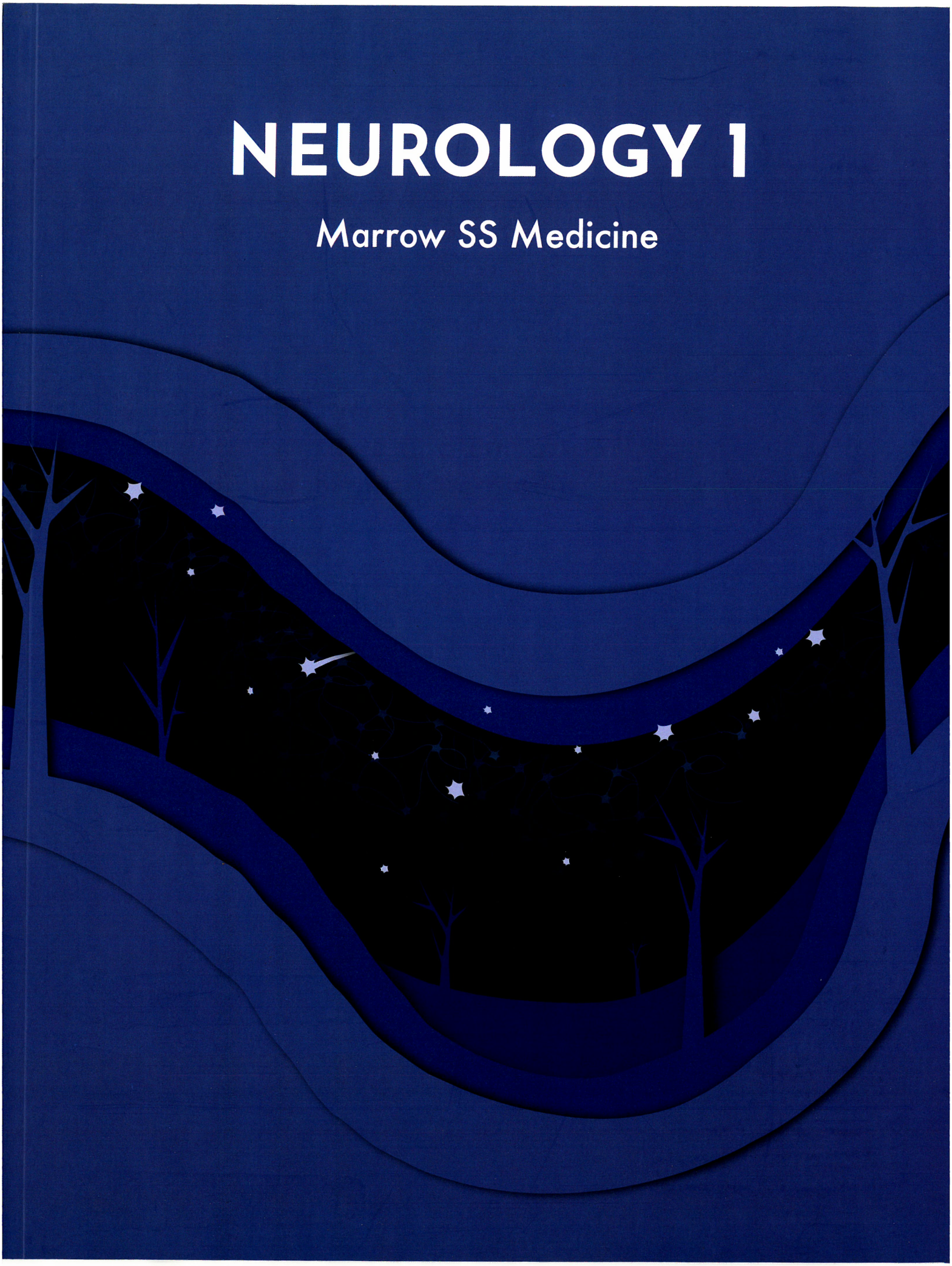
NEUROLOGY 1

Marrow SS Medicine



Instructions

- The notes must be used in conjunction with the Marrow SS Medicine videos and should not be considered standalone material.

Please note:

- The information in this book has been printed based on the transcript of the Marrow SS Medicine videos.
- The information contained in this book is for educational purposes only. The content provided is not intended to substitute for professional medical advice, diagnosis or treatment.
- This book cannot be sold separately. It has been made available to only select eligible users who have an active subscription to Marrow SS Medicine videos.
- The text, images, slides, and other materials used in this book have been contributed by the faculty, who are subject matter experts. We have merely reproduced them as video transcripts in this book.
- The notes have been consciously designed in a way that is concise and revisable. To ensure this, we have intentionally added only the most relevant modules and images that are needed for you.
- Reasonable care has been taken to ensure the accuracy of the information provided in this book. Neither the faculty nor Marrow takes any responsibility for any liability or damages resulting from applying the information provided in this book.
- This set contains notes of all main videos published in the app until Jan 2025. You can find notes for any additional videos published after this date within the app under the videos section.

//ME801SS-SAP

Contents

Volume - 1

Neuro-Ophthalmology

Cranial Nerves

Cognition and Dementia

Hypokinetic Movement Disorders

Hyperkinetic Movement Disorders

Volume - 2

Stroke and Vascular Disorders

Epilepsy

Headache

Demyelinating Disease

Cerebellum and Ataxia

Neuroinfection and Other Specific Conditions

Spinal Cord

Motor Neuron Disease

EEG

Neuromuscular Junction (NMJ) Disorders

APPROACH TO NEUROLOGY

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, palpations, 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 1hour.
- 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) Fasiculation	Absent	Present
5) Tone	Increased	Flacid
6) DTR	Exagerated	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 muscules.
- From spinal cord, it is transmitted to the limbs and trunk.

In the presence of extra pathways :

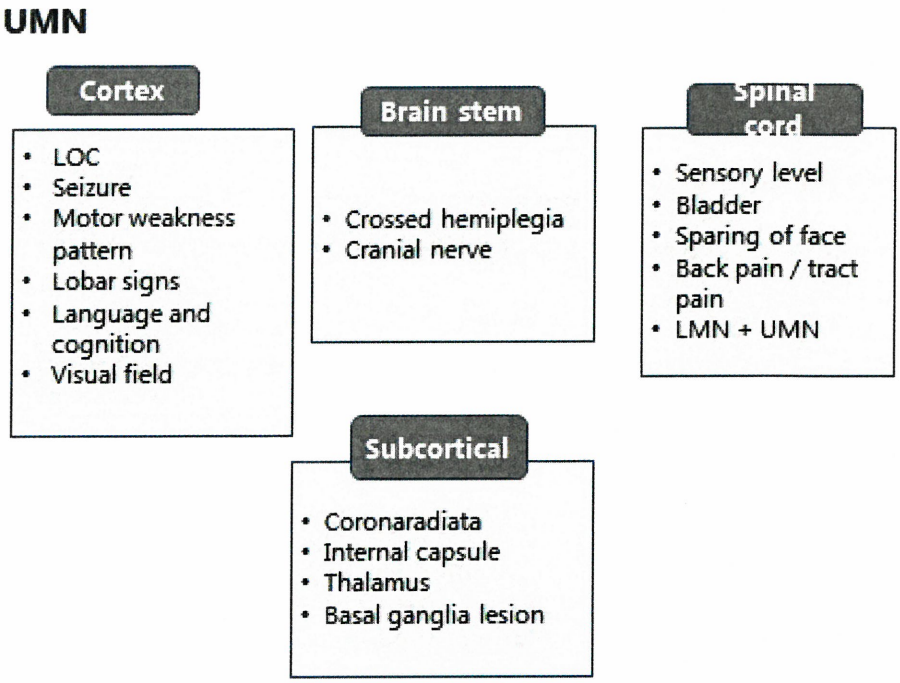
- Basal ganglia and cerebellum helps in further fine-tuning(co-ordination) of activities.
- The motor signals pass from the motor cortex through the basal ganglia to the Internal capsule.
- The cortico-pondo cerebellar fibres carry the signal to the cerebellum which inturn gives back the signal to cerebrum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

Affection of posterior pareital 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 ---



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 hemoplegia :

- In brainstem the UMN fibres are descending into the nucleus of the cranial nerve → Supplies the ipsilateral muscle or craniofacial structures.
- The pyramidal fibres descends downwards and crosses 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 myloradiculopathy or anterior horn involvement.

--- Active space -----

Subcortical :

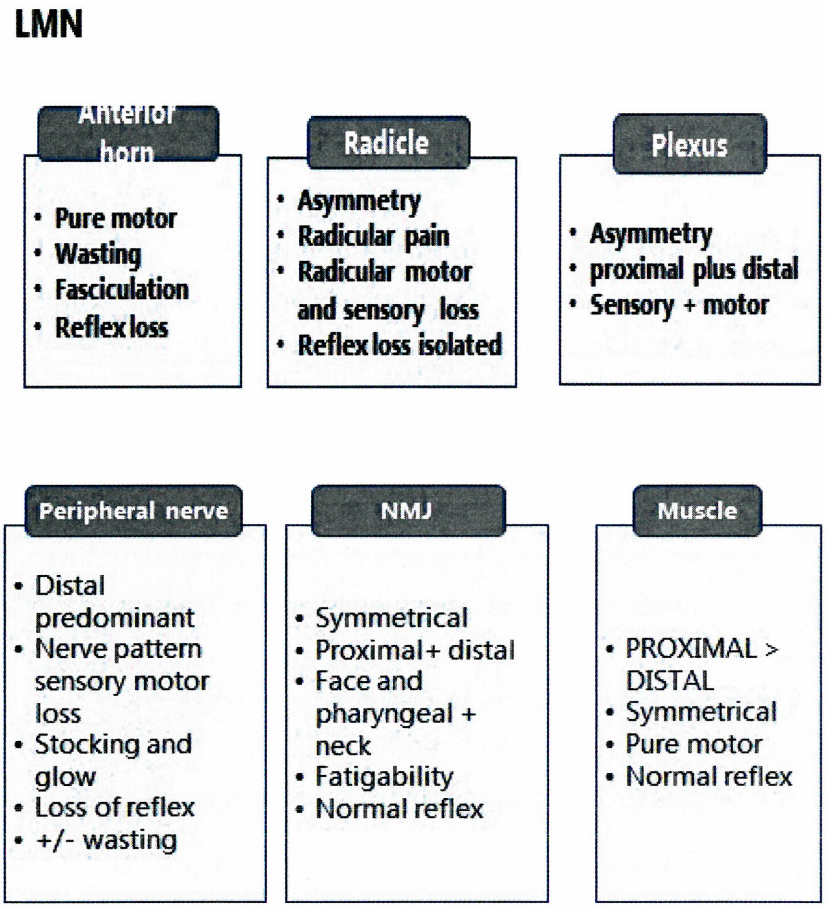
Corona radiata :

- 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 weaknes 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



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 gyras 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 reflect affected.

Language

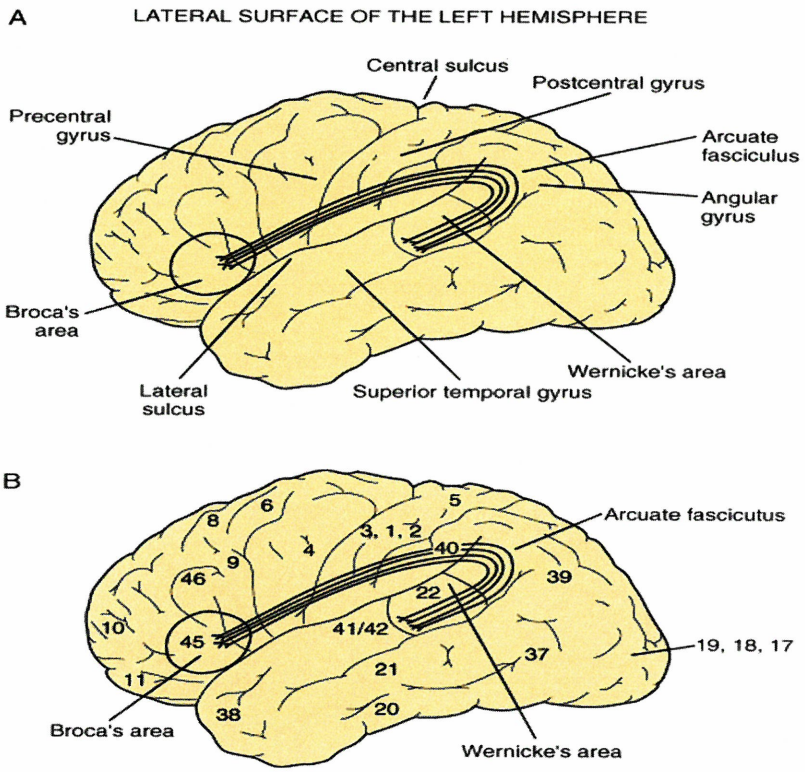
00:16:40

----- Active space ----

Language is the basic concept of communication through writing, speaking, gesture etc.
Motor output of languages is speech using larynx, pharynx.

Mechanism :

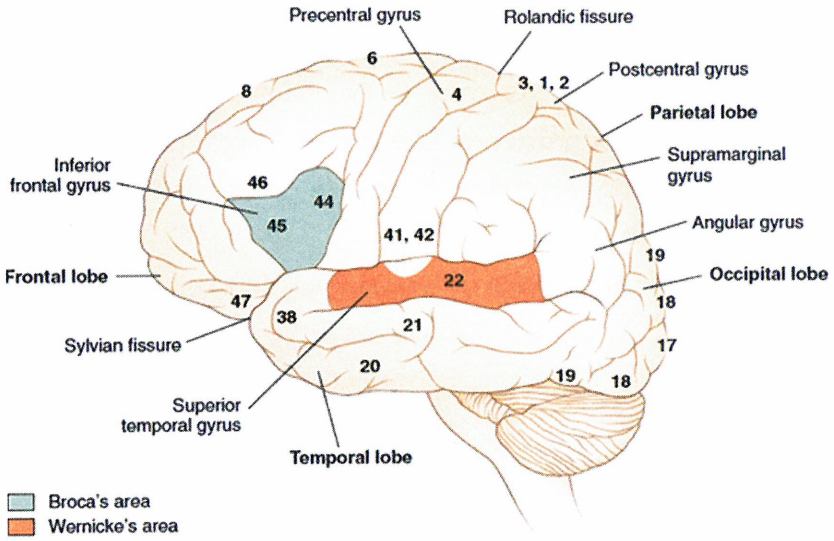
Sound falls on ears → Heschl's gyrus identifies → Tone of sound identified by primary auditory area → Wernicke's area & temporal parietal area function is Comprehension (Contains internal dictionary Lexicon) → Carried by the arcuate fasciculus → Reaches the Brocas area → Produces reply by activating motor neurons → Produce syntax grammar fluency → Supplementary motor area (Planning and programming) → motor cortex → Descends to brainstem cranial nerve nucleus → Speech output.



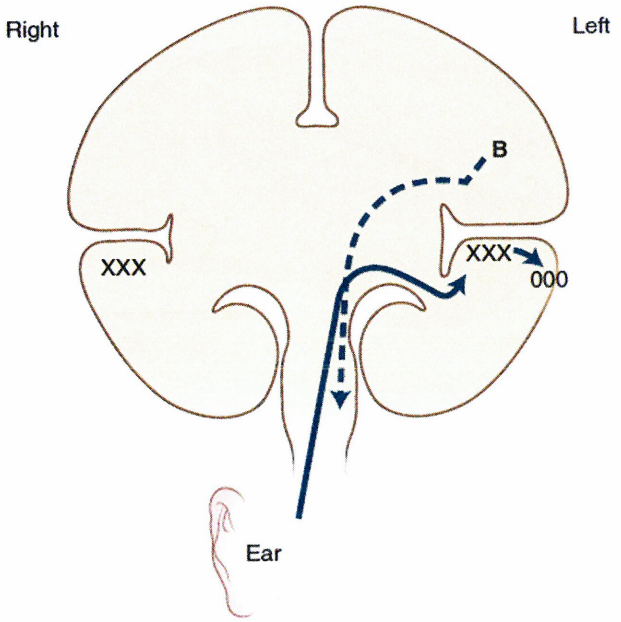
- Right > Left side :
  - Limbic reward system gives emotional intonation.
  - Prosody (Rhythmic sound of speech).
- Rest of language concept lies in left side.
- Intentional activation system cannot activate Broca's area causing akinetic mutism (No movement, no talk).
- Any lesion anterior to central sulcus → Fluency lost.
  Any lesion posterior to central sulcus → Comprehension lost.
  Any lesion involving Perisylvian circuit → Repetition lost.

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

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



Broadmann area



mechanism of language and speech