"Spinal Cord (Part 4/4)"

General Anatomical Features of Clinical Importance:

》 Structure and Composition

- Spinal cord: Columns of motor and sensory nerve cells (gray matter)
- Surrounded by ascending and descending tracts (white matter)
- Located within the vertebral canal

》 Protection and Support:

- Protected by three fibrous membranes: meninges
- Cushioned by cerebrospinal fluid (CSF)
- Held in position by denticulate ligaments (sides) and filum terminale (inferiorly)

》 Nerve Root Pathway

- Lumbar and sacral nerve roots take an oblique downward course
- Forms the cauda equina due to the short spinal cord relative to the vertebral column length

》 Clinical Procedure

- Spinal tap needle insertion:
- Safe below the level of the second lumbar vertebra
- Avoids damage to the spinal cord

"Anterior and Posterior Nerve Root Lesions"

》 Covering and Formation

- Nerve roots covered by pia, arachnoid, and dura mater
- Anterior and posterior roots unite in intervertebral foramina to form spinal nerves
- Meninges fuse with the epineurium of spinal nerves

》 Involvement in Conditions

1) Syphilitic spinal meningitis or pyogenic meningitis
- May involve either or both spinal nerve roots

2) Tabes dorsalis and herpes zoster
- May involve posterior roots

» Compression and Irritation

> Compression Sources:

- Tumors of the vertebral column
- Herniated intervertebral disc
- Primary or secondary vertebral tumors
- Vertebral destruction by tumor or infection
- Fracture dislocation
- Severe scoliosis

> Irritation Sources:

- Abnormal constituents of CSF (e.g., blood from subarachnoid hemorrhage)

» Symptoms of Posterior Nerve Root Lesions

- Pain in the area of skin and muscles innervated by the affected root
- Pain worsens with vertebral column movement, coughing, and sneezing
- Possible hyperalgesia and hyperesthesia before loss of sensation

》 Symptoms of Anterior Nerve Root Lesions

- Paralysis of muscles supplied exclusively by the affected root
- Partial paralysis of muscles supplied partially by the affected root
- Fasciculation and muscle atrophy in affected muscles

"Clinical Significance of Ascending Tract Lamination"

》 Anterolateral White Column (Spinothalamic Tracts)

> Axonal Pathways:

- Axons from sacral and lumbar segments deflected laterally
- Deflection occurs due to axons crossing the midline (decussation) at successively higher levels
- This deflection causes lamination

> Lamination:

- Cervical to sacral segments are arranged from medial to lateral

》 Posterior White Column (Medial Lemniscus System)

> Axonal Pathways:

- Axons from sacral and lumbar segments are pushed medially
- This occurs due to axons from higher segments

> Lamination:

- Sacral to cervical segments are arranged from medial to lateral

[Lamination: Lamination in the context of the spinal cord refers to the organization or arrangement of nerve fibers in specific tracts]

- Side Note -

- DCML pathway decussates in medulla
- Spinothalamic pathway decussates soon after entering spinal cord

• Descending Pathways: Corticospinal Tract:

-> Descending Fibers:

》 Decussation

Most corticospinal fibers cross at the medullary pyramids (pyramidal decussation).

》 Post-Decussation

1) Lateral Corticospinal Tract:
- After decussation, these fibers descend in the lateral columns of the spinal cord.

2) Anterior Corticospinal Tract: Some fibers do not decussate at the medulla but descend ipsilaterally and decussate at the spinal cord level where they terminate !!!

"Ascending Tract Injury"

》 External Pressure on Spinal Cord

- Region: Spinothalamic tracts
- Initial Effects: Loss of pain and temperature sensations in sacral dermatomes
- Increased Pressure: Affects higher segmental dermatomes (a dermatome is an area of skin that is mainly supplied by a single spinal nerve)

1) Lateral Spinothalamic Tract

》 Destruction Effects

- Contralateral loss of pain and thermal sensibilities below lesion
- No response to pinprick
- Inability to recognize hot and cold objects on skin

》 Anterior Spinothalamic Tract

> Destruction Effects

- Contralateral loss of light touch and pressure sensibilities below lesion
- Discriminative touch remains (conducted through fasciculus gracilis and fasciculus cuneatus)
- No sensation of light touch (e.g., cotton) or pressure (e.g., blunt object) on skin

》 Fasciculus Gracilis and Fasciculus Cuneatus

> Destruction Effects

- Cuts off muscle and joint information to consciousness
- Unawareness of ipsilateral limb position and movements below lesion (muscle joint sense)
- Example: Cannot tell position of dorsiflexed big toe with eyes closed
- Impaired muscular control: Jerky or ataxic movements
- Loss of vibration sense below lesion on same side

- Tested with vibrating tuning fork on bony prominence (e.g., lateral radius, malleolus of fibula, stylus)
- Loss of tactile discrimination on side of lesion
- Tested by separating two points of a compass until recognized as separate
- Normal: 3–4 mm separation on fingertips, 65 mm or more on back

> General Light Touch

- Unaffected: Impulses ascend in anterior spinothalamic tracts

> Spinal Cord Lesions

- Localized lesions affecting only one sensory tract are rare
- Typically involve multiple ascending and descending tracts

"Somatic and Visceral Pain"

》 Somatic Pain

> Sense Organs: Naked nerve endings.

> Transmission:

- Initial sharp pain: Transmitted by fast-conducting fibers.
- Prolonged burning pain: Travels in slow-conducting nerve fibers (see p. 143).
- Travel via somatic nervous system

》 Visceral Pain

> Receptors:

- Chemoreceptors
- Baroreceptors
- Osmoreceptors (BOCS)
- Stretch receptors

> Stimuli Sensitivity: Ischemia, stretching, chemical damage.

> Afferent Fibers:

- Travel via sympathetic and parasympathetic parts of the autonomic nervous system to the CNS via visceral afferent fibers

> Pain Impulse Pathway:

- Once in CNS, travel by same ascending tracts as somatic pain.
- Ultimately reach the postcentral gyrus.

> Characteristics of Visceral Pain

- Poor localization.
- Associated symptoms: Salivation, nausea, vomiting, tachycardia, sweating.

> Referred Pain:

- Pain may be referred from the organ involved to a distant area of the body.

"Tabes Dorsalis"

》 Cause: A bacteria called "syphilis"

》 Mechanism:

- Organism causes selective destruction of nerve fibers at the point of entrance of the posterior root into the spinal cord, especially in the lower thoracic and lumbosacral regions.
[Posterior nerve root damaged -> hyperalgesia + hyperesthesia followed by pain in the effected dermatomes]

》 Symptoms and Signs

1) Stabbing Pains (hyperalgesia):
- Location: Lower limbs.
- Severity: May be very severe.

2) Paresthesia:
- Numbness in the lower limbs.

3) Hypersensitivity:
- Skin sensitivity to touch, heat, and cold (hyperesthesia)

4) Loss of Skin Sensation:

- Areas: Skin of parts of the trunk and lower limbs.
- Effects: Loss of awareness that the urinary bladder is full.

5) Loss of Proprioception:

- Posture appreciation.
- Passive movements of the limbs, especially the legs.

6) Loss of Deep Pain Sensation:

- Examples: When muscles are forcibly compressed or the Achilles tendon is compressed between the finger and thumb.

7) Loss of Superficial Pain Sensation:

- Areas: Skin in certain areas of the body such as the side of the nose, medial border of the forearm, thoracic wall between the nipples, or lateral border of the leg

8) Ataxia:

- Affects: Lower limbs.
- Cause: Loss of proprioceptive sensibility.
- Note: Unsteadiness in gait is compensated by vision; worsens in the dark or when eyes are closed, leading to falls.

9) Hypotonia:

- Cause: Loss of proprioceptive information from muscles and joints.

10) Loss of Tendon Reflexes:

- Cause: Degeneration of the afferent fiber component of the reflex arc.
- Early signs: Loss of knee and ankle tendon jerks.

"Muscle Activity"

》 Muscle Tone

> Definition: Continuous partial contraction of a muscle.

> Dependency: Integrity of a monosynaptic reflex arc.

> Receptor Organs: Muscle spindles

> Pathway:

- Afferent neuron enters spinal cord through the posterior root.
- Synapses with the effector neuron (lower motor neuron) in the anterior gray column.
- Lower motor neuron supplies muscle fibers through anterior roots, spinal nerves, and peripheral nerves.

》 Characteristics of Muscle Tone

> Abolishment: Muscle tone is lost if any part of the reflex arc is destroyed.
- Atonic muscle: Feels soft and flabby, atrophies rapidly.

> Normal Muscle Tone:

- Exhibits resilience or elasticity.
- Resistance felt when muscle is passively stretched.

• Dependent on:

- Integrity of the monosynaptic reflex arc.
- Control by impulses from descending tracts from supraspinal levels.

> Influence:

- Muscle spindles: Excitatory to muscle tone.
- Neurotendinous receptors: Inhibitory to muscle tone.

》 Voluntary Movement

> Initiation: By the individual, involving multiple muscle contractions to achieve a goal.

> Influence: Descending tracts affecting lower motor neurons driven by sensory information (eyes, ears, muscles) and past afferent information stored in memory.

> Emotional Input: Limbic structures influence initiation of voluntary movement via projections to the cerebral cortex.

» Pathways and Tracts

> Descending Pathways:

- From cerebral cortex and brainstem (upper motor neurons) influence lower motor neurons directly or through internuncial neurons.
- Brainstem tracts receive input from the cerebral cortex.

> Corticospinal Tracts:

- Control prime mover muscles, especially for highly skilled movements of distal limbs.

> Other Supraspinal Descending Tracts:

- Major role in basic voluntary movements.
- Adjust muscle tone for easy and rapid joint movements.

» Influence of Basal Ganglia and Cerebellum

- Do not give rise to descending tracts affecting lower motor neurons.
- Influence voluntary movements indirectly via projections to cerebral cortex and brainstem nuclei.

» Pyramidal and Extrapyramidal Tracts

> Pyramidal Tract:

- Refers to corticospinal tracts.
- Named for concentration in the anterior part of the medulla oblongata (pyramids)

> Extrapyramidal Tracts:

- Refers to all descending tracts other than the corticospinal tracts.

"Upper Motor Neuron Lesions"

» Corticospinal Tract (Pyramidal Tract) Lesions

1) Babinski Sign

- Great toe dorsally flexes, other toes fan outward when the skin along the lateral aspect of the sole is scratched.
- Normal response: Plantar flexion of all toes.
- Normally present during the first year of life (corticospinal tract not myelinated until end of the first year).

2) Explanation for Babinski Sign

- Normally: Corticospinal tracts produce plantar flexion of toes in response to sensory stimulation of the sole.
- When nonfunctional: Influence of other descending tracts leads to withdrawal reflex, with great toe dorsally flexed and other toes fanning out.

3) Superficial Abdominal Reflexes Absent

- Abdominal muscles fail to contract when skin of the abdomen is scratched.
- Dependent on integrity of corticospinal tracts (tonic excitatory influence on internuncial neurons).

4) Cremasteric Reflex Absent

- Cremaster muscle fails to contract when skin on the medial side of the thigh is stroked.
- Reflex arc passes through the first lumbar segment of the spinal cord.
- Dependent on integrity of corticospinal tracts (tonic excitatory influence on internuncial neurons).

5) Loss of Fine-Skilled Voluntary Movements

- Especially at the distal end of the limbs.

» Lesions of Other Descending Tracts (Extrapyramidal Tracts) [PH-EC]

1) Severe Paralysis (spastic)

- Little or no muscle atrophy (except secondary to disuse).

2) Spasticity or Hypertonicity of Muscles

- Lower limb maintained in extension.
- Upper limb maintained in flexion.

3) Exaggerated Deep Muscle Reflexes and Clonus

- May be present in flexors of fingers, quadriceps femoris, and calf muscles.

4) Clasp-Knife Reaction

- Muscle spasticity produces resistance during passive movement of a joint.
- Muscles suddenly give way due to neurotendinous organ-mediated inhibition.

» Clinical Practice

- Organic lesions restricted only to pyramidal or extrapyramidal tracts are rare.
- Usually, both sets of tracts are affected to varying extents, producing both groups of clinical signs.
- Pyramidal tracts increase muscle tone.
- Extrapyramidal tracts inhibit muscle tone.
- Balance between these effects alters muscle tone to varying degrees.

"Lower Motor Neuron Lesions"

》 Causes

- Trauma
- Infection (e.g., poliomyelitis)
- Vascular disorders
- Degenerative diseases
- Neoplasms

》 Clinical Signs

1) Muscles exhibit flaccid paralysis

2) Muscles atrophy

3) Muscles lose reflexes

4) Muscular fasciculation (muscle twitching): Seen only with slow destruction of the lower motor neuron cell

5) Muscular contracture: Occurs more often in antagonist muscles whose action is no longer opposed by the paralyzed muscles.
(Definition: A condition when your muscles, tendons, joints, or other tissues tighten or shorten causing a deformity)

6) Muscle response to stimulation:

- Normally innervated muscles respond to faradic (interrupted) current (contraction continues as long as current passes).
- Galvanic (direct) current causes contraction only when turned on or off.
- After lower motor neuron cut, muscle stops responding to interrupted current after 7 days and direct current after 10 days.
- This change is known as the reaction of degeneration.

"Types of Paralysis"

-> Hemiplegia: Paralysis of one side of the body (upper limb, one side of trunk, lower limb)

-> Monoplegia: Paralysis of one limb only.

-> Diplegia: Paralysis of two corresponding limbs (e.g., arms or legs).

-> Paraplegia: Paralysis of the two lower limbs.

-> Quadriplegia: Paralysis of all four limbs.

"Relationship of Muscular Signs and Symptoms to Nervous System Lesions"

》 Hypotonia

> Definition: Diminished or absent muscle tone.

> Causes:

- Interruption of any part of the monosynaptic stretch reflex arc.
- Cerebellar disease (diminished influence on γ-motor neurons from cerebellum).
- LMNLs

》 Hypertonia

> Definition: Increased muscle tone (spasticity, rigidity)

> Causes:

- Lesions involving supraspinal centers or their descending tracts (excluding corticospinal tract).
- Local spinal segmental level (e.g., back muscle spasm from prolapsed intervertebral disc, abdominal muscle spasm from peritonitis).
- UMNLs (EP tracts)

》 Tremors

> Definition: Rhythmic involuntary movements from contraction of opposing muscle groups.

> Types:

- Slow (e.g., parkinsonism)
- Fast (e.g., toxic tremors from thyrotoxicosis).
- Resting tremor (e.g., parkinsonism).
- Intention tremor (e.g., cerebellar disease).

» Spasms

> Definition: Sudden, involuntary contractions of large muscle groups.

> Examples:

- Seen in paraplegia (due to lesions involving descending tracts - UMNLs - but not corticospinal tract).

» Athetosis

> Definition: Continuous, slow, involuntary, dysrhythmic movements.

> Characteristics:

- Movements are the same in the same patient.
- Disappear during sleep.
- Impede voluntary movement.

> Cause: Lesions of the corpus striatum.

》 Chorea

> Definition: Series of continuous, rapid, involuntary, jerky, coarse, purposeless movements.

> Characteristics:

- May occur during sleep.

> Cause: Lesions of the corpus striatum.

》 Dystonia

> Definition: Frequent, maintained contractions of hypertonic muscles, leading to bizarre postures.

> Cause: Lesions of the lentiform nucleus.

》 Myoclonus

> Definition: Sudden contraction of an isolated muscle or part of a muscle.

> Characteristics:

- Irregular occurrence.
- Commonly involves a limb muscle.

> Causes:

- Diseases involving the reticular formation and cerebellum.
- Normal myoclonic jerks during sleep onset (sudden temporary reactivation of the reticular formation).

》 Hemiballismus

> Definition: Rare form of involuntary movement confined to one side of the body.

> Characteristics:

- Usually involves proximal extremity musculature.
- Limb moves erratically in all directions.

> Cause: Lesion in the opposite subthalamic nucleus.

Acute Spinal Cord Injury

》 Incidence

- Approximately 10,000 cases per year in the United States.

» Consequences

- Catastrophic injury with little or no nerve tract regeneration.
- Permanent disability.

» Treatment

- Anatomical realignment and stabilization of the vertebral column.

- Decompression of the spinal cord.

- Intensive rehabilitation to optimize remaining neurologic function.

- Improved management of medical complications.

- Use of certain drugs (GM, ganglioside, and methylprednisolone) soon after injury shows some improvement in neurologic deficit.

- Animal experiments suggest these drugs enhance functional recovery of damaged neurons.

"Chronic Compression of the Spinal Cord"

》 Causes

i) Extradural:

- Herniation of an intervertebral disc.
- Infection of vertebrae (e.g., tuberculosis).
- Primary and secondary tumors of vertebrae.
- Leukemic deposits.
- Extradural abscesses.

ii) Intradural:

> Extramedullary:

- Meningiomas.
- Nerve fibromas.

> Intramedullary:

- Primary tumors of the spinal cord (e.g., gliomas).

» Pathophysiology

> Pressure on spinal arteries:
- Ischemia of the spinal cord, degeneration of nerve cells and fibers.

> Pressure on spinal veins:
- Edema of the spinal cord, interference in neuron function.

> Direct pressure on white and gray matter:
- Interference with nerve conduction.

> Obstructed CSF circulation:
- Changes in fluid composition below the obstruction level

» Clinical Signs

> Pain

- Local pain in the involved vertebra.
- Radiating pain along spinal nerve roots.
- Worsens with coughing, sneezing, and at night when recumbent.

> Motor Function Interference

- Early involvement of anterior gray column motor cells: Partial or complete muscle paralysis, loss of tone, muscle wasting.
- Early involvement of corticospinal and other descending tracts: Muscular weakness, increased muscle tone (spasticity), increased tendon reflexes below the lesion, extensor plantar response.

> Sensory Loss

- Posterior white columns lesion: Loss of proprioception, vibration sense, tactile discrimination below the lesion on the same side.

- Lateral spinal thalamic tracts lesion: Loss of pain, heat, and cold sensations on the opposite side of the body below the lesion.

- Diagnosis and Investigation

- Early accurate diagnosis is essential for successful removal of benign spinal tumors.

> Investigations:

- Radiography of the vertebral column.
- Computed tomography (CT).
- Magnetic resonance imaging (MRI).
- Spinal tap.
- Myelography when the diagnosis is difficult.

"Clinical Syndromes Affecting the Spinal Cord"

》 Myelopathy Causes:

- Trauma
- Developmental abnormality
- Infection
- Autoimmune destruction
- Genetic disease

》 Spinal Shock Syndrome

> Definition:

- Clinical condition following acute severe damage to the spinal cord
- All cord functions below the lesion are depressed or lost
- Sensory impairment and flaccid paralysis occur

> Mechanism:

- Depression of segmental spinal reflexes due to removal of influences from higher centers
  - Mediated through:
    - Corticospinal tracts
    - Reticulospinal tracts
    - Tectospinal tracts
    - Rubrospinal tracts
    - Vestibulospinal tracts

> Symptoms:

- Severe hypotension from loss of sympathetic vasomotor tone (especially with high-level lesions)

1) Acute phase: All of LMNLs' symptoms + loss of sensation + bladder control (autonomic fiber lesion)
2) Recovery phase: Symptoms of UMNLs-EP: PHEC

> Duration:

- Shock persists for less than 24 hours in most patients
- Can persist for up to 4 weeks in some cases

> Recovery:

- Neurons regain excitability as shock diminishes
- Effects of upper motor neuron loss appear in segments below the lesion:
  - Spasticity
  - Exaggerated reflexes

> Diagnosis:

- Test for anal sphincter reflex activity:
  - Place gloved finger in anal canal
  - Stimulate anal sphincter contraction by:
    - Squeezing glans penis or clitoris
  - Gently tugging on an inserted Foley catheter
  - Absence of anal reflex indicates spinal shock

> Limitations of Test:

- Ineffective if sacral segments of the cord (S2-S4) are involved
- Neurons giving rise to inferior hemorrhoidal nerve to anal sphincter would be nonfunctioning

"Destructive Spinal Cord Syndromes"

》 Types

- Complete cord transection syndrome
- Anterior cord syndrome
- Central cord syndrome
- Brown-Séquard syndrome (cord hemisection)

[- Their symptoms occur after Spinal Shock has ended]

》 General Findings

- Combination of lower motor neuron injury (at lesion level) and upper motor neuron injury (below lesion level)

Hack: Write following

- UMNLs symptoms
- LMNLs symptoms
- Spinothalamic tracts' sensation loss
- DCML tracts' sensation loss (if involved)

"Complete Cord Transection Syndrome

》 Causes

- Fracture dislocation of vertebral column
- Bullet or stab wound
- Expanding tumor

》 Clinical Features

1) Bilateral lower motor neuron paralysis and muscular atrophy at lesion level

- Damage to anterior gray columns neurons and possibly nerve roots

2) Pyramidal + Extrapyramidal Effects

》 EP tract lesion:

- Bilateral spastic paralysis below lesion level
- PHEC

» P tract lesion:

- Bilateral Babinski sign
- Bilateral loss of superficial abdominal and cremaster reflexes
- Interruption of corticospinal tracts on both sides

3) Bilateral loss of all sensations below lesion level

- Loss of tactile discrimination, vibratory, and proprioceptive sensations (posterior white columns destruction)
- Loss of pain, temperature, light touch (lateral and anterior spinothalamic tracts section)
- Sensory loss two or three segments below lesion

4) Bladder and bowel functions

- No voluntary control (destruction of descending autonomic fibers)

[Special Case: L2-L3 Vertebral Level Fracture Dislocation
- No cord injury
- Neural damage confined to cauda equina (lower motor neuron, autonomic, and sensory fibers involved)]

"Anterior Cord Syndrome"

》 Causes

- Cord contusion during vertebral fracture/dislocation
- Injury to anterior spinal artery or feeder arteries
- Herniated intervertebral disc

》 Clinical Features

1) Bilateral lower motor neuron paralysis and muscular atrophy at lesion level
- Damage to anterior gray columns neurons and possibly anterior nerve roots

2) Bilateral spastic paralysis below lesion level
- Interruption of EP tracts

3) Bilateral loss of pain, temperature, and light touch sensations below lesion level
- Interruption of anterior and lateral spinothalamic tracts

4) Preserved tactile discrimination, vibratory, and proprioceptive sensations
- Posterior white columns undamaged

- Side Note -
Paralysis
- LMNL -> At the level of lesion -> Flaccid paralysis
- UMNL -> 2-3 segments below lesion -> Spastic paralysis

"Central Cord Syndrome"

》 Causes

- Hyperextension of cervical region

》 Mechanism

- Cord pressed anteriorly by vertebral bodies and posteriorly by bulging ligamentum flavum
- Damage to central region of spinal cord

》 Clinical Features

1) Bilateral lower motor neuron paralysis and muscular atrophy at lesion level
- Damage to anterior gray columns neurons and possibly nerve roots

2) Bilateral spastic paralysis below lesion level with sacral "sparing"
- Lower limb fibers less affected than upper limb fibers
- Upper limb fibers -> located medially in laminated EP tracts -> more susceptible to damage than lower limb fibers

3) Bilateral loss of pain, temperature, light touch, and pressure sensations below lesion level with sacral "sparing"
- Upper limb fibers -> located medially in laminated ascending tracts -> more susceptible to damage than lower limb fibers

4) Sparing of lower body evidenced by:
- Presence of perianal sensation
- Good anal sphincter tone
- Ability to move toes slightly

》 Prognosis

- Often good if caused by spinal cord edema alone
- Mild syndrome may include upper arm paresthesias and mild arm and hand weakness

》 Paralysis

- LMNL -> At the level of lesion -> Flaccid paralysis
- UMNL -> 2-3 segments below lesion -> Spastic paralysis

"Brown-Séquard Syndrome (Cord Hemisection)"

》 Causes

- Fracture dislocation of vertebral column
- Bullet or stab wound
- Expanding tumor

》 Clinical Features

1) Ipsilateral lower motor neuron paralysis and muscular atrophy at lesion level
   - Damage to anterior gray column neurons and possibly nerve roots

2) Ipsilateral spastic paralysis below lesion level

- Ipsilateral Babinski sign
- Ipsilateral loss of superficial abdominal and cremasteric reflexes
- Loss of corticospinal tracts (P + EP) on the lesion side

P + EP tracts decussate in pyramids -> hence in spinal cord they remain ipsilateral

3) Ipsilateral band of cutaneous anesthesia at lesion level

- Destruction of posterior root and its entrance into spinal cord

4) Ipsilateral loss of tactile discrimination and of vibratory and proprioceptive sensations below lesion level

- Destruction of ascending tracts in posterior white column

5) Contralateral loss of pain and temperature sensations below lesion level

- Destruction of crossed lateral spinothalamic tracts
- Sensory loss two or three segments below lesion

6) Contralateral but incomplete loss of tactile sensation below lesion level
- Destruction of crossed anterior spinothalamic tracts
- Sensory impairment two or three segments below lesion
- Incomplete loss due to intact discriminative touch in contralateral posterior white column

"Syringomyelia"

》 Cause

- Developmental abnormality in central canal formation

》 Affected Areas

- Brainstem and cervical region
- Cavitation and gliosis in central neuroaxis

》 Clinical Features

1) Loss of pain and temperature sensations in dermatomes related to affected segments
- Shawllike distribution
- Interruption of lateral spinothalamic tracts
- Accidental burning injuries to fingers common

2) Preserved tactile discrimination, vibratory, and proprioceptive sense
- Ascending tracts in posterior white column unaffected

3) Lower motor neuron weakness in small muscles of hand
- Bilateral or one hand affected first
- Lesion expansion in lower cervical and upper thoracic region destroys anterior horn cells

4) Bilateral spastic paralysis of both legs
- Exaggerated deep tendon reflexes
- Positive Babinski response
- Caused by expansion of lesion laterally into white column, involving descending tracts

5) Possible Horner syndrome
- Interruption of descending autonomic fibers in reticulospinal tracts in lateral white column by expanding lesion

"Poliomyelitis"

》 Description:

- Acute viral infection of neurons in anterior gray columns of spinal cord and cranial nerve motor nuclei.
- Immunization has greatly reduced incidence.

》 Effects:

- Paralysis and muscle wasting follow motor nerve cell death.
- Lower limb muscles are more often affected than upper limbs.
- Severe cases: paralysis may spread to intercostal muscles and diaphragm.
- Muscles of face, pharynx, larynx, and tongue may also be paralyzed.

》 Recovery:

- Improvement begins at the end of the first week as edema subsides.
- Function returns to neurons not destroyed.

"Multiple Sclerosis"

» Description:

- Common disease causing demyelination in CNS (ascending and descending tracts).
- Affects young adults; cause is unknown.
- Possible factors: autoimmunity, infection, heredity.

» Pathophysiology:

- Breach in blood-brain barrier allows leukocytes into CNS.
- Inflammation and demyelination result in breakdown of axon insulation.
- Reduced and blocked action potential velocity.
- Myelin is rich in lipids and contains important proteins.
- Basic myelin proteins can cause strong immune response and demyelination.
- Mutations in myelin protein structure may cause inherited demyelination.

》 Symptoms:

- Chronic course with exacerbations and remissions.
- Widespread involvement of different tracts.
- Weakness of limbs is common.
- Ataxia due to cerebellum tract involvement.
- Spastic paralysis may be present.

》 Remissions:

- Remodeled demyelinated axonal plasma membrane with more sodium channels may explain remissions.
- Progressive form involves substantial axonal and myelin damage.

"Amyotrophic Lateral Sclerosis (ALS)"

》 Description:

- Disease affecting corticospinal tracts and motor neurons of anterior gray columns.
- Rarely familial; inherited in about 10% of patients.
- Chronic progressive disease of unknown etiology.

》 Demographics:

- Typically occurs in late middle age.
- Fatal within 2 to 6 years.

》 Symptoms:

- Lower motor neuron signs: progressive muscular atrophy, paresis, fasciculations.
- Upper motor neuron disease signs: paresis, spasticity, Babinski response.
- Involvement of motor nuclei of some cranial nerves.

"Parkinson Disease"

》 Description:

- Associated with neuronal degeneration in substantia nigra, globus pallidus, putamen, and caudate nucleus.
- Degeneration of inhibitory nigrostriate fibers reduces dopamine release in corpus striatum.

》 Effects:

- Hypersensitivity of dopamine receptors in postsynaptic neurons in corpus striatum, leading to overactivity.
- Tremor, cogwheel rigidity (hyperkinetic activity).
- Difficulty initiating voluntary movements (hypokinetic activity).

"Pernicious Anemia"

》 Description:

- Form of megaloblastic anemia caused by vitamin B12 deficiency.

》 Effects:

- Extensive damage to tracts in posterior and lateral white columns of spinal cord.
- Peripheral nerve degeneration.
- Widespread sensory and motor losses due to involvement of ascending and descending tracts.