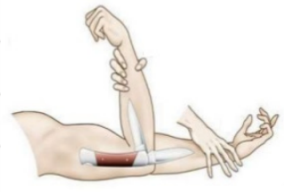
LEC 6 (CONT.) & LEC 7

Motor neuron leisons

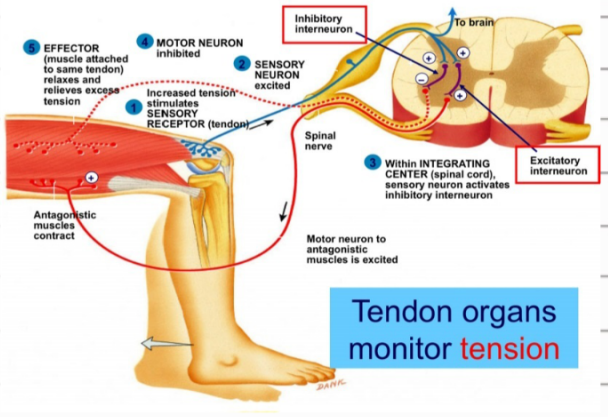
Features	Upper motor neuron lesions(UMN)	Lower motor neuron lesion(LMN)
	UMN starts from motor cortex to the cranial nerve nuclei in brain and anterior horn cells in spinal cord	LMN is the motor pathway from anterior horn cell(or Cranial nerve nucleus)via peripheral nerve to the motor end plate
Bulk of muscles	No wasting	Wasting of the affected muscles (atrophy)
Tone of muscles	Tone increases (Hypertonia)	Tone decreases (Hypotonia)
Power of muscles	Paralysis affects movements of group of muscles Spastic/ clasp knife	Individual muscles is paralyzed Flaccid (flaccid paralysis)
Reflexes	Exaggerated. (Hyperreflexia)	diminished or absent. (Hyporeflexia)
Fasciculation	Absent	Present
Babinski sign	Present	Absent
clasp-knife reaction	Present	Absent
Clonus	Present	Absent

- hypertonia & hyperreflexia are a result of ↑ & motor neuron activity

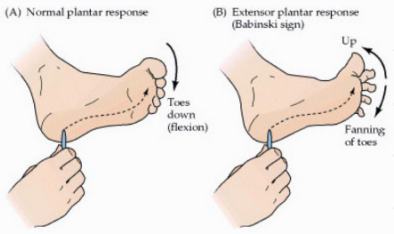
Clasp knife rxn:



- when a clasp knife is open (fully extended) & you try to close it, you face initial resistance but after a specific point it closes suddenly
- a patient with UMN lesion faces will also show initial resistance when his hand is flexed & the doctor tries to extend it
- clasp knife rxn phenomena
  - ① initial resistance (exaggerated stretch reflex) when you stretch a muscle it resists stretching by contracting, pts with UMN leison has bigger reflex effect
  - ② sudden release (activation of golgi tendon reflex, anti stretch reflex) resists excessive contraction
- Tendon reflex ex:
  there is a contraction in quadriceps M → ↑tension → ⊕ golgi tendon organs (sensory receptor organs → ⊕ 2 interneurons in spinal cord:
  - ① inhibitory interneuron → ⊖ LMN going to the muscle to stop contracting (poly synaptic reflex: ⊖ interneuron bet. 2 excitatory neurons)
  - ② excitatory interneuron → ⊕ LMN going to the antagonist M, hamstring in this ex (law of reciprocal innervation: contraction of a muscle is accompanied with simultaneous relaxation of antagonist muscle (or a set of muscles)



Babinski Sign



- test: Stimulate lat. aspect of sole of foot
  - ↳ Normal response: flexion of toes
  - ↳ LMN pts response: fanning of toes with dorsi flexion of the big toe (babinski)
- in Children (1-1.5 yrs) positive babinski is normal (pyramidal tract is not fully developed & myelinated well)
- UMN lesion usually happens in both pyramidal & extrapyramidal tracts, most explanations of this phenomenon (Clasp knife, hypertonia, ...) are related to extrapyramidal tracts except babinski

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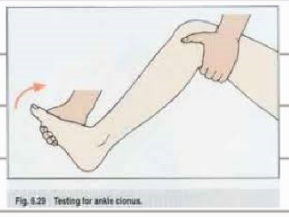
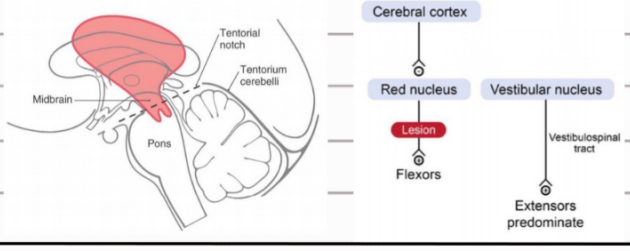
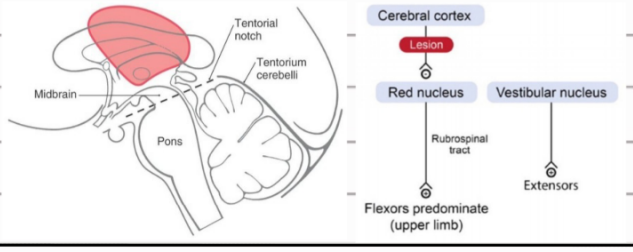
Clonus



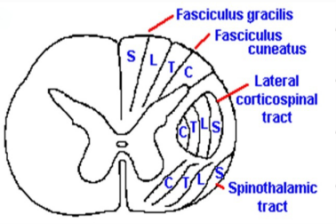
- test: attempt to dorsiflex foot & face resistance
  - ↳ Normal response: sudden release of foot → normal dorsiflexion
  - ↳ UMN lesion pts response: dorsiflexion then clonus starts (rythmic contraction & relaxation of muscles)

Deceberate & Decorticate rigidity:

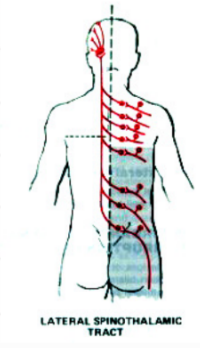
	Decerebate	Decorticate
level of lesion	lower than red nucleus 	higher than red nucleus 
rigidity & limb position	Complete rigidity, upper & lower limbs are extended 	Complete rigidity, lower limbs are extended & upper are flexed & rigid 
affected tract	rubrospinal	pontine reticulospinal (tonically active)
effect	more fatal cuz lesion is closer to brain stem (vital center)	less fatal

Clinical Significance of lamination of ascending tracts:

- ext. pressure on Spinothalamic region of Spinal Cord → loss of pain & temp in Sacral dermatome
- ↑ pressure → other higher dermatomes affected
- Spinothalamic tracts (Cervical to Sacral) are located med. to lat.



- intramedullary tumor → medial effect (Cervical)
- extramedullary tumor → lateral effect (Sacral)

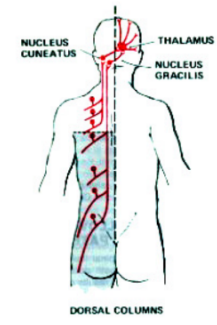


① Destruction of LSTT (lat. spinothalamic tract)

- loss of pain & temp sensation on Contralateral Side (decussation happened at the level of spinal Cord)

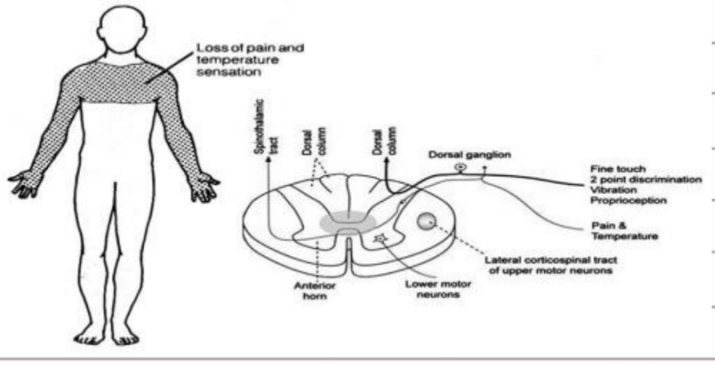
② Destruction of post. column (fasiculus & cuneatus)

- loss of muscle-joint sensation, position, vibration, tactile discrimination sensation on ipsilateral Side (decussation happened at the level of medulla)

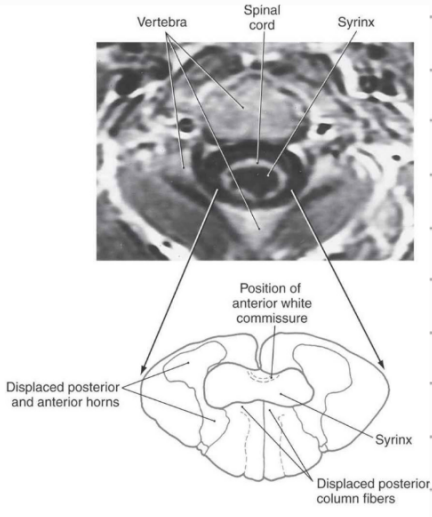


③ Syringomyelia

- Cavitation in Central Canal of Spinal Cord → damage to fibers crossing in ant. white commissure in both directions → bilateral loss of pain & thermal sensation



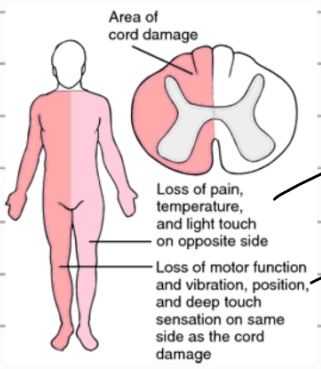
- ex: level C_4-C_5 Syringomyelia: loss of sensation over shoulders down to level of nipple.
- in some cases cavitation extends to ant. horns causing muscle weakness & paralysis (ipsilateral if it affected one side)



- Crossing of nerves occurs in a tilted way (ex: 1st order neuron entered at C_3 then begins to cross over until it reaches C_5

④ Brown Séquard Syndrome

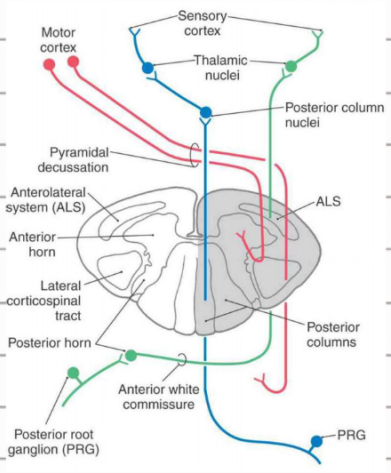
- functional hemisection of spinal cord (1/2 spinal cord damage) → damage to: corticospinal tract, ALS, post. columns
- type of damage:



a) ipsilateral → paralysis or weakness, (hemiparesis, hemiplegia)

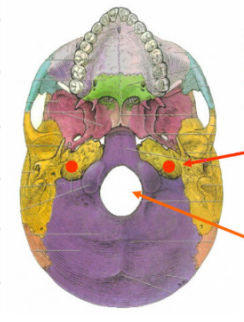
b) contralateral → nociceptive & thermal below lesion

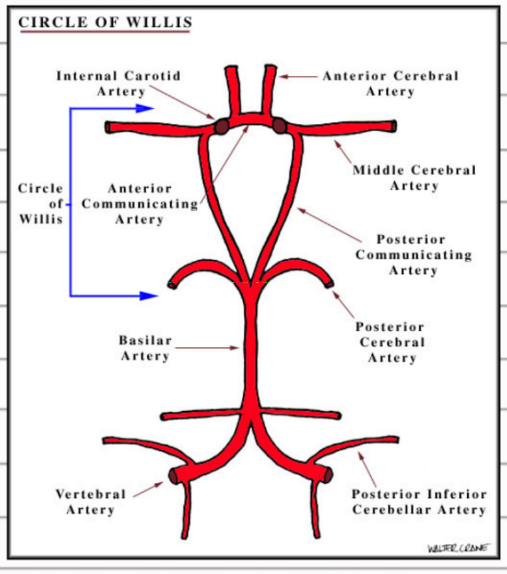
c) ipsilateral → discriminative, tactile, vibratory, position sense below lesion

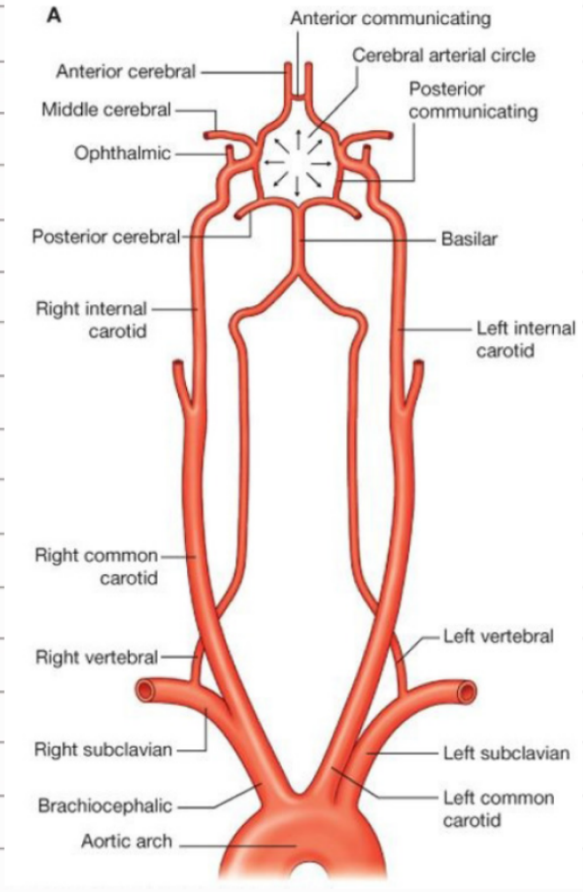


Arterial supply of brain

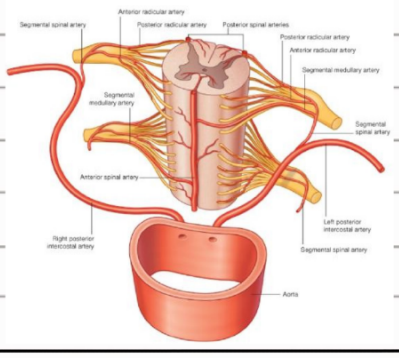
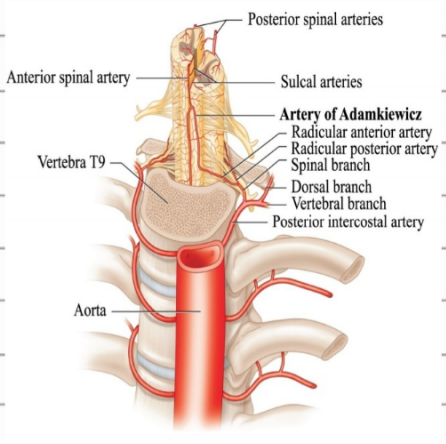
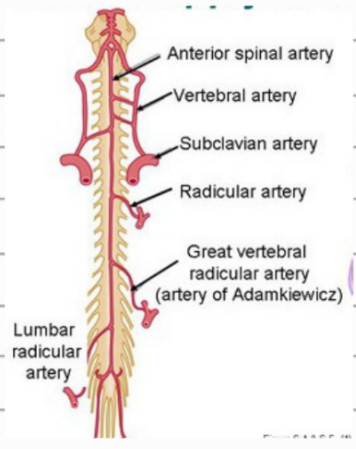
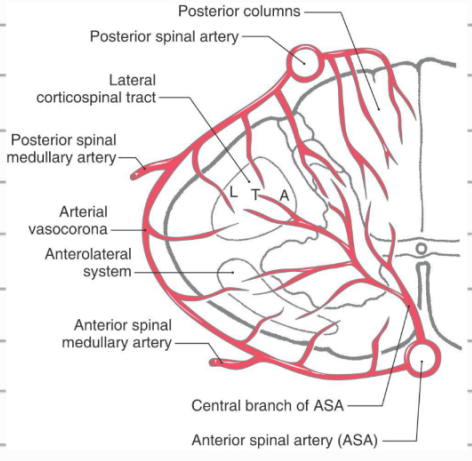
- pair of ICA → enter skull through carotid canal (in petrous part of temporal bone)
- pair of vertebral arteries → enter skull through foramen magnum (in occipital bone)
- their branches anastomose on inf. surface of brain & form circle of willis
- the 4 arteries lie in subarachnoid space, 2 vertebral arteries unite into basilar artery







Arterial Supply of Spinal Cord:

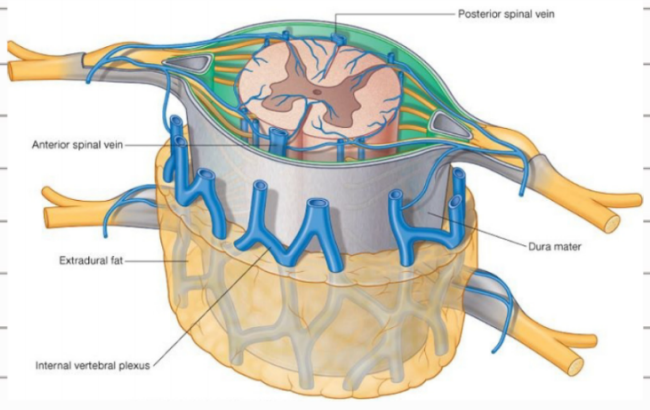
artery	arteries or branches	arise from	pass through
longitudinal	1 ant. spinal artery	vertebral arteries	ant. median fissure
	2 post. spinal arteries	ant. post. cerebellar A (branch of vertebral)	posterolateral sulcus
Segmental horizontal spinal	each segmental A gives: • post. radical A (runs with post. dorsal root to reach spinal cord) • ant. radical A (runs with ant. ventral root to reach spinal cord • segmental medullary A (anastomose with ant. spinal A)	• vertebral As & deep cervical As • post. intercostal As • lumbar As 	intervertebral foramen 
Adamkiewicz	anastomose with ant. spinal A 	left post. intercostal A (branches from aorta) & supplies lower $\frac{2}{3}$ of spinal cord	usually on the left 

- arterial supply of spinal cord at level of segments: terminal branches of spinal medullary As join to form arterial vasocorona
  1. post. spinal As & arterial vasocorona → post. columns & peripheral parts of lat. funiculi
  2. ant. spinal A → most grey matter & adjacent white matter

Venous drainage of Spinal Cord

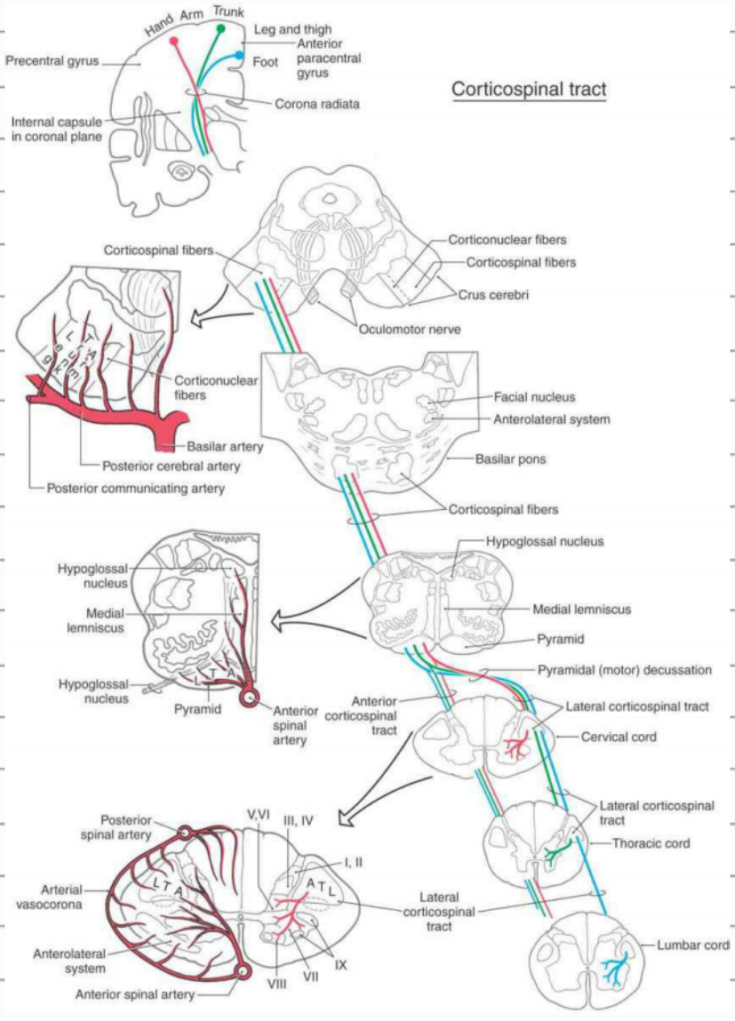
① 2 pairs of pairs on each side
② 1 midline channel parallels ant. median fissure (ant. spinal V)
③ 1 middle channel passes along post. median sulcus (post. spinal V)

- these veins drain into extensive internal vertebral plexus in epidural space of vertebral canal → drain into segmentally arranged vessels → connect with major systemic veins (azygos system & intra cranial veins)



Central Cord Syndrome

Neck hyper extension → occlusion of blood supply of ant. spinal A → bilateral weakness in extremities (more in upper limbs) + bilateral pain & thermal sense loss + bladder dysfxn



- Why bilateral?
  Cuz we have 1 ant. spinal A that supplies both sides
- Why more in upper limbs?
  Cuz ant. spinal A arises from vertebral A (blood is coming from above so upper limbs are more affected) + lower extremities have another supply (adamkiewicz A)
- ↓ blood flow in post. spinal A → ipsilateral reduction or loss of discriminative positional, vibratory, tactile sense below lesion

by INSAF IYAD