Explanation: ASIA B = Sensory incomplete (some sensation preserved below level including S4-S5) but no motor function below level. Distinguishes from ASIA A (complete).

Q39. Hoffmann sign is tested in:

- (A) Lower limb
- ✓ (B) Upper limb — middle finger flick
- (C) Lower limb — ankle
- (D) Jaw

Explanation: Hoffman sign: Flick/snap terminal phalanx of middle finger → flexion of thumb + index finger = positive = UMN lesion (particularly cervical myelopathy above C5).

Q40. Muscle atrophy in LMN lesion occurs due to:

- (A) Disuse only

✓ (B) Loss of trophic influence from nerve + ACh receptor hypersensitivity + fibrillations

- (C) Vascular changes only
- (D) Increased muscle metabolism

Explanation: Neurogenic atrophy in LMN: Due to (1) loss of trophic factors from nerve, (2) denervation hypersensitivity, (3) fibrillations dissipate energy, (4) structural changes. Much more severe than disuse atrophy.

Q41. House-Brackmann grading is used for:

- (A) Hand function
- ✓ (B) Facial nerve palsy
- (C) Spinal cord injury
- (D) Stroke outcome

Explanation: House-Brackmann (I-VI): Grade I = normal; Grade VI = no movement. Used to grade severity and monitor recovery of peripheral facial nerve (Bell's palsy, surgical trauma).

Q42. The pendulum test assesses:

- (A) Proprioception
- (B) Coordination
- ✓ (C) Muscle tone (spasticity)
- (D) Strength

Explanation: Pendulum test (Wartenberg): Raise leg passively and release; in spasticity, pendular oscillations are abnormal. Useful objective measure of lower limb tone.

CHAPTER 13

Case Studies with Detailed Analysis

CASE STUDY: CASE 1 — Acute Stroke with Hemiplegia

A 65-year-old right-handed male presents with sudden onset left-sided weakness, facial droop, and slurred speech for 2 hours. BP 170/100. CT head shows hyperdense MCA sign on right. Neurological exam: Left hemiplegia (MRC 1/5 arm, 2/5 leg), left facial palsy (lower face), left hemisensory loss, Broca's aphasia, left homonymous hemianopia. Brunnstrom Stage 2.

Q1: What type of stroke and territory is involved?

Ans: Right Middle Cerebral Artery (MCA) ischemic stroke. Features: Contralateral (left) hemiplegia + lower face palsy + hemisensory loss + homonymous hemianopia + aphasia (dominant hemisphere = left cortex, BUT lesion is RIGHT → non-dominant; HOWEVER right-handed patients are left dominant; therefore right MCA with Broca's aphasia means LEFT MCA is more likely — re-examine: Broca's aphasia + LEFT hemiplegia = LEFT MCA territory). This is LEFT MCA ischemic stroke.

Q2: What are the physiotherapy goals in the first 48-72 hours?

Ans: Early mobilization (within 24-48 hours if hemodynamically stable); Positioning: anti-spastic pattern (affected shoulder neutral, elbow extended, wrist neutral; hip extended, knee slightly flexed); PROM to prevent contractures; Chest physiotherapy if dysphagia/aspiration risk; Pressure area care; DVT prophylaxis (passive exercises, pneumatic compression); Communication strategy with SLT; Sensory stimulation.

Q3: Describe an appropriate 4-week rehabilitation plan.

Ans: Week 1-2 (Flaccid/Early Spastic): Positioning, PROM, sensory stimulation, bed mobility, supported sitting balance, early standing using tilt table. Week 2-3 (Brunnstrom 2-3): Weight bearing on affected limbs, facilitation within synergies, trunk control training, transfer training (bed to chair), assisted standing. Week 3-4 (Brunnstrom 3-4): Breaking synergy patterns, parallel bar walking, ankle-foot orthosis if needed, upper limb task training, ADL training, balance exercises on foam/unstable surface.

Q4: What is the prognostic indicator for arm recovery?

Ans: BEST prognostic sign: Finger extension and shoulder abduction within 72 hours. SAFE predictive rule: Shoulder Abduction + Finger Extension at 72 hours. If present → 98% chance of useful arm function at 6 months. Absent = poor prognosis for arm recovery.

CASE STUDY: CASE 2 — Guillain-Barré Syndrome

A 28-year-old male presents with ascending weakness starting in legs 2 weeks after a bout of diarrhea. He now has bilateral leg weakness (MRC 2/5), arm weakness (MRC 3/5), facial weakness, and difficulty breathing. Reflexes are absent throughout. Sensation is mildly reduced in feet. CSF: Protein 180 mg/dL, cells 3/mm³. NCV: Prolonged F-wave latencies, reduced conduction velocity. FVC 18 ml/kg.

Q1: What is the diagnosis and immediate management priority?

Ans: Diagnosis: Guillain-Barré Syndrome (AIDP variant) — ascending flaccid LMN paralysis + areflexia + albuminocytologic dissociation in CSF (high protein, normal cells). IMMEDIATE PRIORITY: Respiratory monitoring (FVC 18 ml/kg is below 20 ml/kg threshold) → INTUBATE and ventilate. Also: Monitor for autonomic instability (BP fluctuations, arrhythmias — most common cause of death).

Q2: What physiotherapy interventions are appropriate in the acute phase?

Ans: Respiratory physiotherapy: Chest auscultation, assisted coughing, secretion management, positioning for ventilation; PROM to all joints 2x daily to prevent contractures; Positioning: Limbs in neutral, anti-deformity splints (AFO for ankles in neutral); Pressure area care (2-hourly turns); Eye care if facial palsy (taping); Sensory stimulation. CONTRAINDICATIONS: No strengthening exercises of denervated muscles (may cause overwork damage); avoid painful stretching; no faradic stimulation (use galvanic for denervated muscles).

Q3: When should active rehabilitation begin and what does it include?

Ans: Active rehabilitation begins when: (1) Plateau reached (no further deterioration); (2) FVC >15ml/kg off ventilator; (3) No active autonomic instability. Progressive rehabilitation: MRC 2 → gravity-eliminated exercises, hydrotherapy; MRC 3 → active exercises, standing frame, tilt table; MRC 4 → progressive resistance training, gait re-education, ADL training. Fatigue management is crucial — GBS patients fatigue easily; aerobic pacing.

CASE STUDY: CASE 3 — Cervical Cord Injury (C5 Complete SCI)

A 22-year-old male sustains a C5 complete (ASIA A) spinal cord injury in a diving accident. He has complete motor and sensory loss below C5. He has deltoid and biceps function (MRC 3/5) bilaterally but no triceps, wrist extension, or hand function. Autonomic features: HR 55, BP 90/60. Bladder: catheterized.

Q1: What is the expected functional outcome at C5 ASIA A?

Ans: At C5 complete SCI: Preserved = Deltoid (shoulder abduction), Biceps (elbow flexion), Brachialis, Brachioradialis. Paralyzed = Triceps, wrist/hand, trunk, lower limbs. Expected function: Can propel manual wheelchair on level surfaces with special hand rims; requires adapted ADL equipment (universal cuff for feeding/grooming); Dependent for transfers (requires assistance); Can drive with hand controls and vehicle modifications. Independent with electric wheelchair (if biceps available).

Q2: What is neurogenic shock and how does it differ from spinal shock?

Ans: Neurogenic shock: Cardiovascular phenomenon; Loss of sympathetic tone due to SCI → vasodilation + bradycardia → hypotension (BP <90 systolic, HR <60 = bradycardia = distinctive). Treat with IV fluids, vasopressors if needed, atropine for bradycardia. Duration: Days to weeks. Spinal shock: Neurological phenomenon; Temporary cessation of all spinal cord function below lesion (flaccidity, areflexia, even if cord is incomplete). Ends with return of bulbocavernosus reflex (often 24-48 hours). CANNOT assess prognosis during spinal shock.

Q3: What physiotherapy program would you design for the first 3 months?

Ans: Phase 1 (ICU/Acute: 0-2 weeks): Respiratory PT (diaphragmatic breathing training, incentive spirometry, assisted cough, postural drainage); PROM all joints; Positioning in neutral alignment; DVT prophylaxis; Skin integrity monitoring 2-hourly turns. Phase 2 (2-8 weeks): Progressive strengthening of C5 muscles (deltoid, biceps); Sitting balance training; Wheelchair prescription and training; BWSTT if appropriate; Upper limb training with orthoses; Tenodesis grasp training. Phase 3 (2-3 months): Transfer training; ADL with adaptations; Wheelchair sports; Community reintegration; Home assessment and modification; Vocational rehabilitation.

CASE STUDY: CASE 4 — LMN: Bell's Palsy with Facial Nerve

A 35-year-old female presents with right-sided facial weakness developing over 24 hours. She cannot close her right eye, has loss of right forehead wrinkles, right facial droop, cannot whistle, and has altered taste on right anterior 2/3 tongue. No ear pain. BP normal. No limb weakness.

Q1: What distinguishes this from a central (UMN) facial palsy?

Ans: This is PERIPHERAL (LMN) Bell's palsy because: (1) FOREHEAD IS INVOLVED (cannot wrinkle forehead) — in UMN/central palsy, forehead is SPARED due to bilateral cortical representation; (2) ALL facial branches affected (temporal, zygomatic, buccal, marginal mandibular, cervical); (3) Taste affected (chorda tympani branch — only present in peripheral lesion near geniculate ganglion or more proximal); (4) No limb weakness/UMN signs. Central (stroke) → forehead spared + contralateral arm/leg weakness.

Q2: What physiotherapy interventions are evidence-based?

Ans: Acute phase: (1) Eye care — artificial tears, tape eyelid closed at night, moisture chamber goggles; prevent corneal exposure keratitis; (2) Facial massage — gentle circular massage to maintain muscle tone; (3) Thermal stimulation — warm/cold alternating; (4) Electrical stimulation: NMES once reinnervation begins (not in acute phase); (5) Facial exercises: mirror biofeedback; specific muscle activation exercises (NOT grimacing — avoid synkinesis); (6) Taping: facial strapping to prevent overstretching. Prognostic: 70-80% complete recovery within 6 months; Poor prognosis if complete palsy + no recovery at 3 weeks.

CHAPTER 14

Quick Revision — Mnemonics & Summary Tables

14.1 Key Mnemonics

UMN vs LMN: HYPER vs HYPO	UMN = HYPERTonia, HYPERreflexia, +Babinski, -Fasciculations LMN = HYPOtonia, HYPOreflexia, -Babinski, +Fasciculations
Seddon's Classification: "NAT"	N = Neuropraxia (Neurapraxia — No axon damage, Nucleus intact) A = Axonotmesis (Axon cut, A-tube intact) T = Teuotmesis/Neurotmesis (Total disruption)
GBS CSF: "HIGH Protein LOW cells"	Albuminocytologic Dissociation = High protein (>45mg/dL), Low cells (<10/mm ³). Remember: Protein is HIGH because nerve roots (near CSF) are inflamed; cells low because no infection.
PNF D2 Flex: "FARE"	F = Flexion (shoulder), A = Abduction (shoulder), R = External Rotation, E = Extension (elbow). Think "wave to the sky."
Brunnstrom Stages: "FSVVM"	F=Flaccid, S=Synergy develops, V=Voluntary in synergy (spasticity peaks), V=Variable (some out of synergy), V=Voluntary out of synergy, M=Motor normal (7 stages total — add N=Normal at end)
Cranial nerve motor nuclei (UMN exceptions)	Bilateral cortical input: Upper face (VII), pharynx (IX,X), larynx (X), jaw (V) → SPARED in unilateral UMN lesion. Bilateral cortical control = bilateral motor.
ASIA Grade: "ABCDE"	A=Absent (complete), B=Busted sensation only, C=Cannot lift against gravity (motor grade <3), D=Does oppose gravity (motor grade ≥3), E=Excellent (normal)
Foot drop nerves: "CPN = Dorsiflexion + Eversion"	Common Peroneal Nerve damage → Cannot Plantarflex? No! = Cannot Dorsiflex + Cannot Evert = Foot Drop. Tibial nerve → cannot plantarflex + invert.

14.2 High-Yield Facts for Exam

High-Yield Fact	Detail
Babinski sign is normal in infants	Until 12-18 months when corticospinal tract completes myelination
Fasciculations are pathognomonic of	LMN disease (specifically anterior horn cell disease)
Albuminocytologic dissociation is diagnostic of	Guillain-Barré Syndrome; also seen in spinal cord tumors, DM
Recovery rate of peripheral nerve	1 mm/day or 1 inch/month (applies to axonotmesis/neurotmesis)
GBS treatment NOT used	Steroids/Corticosteroids — worsen outcome; Use IVIG or plasmapheresis
FVC threshold for ventilation in GBS	<20 ml/kg body weight or declining rapidly
Best prognosis SCI syndrome	Brown-Sequard (hemisection) > Central > Anterior > Posterior (rare)
Most common SCI syndrome	Central Cord Syndrome (especially elderly with cervical spondylosis + hyperextension)
CIMT target population	MRC ≥3 wrist/finger extension; minimal arm movement needed; chronic stroke

Clonus significance	More than 3 beats sustained = UMN lesion; ankle clonus most common
CPG location	Lumbar spinal cord (T12-L2) for locomotion; basis of BWSTT
MAS vs Tardieu	Tardieu is more reliable for spasticity (tests velocity-dependent component specifically)
Charcot triad of MS	Nystagmus + Intention tremor + Scanning (staccato) speech/dysarthria
Uhthoff phenomenon	Worsening of MS symptoms with heat (exercise, hot bath, fever) — NOT Bell's palsy
House-Brackmann Grade VI	Complete facial nerve palsy — no movement
ASIA Grade conversion	Most improve 1 grade; 30% ASIA A improve to B; 50% ASIA B improve to D/E

This guide covers the core UMN/LMN content for GPAT, NPAT, DPT Entrance, and State PG Physiotherapy Exams. Focus on comparative features, clinical signs, and physiotherapy techniques. Revise the MCQ bank and case studies repeatedly. Best of luck!