



27th Lecture

Spasticity

PHYSIOLOGY TEAM – 430

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Spasticity and Increased Muscle Tone

- **What is Spasticity?**

- Spasticity is a motor disorder, characterised by increase in tonic static stretch reflexes (muscle tone) -with exaggerated tendon jerks, resulting from hyper-excitability of the dynamic stretch reflex as one component of the upper motor neurone (UMN) syndrome
- Clinically spasticity is defined as velocity dependent resistance to stretch, where a lack of inhibition from the CNS results in excessive contraction of the muscles
- It can also be defined as increased resistance to passive stretch
- Patients complain of stiffness & inability to relax
- Muscles become permanently "tight" or spastic
- The condition can interfere with walking, movement, or speech

- **What causes Spasticity?**

- It is caused by diseases that disrupt the normal physiological inhibition mechanisms of the CNS, which leads to increased excitability of the stretch reflex → hypertonia & continuous, unremitting contractions of the muscle involved
- This continuous contraction results in hypertonia, tightness and stiffness of the muscles concerned and, depending on which muscle groups are involved, posture, voluntary movement and speech can be involved

- **Some conditions associated with spasticity:**

- 1) UMNS (Upper Motor Neuron Syndrome)
- 2) Parkinsonism
- 3) Decerebrate & Decorticate rigidity

1) UMNS:

- Features:

1. Weakness and decreased muscle control
2. No remarkable muscle wasting, but disuse atrophy
3. Spasticity (hypertonia), frequently called “clasp-knife spasticity”= increased extensor muscle tone then a sudden collapse in resistance due to inhibition of extensor motor neurons by GTOs (golgi tendon organs)
4. Clonus Repetitive jerky motions (clonus), especially when limb moved & stretched suddenly

Note:

- Clonus is a series of involuntary muscular contractions and relaxations
- Clonus is a sign of certain neurological conditions, and is particularly associated with upper motor neuron lesions such as in stroke, multiple sclerosis, spinal cord damage and hepatic encephalopathy. clonus causes large motions that are usually initiated by a reflex

5. Exaggerated tendon jerks
6. Extensor plantar reflex = Babinski sign (dorsiflexion of the big toe and fanning out of the other toes)
7. Absent abdominal reflexes

- In UMN syndrome, the motoneurons are free from the descending inhibitory influence of the Higher Motor-Controlling centers (medullary reticular formation ,red nucleus ,basal ganglia) resulting in unantagonized excitatory input (pontile reticular formation , vestibular nucleus) to gamma motoneurons causing hypertonia & spasticity, This results in:

- State of ongoing (unremitting)contraction of muscles (due to hyperactive gamma activity)
- Decreased ability to control movement
- Increased resistance felt on passive stretch

• UMNS include:

1. Cerebral palsy
2. Stroke
3. Spinal cord injury
4. Multiple Sclerosis
5. Acquired brain injury (trauma ,etc)

1. Cerebral Palsy:

- Cerebral palsy (CP) is an umbrella term encompassing a group of non-progressive, non-contagious motor conditions that cause physical disability in human development, chiefly in the various areas of body movement.
- Caused by brain damage due to lack of oxygen, as (near drowning or near suffocation) that cause damage to the motor control centers of the developing brain
- It can occur during pregnancy, during childbirth(Or after birth up to about age three by meningitis)

Contagious: capable of being transmitted from one individual to another
Cerebral Palsy is a non-contagious disease

2. Stroke:

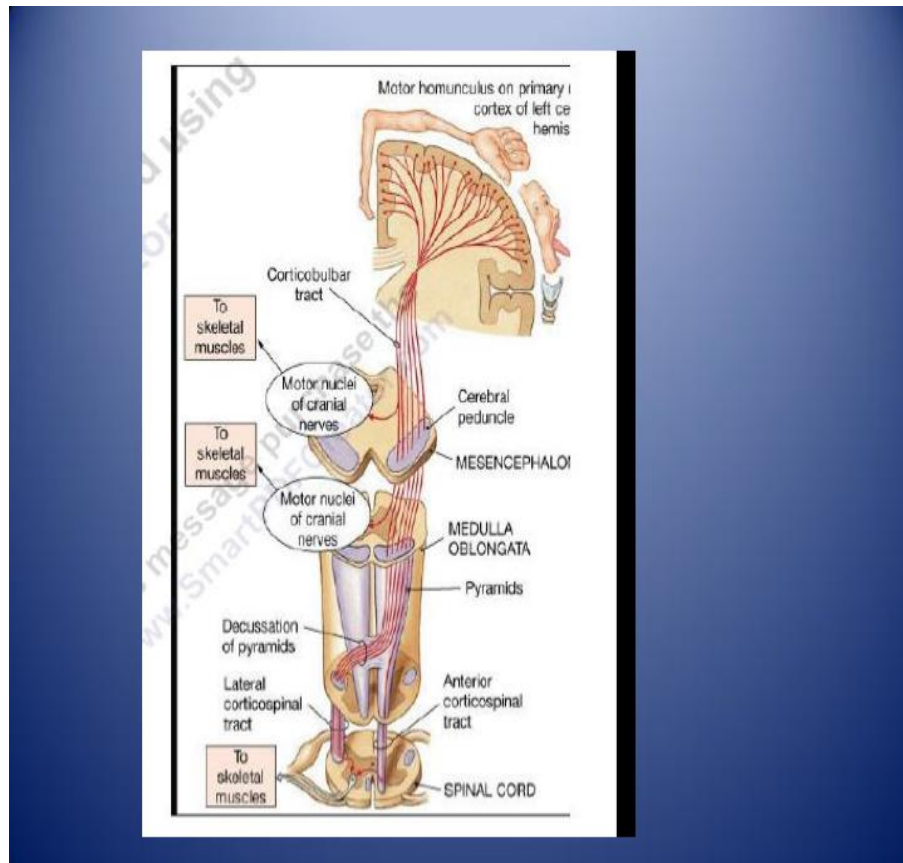
- **Causes:**
- ✓ Haemorrhagic stroke as in cerebral hemorrhage
- ✓ Ischaemic stroke as in thrombosis or embolism
- All cause death of brain tissues
- Results in paralysis in the opposite half of the body
- ☒ A lesion in Corona Radiata on one side can cause Monoplegia in a contralateral limb (upper limb or lower limb, according to site of injury)

Monoplegia: Paralysis of a single part

- ☒ A lesion in the Internal Capsule on one side may cause Hemiplegia or Hemiparesis on the contralateral side

Hemiplegia: paralysis of one side of the body; usually caused by a brain lesion, such as tumor. Or by stroke syndrome. The paralysis occurs on the side opposite the brain disorder. Because motor axons from the cerebral cortex travel in two bands called the pyramidal tracts, and most of the fibers cross the opposite side (decussate) in the medulla oblongata.

Hemiparesis: muscular weakness or partial paralysis affecting one side of the body



3. Spinal Cord Injury:

- Complete transection of spinal cord:

Transection: a cross section; division by cutting transversely
E.g. following tumor, trauma

- ✓ The higher the level of the section, the more serious are the consequences
- ✓ If the transection is in the upper cervical region immediate death follows, due to paralysis of all respiratory muscles.
- ✓ In the lower cervical region below the 5th cervical segment diaphragmatic respiration is still possible, but the patient suffers complete paralysis of all four limbs (quadriplegia), could be due to bullet injury or fractures of the spine
- ✓ Transection lower down in the thoracic region allows normal respiration but the patient ends up with paralysis of both lower limbs (paraplegia)

- **Stages of Paraplegia:**

- 1- Spinal shock (2-6 weeks)
- 2- Stage of return of reflex activity
- 3- Paraplegia in extension

1- Spinal shock:

- **In the immediate period following transection there is:**

- 1) Complete loss of spinal reflex activity below the level of the lesion
- 2) Loss of all sensations (anesthesia) and voluntary movement (paralysis) below the level of the lesion, due to interruption of all sensory and motor tracts
- 3) Loss of tendon reflexes and superficial reflexes (abdominal, plantar & withdrawal reflexes)
- 4) The loss of muscle tone (flaccidity) and absence of any muscle activity (muscle pump) lead to decreased venous return causing the lower limbs to become cold and blue in cold weather (they become blue and cold because of poor blood circulation)
- 5) The wall of the urinary bladder becomes paralyzed and urine is retained until the pressure in the bladder overcomes the resistance offered by the tone of the sphincters and dribbling occurs. This is known as retention with overflow
- 6) Loss of vasomotor tone occurs, due to interruption of fiber that connects the vasomotor centers in the medulla oblongata with the lateral horn cells of the spinal cord, which project sympathetic vasoconstrictor impulses to blood vessels. Vasodilatation causes a fall in blood pressure; the higher the level of the section, the lower the blood pressure

Vasomotor centers: nerve centers in the medulla oblongata and lower pons that regulate the caliber of the blood vessels and increase or decrease the heart rate and contractility

- This stage varies in duration but usually lasts a maximum of 2-6 weeks, after which some reflex activity recovers

2- Stage of return of reflex activity:

- **As the spinal shock ends, spinal reflex activity appears again, this partial recovery may be due to:**

Increase in degree of excitability of the spinal cord neurons below the level of the section, due to disinhibition of motoneurons as a result of absence of inhibitory impulses from higher motor controlling centers + sprouting of fibers from remaining other + denervation supersensitivity to excitatory neurotransmitters

- **Features of the stage of recovery of reflex activity:**

1) **Gradual rise of arterial blood pressure:**

Due to return of spinal vasomotor activity in the lateral horn cells. But, since vasomotor control from the medulla is absent, the blood pressure is not stable

2) **Return of spinal reflexes:**

- ✓ Flexor reflexes return earlier than extensor ones → Paraplegia in Flexion.
- ✓ Extensor plantar reflex or complete Babiniski sign

3) **Recovery of visceral reflexes:**

Return of micturition, defecation & erection reflexes

However, voluntary control over micturition and defecation, and the sensation of bladder and rectal fullness are permanently lost (Automatic Micturition)

4) **Mass reflex appears in this stage:**

A minor painful stimulus to the skin of the lower limbs will not only cause withdrawal of that limb but will evoke many other reflexes through spread of excitation (by irradiation) to many autonomic centers.

Therefore, the bladder and rectum will also empty, the skin will sweat, and the blood pressure will rise

- Since effective regeneration never occurs in the human central nervous system, patients with complete transection never recover fully
- Voluntary movements and sensations are permanently lost; however, patients who are rehabilitated and properly managed may enter into a more advanced stage of recovery

3- Stage of extensor paraplegia:

- 1) During this stage, the tone in extensor muscles returns gradually to exceed that in the flexors. The lower limbs become spastically extended. Extensor reflexes become exaggerated, as shown by tendon jerks and by the appearance of clonus
 - The positive supportive reaction becomes well developed and the patient can stand on his feet with appropriate support
- 2) The flexor withdrawal reflex which appeared in the earlier stage is associated during this stage with the crossed extensor reflex.

• Hemisection of the Spinal Cord:

- Occurs as a result of unilateral lesion or hemisection of the spinal cord (e.g. due to stab injury, bullet, car accident, or tumor)
Let us take an example of such injury involving the thoracic spinal cord: The manifestations of the Brown-Sequard syndrome depend on the level of the lesion

Brown-Sequard syndrome: paralysis and loss of discriminatory and joint sensation on one side of the body and of pain and temperature on the other, due to a lesion involving one side of the spinal cord

- The manifestations:

✓ At the level of the lesion, all manifestations occur on the same side:

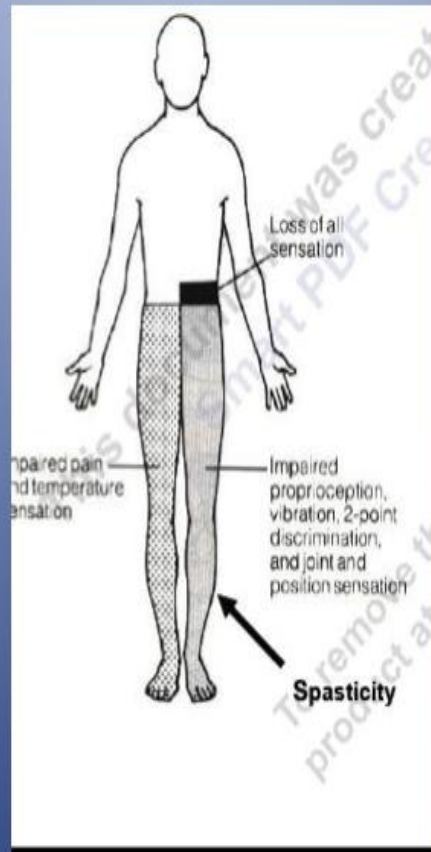
1. Paralysis of the lower motor neuron type
2. Loss of all sensations in the areas supplied by the afferent fibers that enter the spinal cord in the damaged segments

✓ Ipsilaterally below the level of the lesion:

1. Spastic lower limb .
2. Fine touch, position and vibration sense are lost
3. Vasodilatation

✓ Contralaterally below the level of the lesion:

Pain and temperature sensations are lost, because ascending fibers carrying pain and temperature sensation (spinothalamic tract) decussate in the spinal cord, while dorsal column tracts decussate in the medulla so there affect appear on the ipsilateral side because they are not decussated in the spinal cord



4. Multiple Sclerosis:

- Is an auto-immune demyelinating disease, in which the body's own immune system attacks and damages the myelin sheath of myelinated nerves mainly of brain, SC, and optic nerve
- Loss of myelin sheath (demyelination) prevents axons from salutatory conduction of action potentials causing muscle weakness & wasting
- Disease onset usually occurs in young adults, and it is more common in females
- The disease can attack any part of the CNS, and when it causes demyelination of descending motor tracts in the brainstem & spinal cord, the subject develops spasticity and other signs of UMNS
- The disease frequently remits and relapses because of re-myelination & restore of function and during acute attacks, intravenous corticosteroids can improve symptoms.

2) Parkinsonism:

- Cog-wheel rigidity
- Lead-pipe rigidity

3) Decerebrate & Decorticate rigidity:

Decerebrate rigidity: the patient lies in rigid extension with the arms internally rotated at the shoulders, elbows, knees, and hips extended, and fingers, ankles, and toes flexed. The jaw may be clenched with the neck hyperextended.

Decorticate rigidity: a unilateral or bilateral postural change, consisting of the upper extremities flexed and adducted and the lower extremities in rigid extension.

In Conclusion

Spasticity (hypertonia) is a feature of altered muscle performance, occurring in disorders of the central nervous system, which give rise to the Upper Motor Neuron Syndrome (UMNS)

When there is a loss of descending inhibition from the brain to Brain Stem Excitatory centers (vestibulospinal & reticulospinal), excitatory signals cause muscles to become overactive and spastic

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