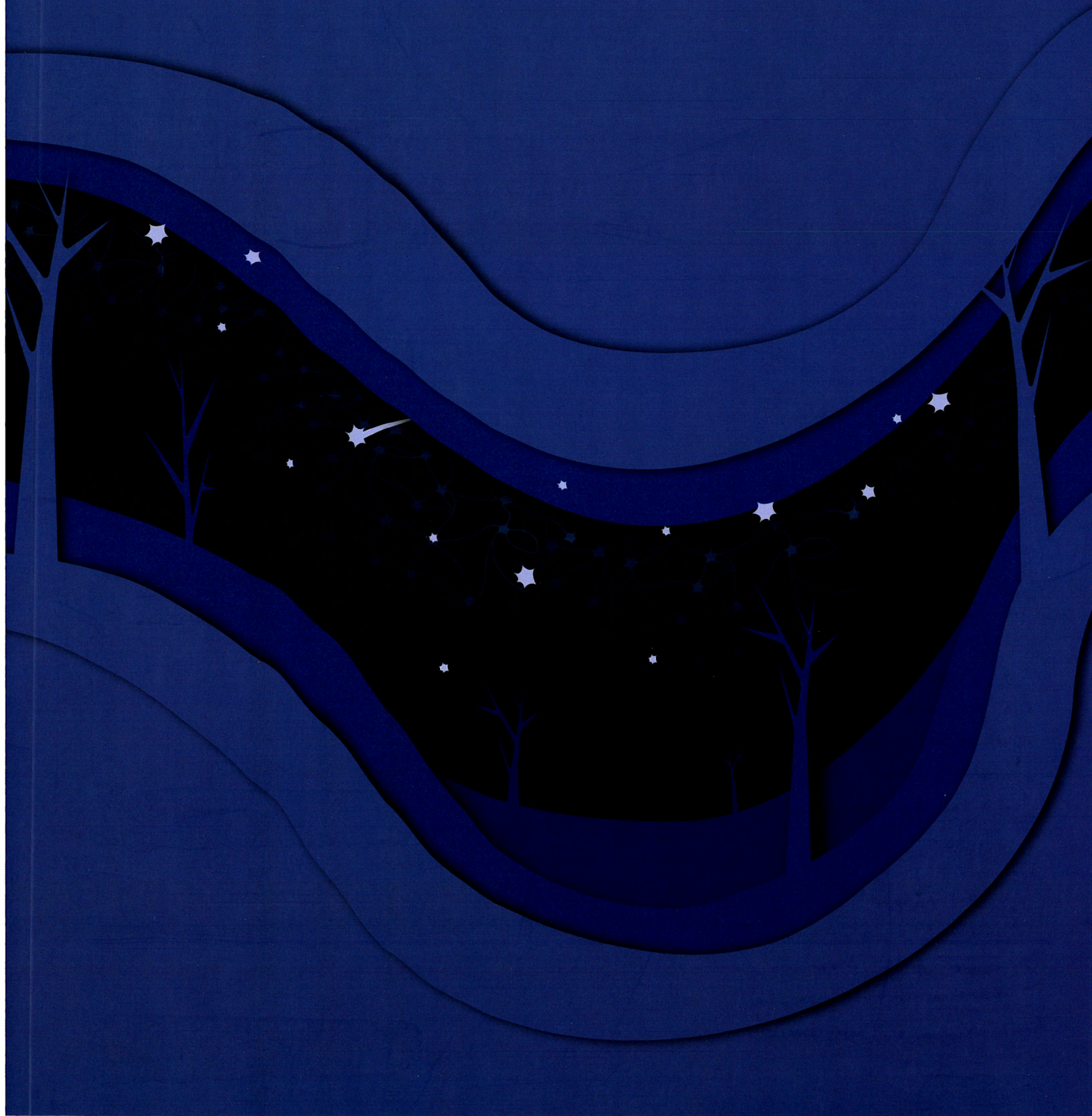


NEUROLOGY 1

Marrow SS Medicine



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APPROACH TO NEUROLOGY

----- Active space -----

Overview of the steps

00:02:17

1. Pathology
2. LMN v/s UMN
3. Specific etiology.
 - UMN : Cortical, Subcortical, Brainstem or spinal cord involvement.
 - LMN : Anterior horn cells, root, plexus, nerve, neuromuscular junction or muscle involvement.
4. Associated features like fever or constitutional symptoms, weight loss, palpitations, chest pain, skin rashes, joint pains and swelling, cough and breathlessness.

Assessing the pathology

00:04:10

The approach :

1. Onset : Acute/sub-acute/chronic onset of symptoms.
2. Duration : Days/ hours/ months/ years
3. Progression : Progressive/ improving/ recurrent

Order of examination of nervous system :

- Higher mental functions : Cognitive symptoms.
- Cranial nerves.
- Motor system + coordination and cerebellum including gait.
- Sensory.

Acute onset :

Hyper acute : e.g. when patient tells yesterday morning by 9 am when I was writing with pen at, suddenly I felt weakness in upper limb.

- Vascular causes and seizures present with hyper acute.

Acute :

- E.g. morning when I got up I had paraesthesia of right hand and then it worsened and reached shoulder by 1 hour.
- And by afternoon, right upper limb weakness.
- And by evening weakness worsened.
- By next day morning complete weakness.

---- Active space ----

Classical acute presentations :

1. Vascular : LMN weakness with pain.
2. Demyelination like in GBS.
3. Trauma.
4. Bleed into a tumour.

Sub-acute : Progressed over days to months.

1. Demyelinating.
2. Thrombotic stroke or multi infarct stroke.
3. Nutritional or metabolic.
4. Tumour : malignant.

Chronic :

1. Degenerative.
2. Genetic or hereditary : Spino cerebellar ataxia.
3. Benign tumours : Over years.

Duration :

- Symptom of 2 or 3 days - acute.
- Symptoms lasting for years - degenerating or genetic disease.

Progression :

- Worsening
- Improving : Stroke/Demyelination.
- Static : Gliotic or somatic lesions.
- Recurrent illness : Demyelination.

LMN vs UMN based on examination

00:13:20

LMN v/s UMN

Higher mental function can be remembered as : **AIME LOSS handedness**

- Appearance, Intelligence, memory, emotional status.
- Level of consciousness, orientation, speech and handedness.
- +/- Lobar functions and mmSC.

----- Active space -----

| | UMN | LMN |
|------------------------------|--------------------------------------------|------------------------------|
| 1) Function | Inhibitory effect on muscle stretch reflex | Motor part of stretch reflex |
| 2) Type of paralysis | spastic | Flacid |
| 3) Bulk | Normal / disuse | Wasting |
| 4) Fasciculation | Absent | Present |
| 5) Tone | Increased | Flacid |
| 6) DTR | Exaggerated | Areflexic |
| 7) Babinski | Positive | negative |
| 8) Abdominal and cremasteric | Absent | Present usually |
| 9) Cortical signs | present | Absent |
| 10) Pattern | Pyramidal pattern | Nerve / root pattern |

| | LMN | UMN |
|---------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Higher motor functions | - | +/- |
| Cranial nerves | + and LMN type | + and UMN type |
| Motor : 1. Bulk 2. Tone 3. Power 4. Reflexes 5. Gait 6. Co-ordination 7. Involuntary movements | Atrophy Decreased Plexus/Nerve/ Polyradiculopathy Hypo or Areflexia High stepping gait Cerebellum normal Polymini myoclonus, segmental myoclonus | Disuse atrophy Rigidity/spasticity Pyramidal Pattern Exaggerated reflexes Circumduction gait Cerebellum affected +/- myoclonus, dystonia, tremors |
| Reflexes : Superficial - Plantar & abdominal. DTR | Normal. unless the segment is affected Decreased | Babinski positive - plantar Abdominal absent Brisk/exaggerated |
| Sensory | Depends on small or large fibres | Seen in spinal cord injury - funicular, circumferential pain. Ankle jerk is checked. |

Ankle jerk :

- In large fibre involvement (peripheral neuropathy) : Lost.
- In posterior column involvement : Normal.

Pyramidal Pattern : seen in UMN**Upper limb :**

- Wrist extension : weak.
- Supination > Pronation : weak.
- Elbow extension is weak.
- Shoulder abduction is weak.

---- Active space ----

Lower Limb :

- Hip flexion, knee flexion and ankle dorsiflexion is weak.
- Adductor spasticity is present
- Abduction of hip is weak.

Motor features : UMN and LMN

00:31:48

motor :

- Distal (E.g : Picking coins, buttoning and un-buttoning clothes)
Proximal muscle weakness (Combing of hair)
- Symmetrical or asymmetrical.
- Nerve pattern/ plexus pattern/pyramidal pattern.
- Any sign of spasticity :
 - Foot clearance from ground is more affected in UMN.
 - Tripping, heaviness of foot and falls is seen in UMN.
 - Wasting, fasciculations are seen in LMN.
- Reflex.
- Involuntary movements.
- Cerebellum and ataxia.
- Stance and gait : UMN v/s LMN.

Origin of motor activity in the cortex :

Normally,

- Posterior parietal area sends the signals for motor function to the SMA which ultimately reaches motor cortex → Signal carried to the internal capsule → Signal reaches either brainstem nucleus or the spinal cord.
- From brainstem nucleus, the signal is transmitted to facial or craniofacial muscles.
- From spinal cord, it is transmitted to the limbs and trunk.

In the presence of extra pathways :

- Basal ganglia and cerebellum helps in further fine-tuning(co-ordination) of activities.
- The motor signals pass from the motor cortex through the basal ganglia to the internal capsule.
- The cortico-pondo cerebellar fibres carry the signal to the cerebellum which in turn gives back the signal to cerebrum and the descending circuit.

Affection of posterior parietal area, SMA and motor cortex → Apraxias

Affection of basal ganglia → Extrapyramidal symptoms.(Bradykinesia)

Affection of Cerebellum → Inco-ordination and cerebellar symptoms(Ataxia)

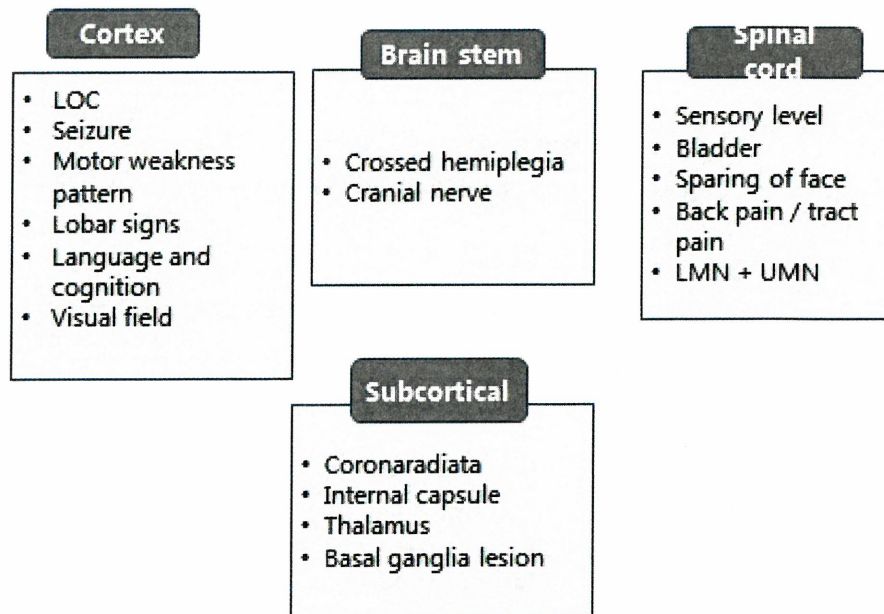
Affection of motor cortex → Spasticity and classical UMN features.

UMN approach : Summary

00:40:43

----- Active space ---

UMN



In motor cortex :

- maximum representation is for face, lips and hands.
- Hence, pattern of weakness will be facio-brachial predominantly, dexterity loss and distal weakness (more than one nerve is affected).

Brain stem :

Crossed hemiplegia :

- In brainstem the UMN fibres are descending into the nucleus of the cranial nerve → Supplies the ipsilateral muscle or craniofacial structures.
- The pyramidal fibres descend downwards and cross to opposite side (C/L) at the medulla.
- Hence, if there is a lesion in the brainstem, **I/L cranial nerve and C/L pyramidal fibres are affected** → **Crossed hemiplegia**.
- vertigo and vestibular symptoms s/o brainstem involvement.

Spinal cord : If affected :

- Non affection of craniofacial musculature,
- worsening of pain in the involvement of **5th nerve**.
- **Involvement of limbs and lower limbs is seen without cranial nerve involvement.**
- Sensory level : **Involvement of bladder, sparing of face, backpain/tract pain.**
- LMN + UMN : Either a cervical myelopathy or anterior horn involvement.

--- Active space ---

Subcortical :

Corona radiata :

- If there is a lesion (1 cm) in corona radiata, it will affect only few fibres weakness is less dense.
- 1 cm lesion in internal capsule → Dense weakness as many fibres will be affected.

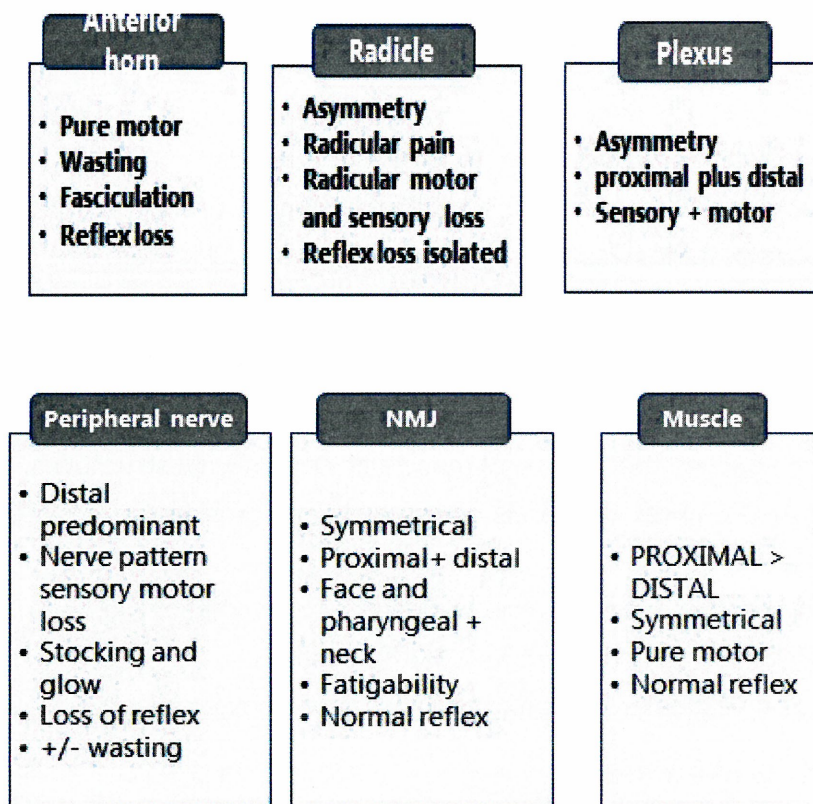
Thalamus : Fluctuating sensorium and classical thalamic syndrome will be present.

Basal ganglia : Parkinsonian phenomena, hyperkinetic movement disorders.

LMN approach summary

00:46:00

LMN



Peripheral nerve involvement : Axonal neuropathy presents with wasting.

APPROACH TO UMN DISORDERS

----- Active space -----

UMN structures :

- Cortex.
- Sub cortex.
- Brainstem :
 - **Crossed hemiplegia** : Ipsilateral cranial nerve palsy + Contralateral hemiplegia.
 - Cranial nerves symptoms.
 - Vertigo : Also seen in insular cortex involvement.
- Spinal cord :
 - Sensory level.
 - Bladder involvement (Depending on level of spinal cord involved).
 - Tract pain.

Cortical involvement in UMN lesion

00:03:28

Loss of consciousness : Classical presentation.

Seizure : Origin is cortex.

Cortex predominantly is grey matter.

Different lobes of cortex :

Frontal lobe :

medial frontal cortex :

- Containing anterior singulum.
- Lack of enrgy/**apathy**.
- **Akinetic mutism**.

Dorsolateral prefrontal cortex :

- **Executive dysfunction** : Not able to do planned & sequential activity.

Orbitofrontal area :

- Personality problem.
- Disinhibition.

----- Active space -----

motor cortex :

- Weakness predominantly involves face + upper limb.
- Distal > Proximal.
- Dextrective loss.
- Loss of fine activities : Pyramidal dysfunction.

Parietal lobe :

Function : Calculation, language, apraxias, proprioception.

- Left parietal :
 - Involved in calculation & language apraxia.
 - Dominant.
 - Lesion of angular gyrus produce Gerstman's syndrome (Finger agnosia, agraphia, acalculia).
- Right parietal :
 - Dressing apraxia.
 - Constructional apraxia.
 - Neglect.
 - Geographical.

Temporal lobe :

- Lateral temporal lobe :
 - Auditory area.
 - Wernicke's area.
 - Visual processing.
- medial temporal lobe :
 - memory.
 - Olfaction.
 - Limbic part.

most epileptogenic area.

Occipital lobe :

Occipital blindness : Pupillary reflex unaffected, but patient c/o visual loss.

- Retina → Fibres cross at chiasma → Lateral geniculate body (LGB).
- Pupillary fibres before reaching LGB descends to mid brain to form pupillary reflex.
- When retina/optic nerve/optic chiasma involved : Pupillary reflex affected.

----- Active space -----

| | Broca's area | Wernicke's area | TCM | TCS | TC mixed |
|---------------|--------------|-----------------|-----|-----|---------------|
| Naming/anomia | - | - | - | - | - |
| Fluency | - | + | - | + | - |
| Comprehension | + | - | + | - | - |
| Repetition | - | - | + | + | +, Echolalia. |
| Naming | - | - | - | - | - |

