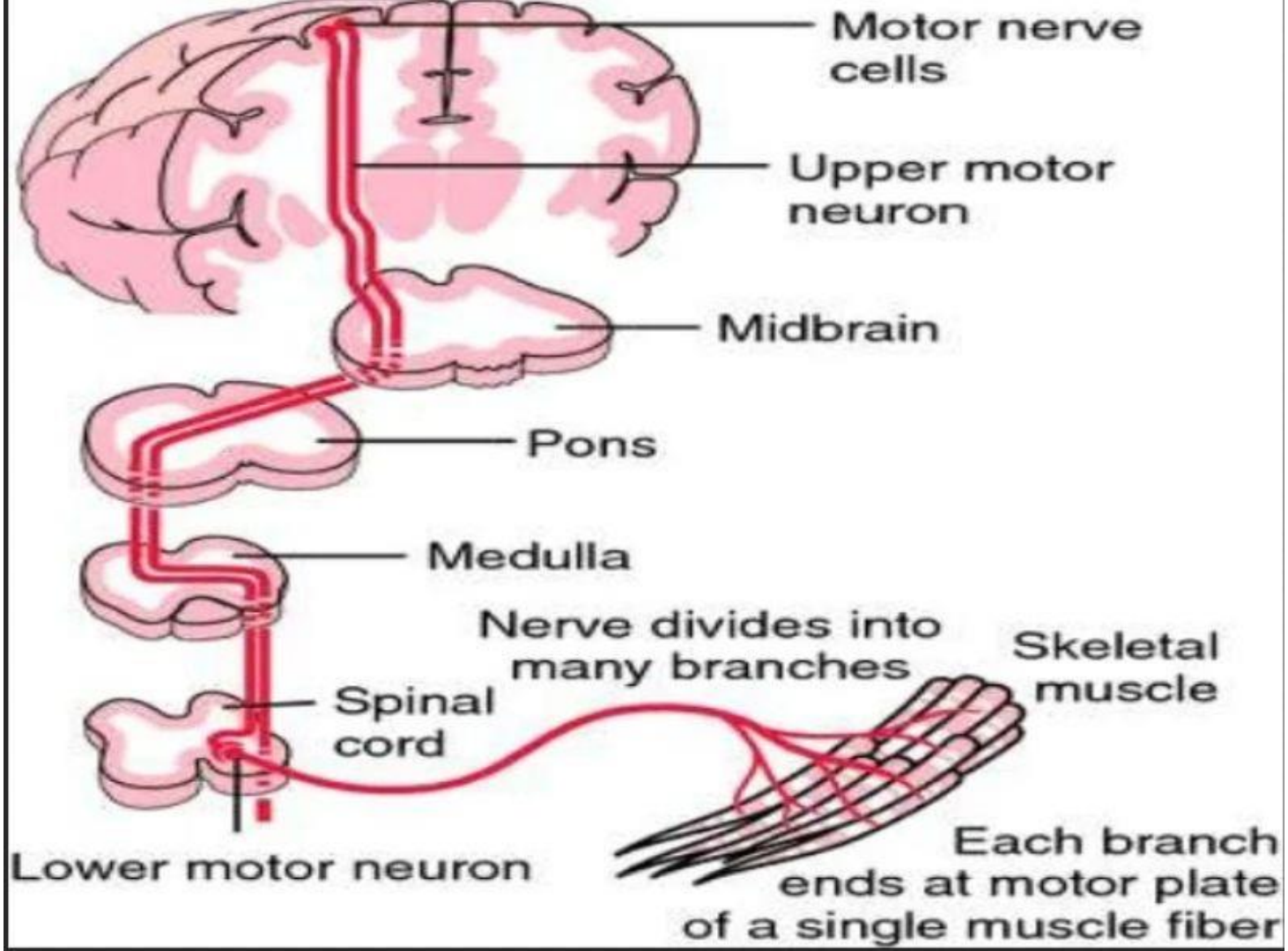
Upper Motor and Lower Motor Neuron Lesion

UMN and LMN lesion

The upper and lower motor neurons together comprise a two-neuron pathway that is responsible for movement.

The pyramidal tract provides a direct pathway between the cerebral cortex and the spinal cord, in contrast with extra-pyramidal tracts which provide indirect pathways for the coordination of movement

The pyramidal tract divides into the corticospinal tract and the corticobulbar tract. Corticospinal tract fibers synapse with spinal nerves while corticobulbar fibers synapse with cranial nerves.


Motor nerve cells
Upper motor neuron
Midbrain
Pons
Medulla
Nerve divides into many branches
Skeletal muscle
Spinal cord
Lower motor neuron
Each branch ends at motor plate of a single muscle fiber

PYRAMIDAL TRACTS

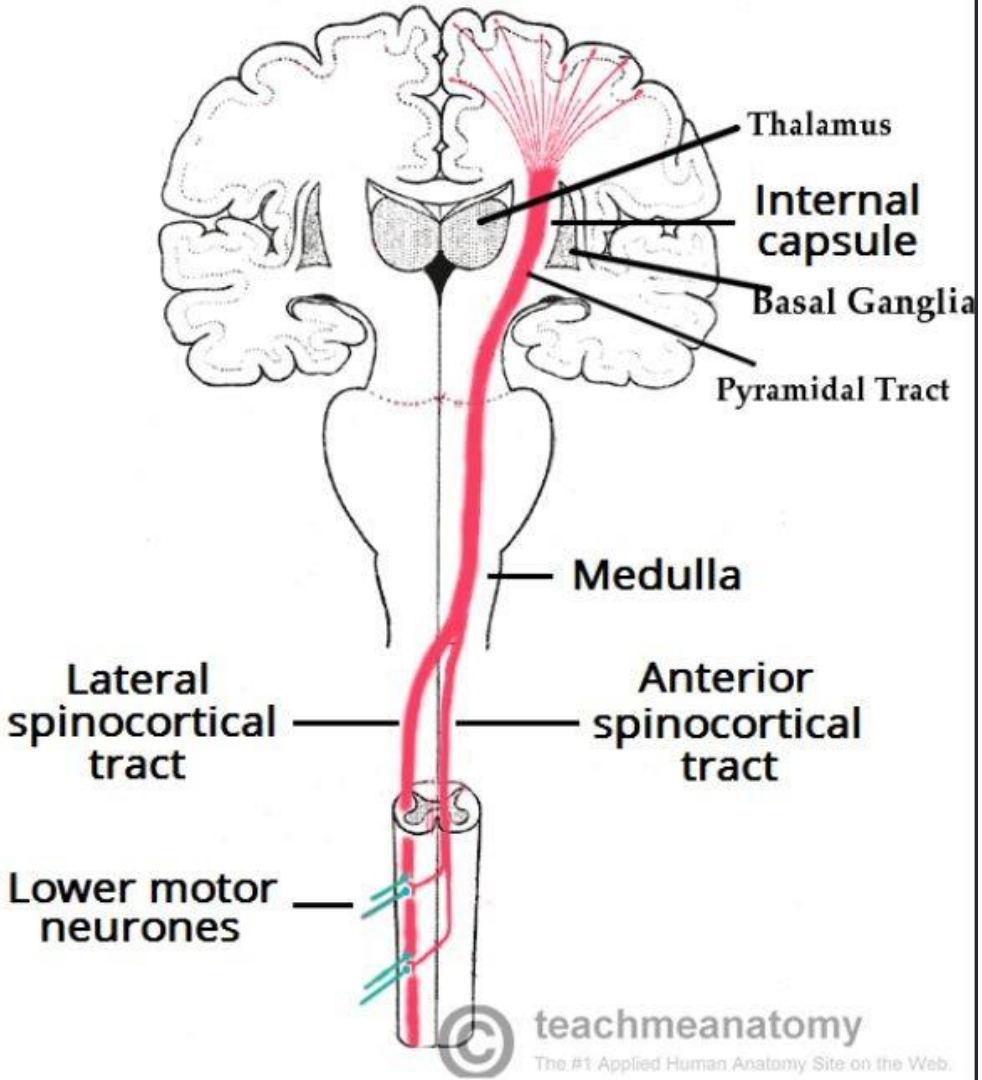
- corticospinal tract

EXTRAPYRAMIDAL TRACTS-

- Reticulospinal
- Olivospinal
- Vestibulospinal
- Tectospinal
- Rubrospinal tract
- Corticobulbar tract
- Corticorubral tract

Descending Pathways

Pathway	Upper limb	Lower limb
Cortico/-pyramidal	This Tract functions to modulate the activity of Alpha or Gamma Motor Neurons as directed by the Motor Cortex.	
Rubro-spinal	Stimulates flexors	
Reticulo-spinal	Medullary inhibits extensors and excites flexors Pontine excites extensors and inhibits flexors (Generally upper limb)	
Vestibulo-spinal	Doesn't affect upper limbs but helps position head and neck in response to body tilting (medial)	Stimulates extensors (lateral)
Tecto-spinal	Control of head, neck and eye movements.	


Thalamus
Internal capsule
Basal Ganglia
Pyramidal Tract
Medulla
Lateral spinocortical tract
Anterior spinocortical tract
Lower motor neurones
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Anatomy

-The pathway of the corticospinal tract descends through the cerebral peduncle in the midbrain, ventral pons, and the pyramids of the medulla.

-At the inferior aspect of the medulla, the majority of corticospinal tract axons decussate at the pyramidal decussation.

-The axons continue their descent contralateral from their cell bodies of origin and enter the spinal cord at the lateral funiculus, The tract is now the lateral corticospinal tract.

-At the pyramidal decussation, approximately 10% of the corticospinal tract axons do not decussate and continue their descent down the brain ipsilateral to their cell bodies of origin, These fibers enter the ventral aspect of the spinal cord and are known as the anterior corticospinal tract.

This crossing means that an upper motor neuron lesion above the medulla will cause symptoms on the contralateral side of the body. However, a lesion to the lateral corticospinal tract after it decussates will present on the ipsilateral side of the body.

Lower motor neurons transmit impulses via spinal peripheral nerves or cranial nerves to skeletal muscles.

DIFFERENCE BETWEEN UPPER & LOWER MOTOR NEURON LESION

UMN LESION	LMN LESION
• Paralysis affect movement rather than muscles	• Individual muscle or group of muscles are affected.
• Muscle wasting is only from disuse, therefore slight. Occasionally marked in chronic severe lesions.	• Wasting pronounced.
• Spasticity of clasp-knife' type. Muscles hypertonic.	• Flaccidity. Muscles hypotonic.

- Tendon reflexes increased. Clonus often present.
- Superficial reflexes diminished or modified.

  Abdominal reflex absent.

  Babinski's sign +ve, Increased jaw jerk.

- Tendon reflexes diminished or absent.
- Superficial reflexes often unaltered.

Clinical Significance

The symptoms of UMN lesion can include:

Spasticity

The definition of spasticity is a velocity-dependent increase in a muscle's resistance to a passive stretch. Slow passive movements of the arms or legs will not elicit the increased resistance. Brisk stretches of muscles will cause an abrupt increase in tone followed by a decrease in muscular resistance with continued stretch. This phenomenon is called clasp-knife rigidity. The antigravity muscles of the arms and legs are most affected. These include the flexors of the arms and the extensors of the leg. Because of the decreased modulation of spinal reflexes in UMN syndrome, patients will often exhibit flexor and extensor spasms.

Clonus

Clonus is a sequence of rhythmic, involuntary muscle contractions. These contractions occur at a frequency of 5 to 7 Hz and are a response to abruptly applied stretch stimuli. Clonus is most easily elicited at the ankle with brisk dorsiflexion and plantar-flexion movements. Clonus can also be observed during voluntary movement or through cutaneous stimulation.

Hyperreflexia of deep tendon reflexes

Patients can be seen to have abnormally brisk reflexes which are due to decreased modulation by descending inhibitory pathways. Radiation of reflexes is a regular observation with the hyperreflexia of UMN lesions. For example, tapping of the supra-patellar tendon would elicit a knee-jerk reflex.

Hyporeflexia of superficial reflexes

The superficial abdominal reflex and the cremasteric reflex are seen to be decreased or abolished following UMN lesions. The superficial abdominal reflex is the tensing of abdominal by stroking the overlying skin while the cremasteric reflex is the elevation of the scrotum in response to stroking the medial thigh.

Synkinesias

Synkinesias are involuntary movements in a limb that have associations with the voluntary movements in other limbs. For example, flexion of the arm may result in flexion of the leg. These involuntary movements can also occur with yawning or sneezing. Volitional movements of one arm or leg may also result in mirror movements of the opposite limb.

Co-Contraction

Co-contraction is defined as the simultaneous contraction of agonist and antagonist muscles around a joint. This increases the stiffness and stabilization around the joint which prepares it for activity in healthy individuals. The pathological co-contraction in UMN lesions causes a decreased rate of rapid alternating movements and creates greater fatigability for voluntary movements in weakened muscles.

Babinski Sign and other reflexes

The Babinski sign can be elicited by stroking the sole of the foot with a firm stimulus. The normal adult response is plantar-flexion. The sign is positive when the application of the stimulus elicits extension of the large toe and fanning of the other toes. The Babinski sign is known to be a normal response in infants before full maturation of the corticospinal tract. However, in adults, a positive sign is indicative of underlying UMN damage. Other reflexes exist which represent lesions of the corticospinal tract. The Brissaud reflex is linked with the extensor response of the Babinski sign and is positive when stroking the sole of the foot elicits contraction of the tensor fascia latae of the ipsilateral leg. The Hoffman sign is an analog of the Babinski reflex for the upper limbs. The test is performed by loosely holding the patient's middle finger and quickly flicking the fingernail downward. A positive sign is the flexion and adduction of the thumb

Pseudobulbar Palsy

As previously stated, most cranial nerves have bilateral innervation from the brain with the exception of CN VII and CN XII. The muscles of cranial nerves with bilateral innervation include the eyes, jaw, pharynx, upper face, larynx, and neck. These muscles would only show deficits with bilateral UMN lesions. Bilateral damage of UMN's to cranial nerves is known as a pseudobulbar palsy. Slurred speech is often the first presenting symptom. Other characteristic deficits include dysphagia, dysarthria, brisk jaw jerk, spastic tongue, and pseudobulbar affect

CN VII and CN XII UMN lesions

CN VII and CN XII innervate muscles of the lower face and the tongue, respectively. These cranial nerves receive unilateral innervation from the pyramidal tract. Unilateral lesions of UMN's to CN VII or CN XII would manifest as a lower facial droop or tongue deviation away from the side of the lesion, respectively.

Spinal Shock

Spinal shock refers to the period of acute flaccid paralysis following spinal cord injury. Hypotonia and hyporeflexia are the most characteristic symptoms. The paralysis is most evident in the arms and legs with the preservation of truncal musculature. The duration can range from a few days to weeks after which spasticity and hyperreflexia replace the prior symptoms. The symptoms of spinal shock are most pronounced with lesions of the spinal cord versus cerebral lesions.

Features and manifestation of LMNL :

1- Motor affection:

- Flaccid paralysis : Defection in the muscle.

- Muscle wasting : Atrophy in the muscle due to losing of muscle function (Muscle can not contract voluntary but reflex).

- Atonia : complete loss of muscle tone as the nerve fibers is affected**.**

- Facial nerve: it affects one half of face.

2- Reflex affection in LMNL:

- Deep reflex : Loss of deep reflex.

-superficial reflex : still going if the muscle isn't affected.

-Absence of tonic neck reflex.

Diseases associated with UMN Type Lesion

Detailed patient history and a complete physical exam are essential for differentiating the cause of UMN lesions. Some diseases that can damage upper motor neurons include cerebrovascular accidents, amyotrophic lateral sclerosis, primary lateral sclerosis, multiple sclerosis, Brown-Sequard Syndrome, vitamin B12 deficiency.

Cerebrovascular Accidents

Cerebrovascular accidents or strokes are the sudden cessations of blood flow to areas of the brain leading to cell death. Strokes fall into two etiologic categories, being either ischemic or hemorrhagic. Ischemic strokes are the sudden interruption of blood flow to the brain which can be due to thrombi, emboli, or compression. Hemorrhagic strokes are characterized by bleeding into the brain due to the rupture of a blood vessel. The cerebral blood supply has many branches which supply different areas of the brain. Occlusion of the middle cerebral artery or anterior cerebral artery can damage the motor areas of the cerebral cortex. Given the extensive areas of the brain supplied by the middle cerebral and anterior cerebral artery, strokes in those regions are likely to present with sensory, language, perceptual, and visual deficits in addition to UMN signs. Occlusion of the lenticulostriate arteries can damage the internal capsule. A stroke that targets the posterior limb of the internal capsule presents with pure motor deficits of the contralateral face, arm, and leg. Occlusion of the various branches of the vertebral artery or basilar artery can lead to strokes in different areas of the brainstem. Notable brainstem strokes that damage the corticospinal tract are medial medullary syndrome, medial pontine syndrome, and Weber Syndrome.

Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) is the most prevalent neurodegenerative disease that is characterized by its involvement of both upper and lower motor neurons. Hence, the clinical presentation is a combination of upper motor signs and lower motor neuron signs. Nerve conduction studies and electromyography are utilized to confirm the diagnosis. Labs are generally used to rule out other disease processes that can manifest with weakness in patients. ALS is currently incurable however various treatments have been developed to extend life in patients. Riluzole is a glutamate pathway antagonist that is the only current drug shown to extend life in patients with ALS.

Primary Lateral Sclerosis

Primary lateral sclerosis (PLS) is a neurodegenerative disorder that targets upper motor neurons. PLS is generally seen in adults and is sporadic in nature, though hereditary variants have been observed. Compared to ALS, PLS has a slower progression and lacks lower motor neuron signs. However, some individuals with PLS do develop lower motor neuron signs as their disease progresses. The condition would then be considered upper motor neuron onset ALS. There are no cures for PLS and treatment is aimed at alleviating symptoms of spasticity and weakness through medications and physical therapy.

Brown-Sequard Syndrome

Brown-Sequard Syndrome is a spinal cord lesion caused by a hemisection injury to the spinal cord. The most common etiology is from penetrating trauma to the spine. However, other etiologies include blunt trauma, hematoma, tumors, or disc herniation. As a result of the hemisection of the spinal cord, the symptoms are manifestations of damage to the lateral corticospinal tract, dorsal column, and the lateral spinothalamic tract. Patients present with upper motor neuron signs ipsilateral and below the level of the lesion. Patients will also present with ipsilateral loss of fine touch, vibration, and proprioception in addition to the contralateral loss of pain and temperature sensation.

Multiple Sclerosis

Multiple Sclerosis (MS) is an immune-mediated, inflammatory demyelinating disease. The symptomatology of MS is characterized by episodes that occur in different anatomic locations in the central nervous system and occur months or years apart. The presenting symptoms of patients are highly variable. Symptoms can include cognitive disturbance, visual changes, hemiparesis, ataxia, and sensory deficits. The UMN signs of MS are due to the demyelination of upper motor neurons. MRI is the imaging test of choice used to diagnose MS. CSF studies may also be used to aid in diagnosis. Oligoclonal bands and intrathecal immunoglobulin G are classically seen in the CSF of MS patients.

Vitamin B12 Deficiency

The most prevalent etiologies of vitamin B12 deficiency are pernicious anemia, bariatric surgery, small intestine surgery, and gastritis. Other etiologies include pancreatic insufficiency, inadequate dietary intake, and drug side effects. Vitamin B12 deficiency causes degeneration of the dorsal column and lateral white matter of the spinal cord. This can lead to degeneration of the lateral corticospinal tract with subsequent UMN signs. Degeneration of the dorsal column manifests as sensory ataxia. Deficiency also leads to macrocytic anemia. Supplementation with vitamin B12 generally corrects the anemia and stops the progression of the degeneration of the spinal cord.

Diseases associated with LMN Type of Lesion

Poliomyelitis

A classic example of solely LMN paralysis, poliomyelitis, has a fecal-oral transmission and is caused by a type of picornavirus: poliovirus. Once infected, the virus replicated in the oropharynx and small intestine before spreading via the bloodstream to the CNS. While replicating in the Peyer's patches of the small intestine, 95% of patients are asymptomatic, and it can only be found in the stool or via an oral swab.In the CNS, the virus destroys the anterior (ventral) horn of the spinal cord, resulting in LMN paralysis. Because LMNs originate in the anterior horn of the spinal cord, this results in LMN signs such as *asymmetric* weakness, flaccid paralysis, fasciculations, hyporeflexia, and muscle atrophy. The infection could also result in respiratory involvement leading to respiratory paralysis. Other systemic signs of infection include fever, headache, nausea, and malaise. The cerebrospinal fluid would demonstrate increased WBC's and a slight elevation of protein, which is consistent with the viral infection. Once a prominent cause of paralysis, poliomyelitis has almost been eradicated due to widespread vaccination.

Spinal Muscular Atrophy

This disease is due to congenital degeneration of the anterior horn of the spinal cord. Unlike polio, this results in *symmetric* weakness, flaccid paralysis, fasciculations, hyporeflexia, and muscle atrophy. Because it is congenital, it has classically had associations with a "floppy baby" with marked hypotonia and tongue fasciculations. This disease carries an autosomal recessive inheritance and is due to a mutation in the SMN1 gene. Spinal muscular atrophy type I is also known as Werdnig-Hoffmann disease, which is the most severe form of the disease and usually results in childhood death due to respiratory failure Spinal muscular atrophy type II and III are less severe and often result in a lifelong inability to ambulate.

Bell Palsy

Bell palsy is the most common etiology of peripheral facial nerve palsy. Although it is not always a lower motor neuron deficit, it is a perfect example to demonstrate LMN signs. It usually develops after herpes virus reactivation, but it can also result from Lyme disease, herpes zoster (Ramsay-Hunt syndrome), sarcoidosis, tumors of the parotid gland, and diabetes mellitus.

If any part of the corticobulbar tract from the motor cortex to the facial nerve nucleus is damaged, it will result in UMN deficits; this will result in contralateral facial paralysis involving the lower muscles of facial expression. Because there is bilateral UMN innervation to the muscles of the forehead, there is sparing of the forehead.

However, if the lesion occurs anywhere from the facial nucleus along CN VII, it will result in LMN deficits, affecting the ipsilateral side of the face and involve both the upper and lower muscles of facial expressions. This condition presents as incomplete eye closure (orbicularis oculi), dry eyes, corneal ulceration, hyperacusis, and taste sensation loss to the anterior tongue. Because the forehead is involved, the affected individual will be unable to wrinkle their forehead (lift their eyebrows).

Neurological Assessment

Date of Assessment-

Demographic Details

1. Name
2. Age
3. Sex
4. Address
5. Occupation
6. Inpatient number
7. Outpatient number

Chief Complain-

History of present illness-

History of past illness

Mode of injury

Medical history

Personal history

Pain assessment

On Observation- built

- posture (sitting, standing,walking)
- Swelling
- Muscle wasting

- Contracture nd deformity (neck,back,chest,nd limb).

Palpation-

- Temperature
- Tenderness
- Odema

Examination-

Note the vital signs -

cranial nerve abnormalities

-Higher functions (memory, intelligence,speech behaviour,level of consciousness, etc.)

- Breathing pattern

Sensory assessment

- Superficial sensation
- Deep tendon sensation
- Combine sensation

Motor assessment

- ROM (active and passive)
- muscle girth
- MMT

MMT

OXFORD SCALE	EXPLANATION
0	No contraction is present
1	There is Flickering contraction
2	Full Range of Motion with gravity counter balance *(Eliminated)
3	Full Range of Motion with Against gravity
4	Full Range of Motion with Against gravity + added Resistance
5	Muscle function normally.

Modified Ashworth Scale

0	• No increase in tone
1	• Slight increase in tone • Catch/release at end ROM
1+	• Slight increase in tone • Catch/release and resistance through rest ROM (1/2 ROM)
2	• More marked increase in tone through ROM, but affected part moved easily
3	• Considerable increase in tone, passive movement difficult
4	• Affected part in rigid flexion and extension

Tardieu scale

A

Tardieu scale	
Quality of muscle reaction is measured as:	
0	No resistance throughout the course of the passive movement
1	Slight resistance throughout the course of the passive movement
2	Clear catch at precise angle, interrupting the passive movement, followed by release
3	Unsustained clonus (less than 10 sec when maintaining the pressure) occurring at a precise angle, followed by release
4	Sustained clonus (more than 10 sec when maintaining the pressure) occurring at a precise angle

Angle of muscle action is measured relative to the position of minimal stretch of the muscle (corresponding to angle zero) for all joints except the hip where it is relative to the resting anatomical position.

Grading of reflexes

Deep tendon reflex

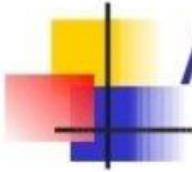
- 0- absent
- + present but diminished
- ++ normal
- +++ increased
- ++++ hyperactive or clonic

Superficial reflex

- 0 absent
- +present

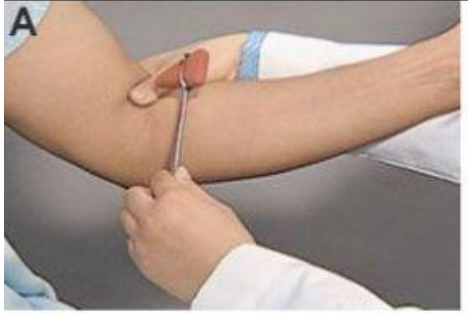
Superficial reflexes

- Corneal- trigeminal or facial nerve
- Palatal- IX-X cranial nerves
- Scapular- C5-T1
- Abdominal- T7-12
- Cremasteric- L1/2
- Plantar- L5/S1- extensor always abnormal except age <1 year & postictal
- Bulbocavernosus- S3/4
- Anal- S3/4



Assessing the reflexes

- Deep tendon reflexes
  - Biceps
  - Triceps
  - Brachioradialis
  - Patellar
  - Assessing the sensory function Achilles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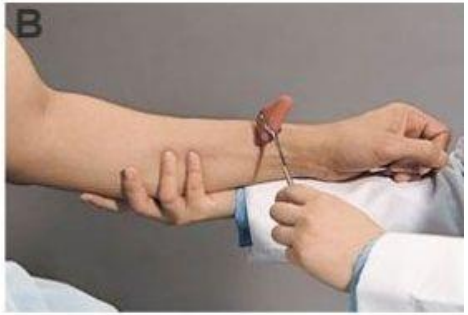
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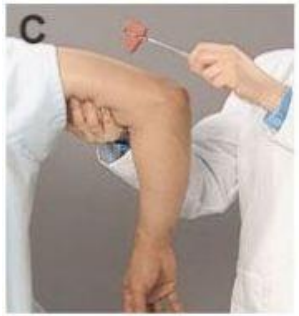
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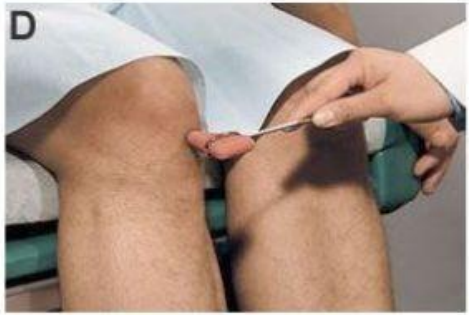
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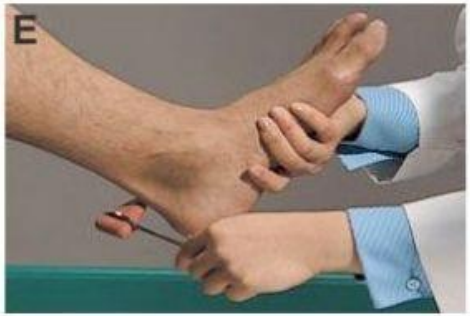
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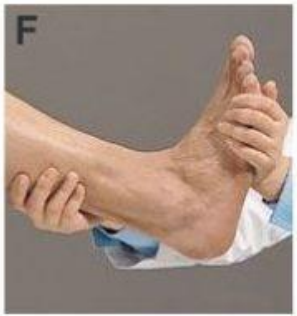
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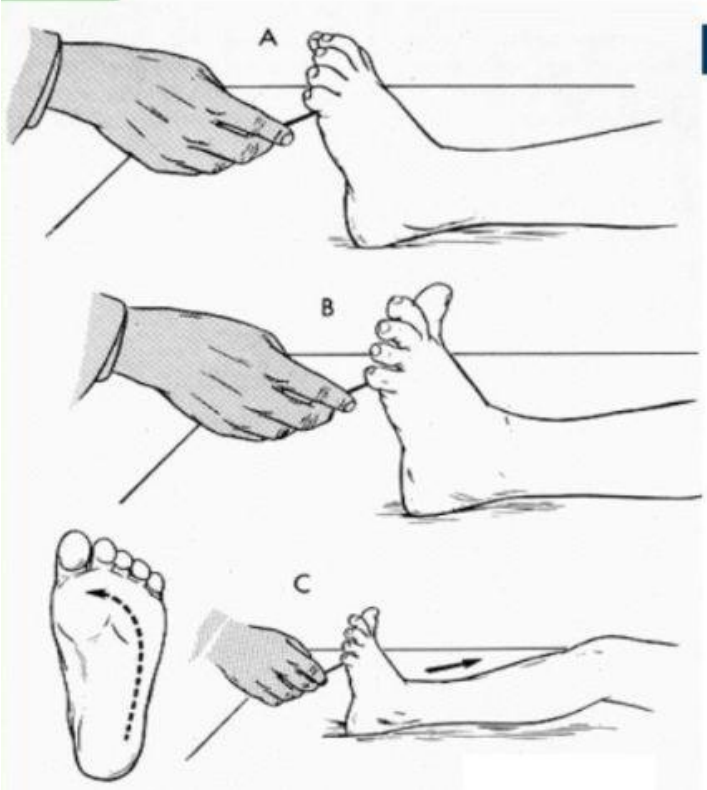
Hoffman's sign

"Babinski of the Upper Extremity"

- Test for UMN lesion
  - Flick middle finger
  - Watch for reflexive flexion/adduction of thumb

Planter Reflex and Babinski testing



Babinski's reflex – present when the great toe flexes toward the top of the foot and the other toes fan out after the sole of the foot has been firmly stroked.

- Postitive response indicates damage to nerve paths connecting the spinal cord and the brain (corticospinal tract)
- **Abnormal after the age of 2.**

Sensory Grading System (ASIA)	
0	Absent
1	Impaired
2	Normal
NT	Not Testable

Management

-The management of spasticity involves physical and medical modalities.

-Physical rehabilitation is a mainstay in treatment and involves proper positioning of patients, stretching of muscles, cooling of muscles, heat, and orthosis.

-Pharmacologic management should be an adjunct to these physical modalities.

-Correct positioning is an important area of management, especially for immobilized patients.

-The main goal is to produce a stretch of spastic muscles to reduce spasticity and facilitate the function of antagonistic muscles.

-Proper stretching maintains muscle length throughout passive and active exercises.

- Application of heat can increase the elasticity and thus relax spastic muscles.

-Finally, orthosis can be used to distribute or remove forces from the body to allow better control of movement or alter body shape.

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Thank You