physiology team 429

Spasticity and Increased Muscle Tone

Done By :

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- **Spasticity (hypertonia) is a feature of altered muscle performance, occurring in disorders of the central nervous system , It can be defined as increased resistance to passive stretch.**

- **definition of spasticity(1980) : "it is a motor disorder, characterised by increase in tonic 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"**

- **Spasticity2004: The misunderstood part of the upper motor neuron syndrome.**

- Patients complain of **stiffness** & **inability to relax**
(Muscles become permanently "tight" or *spastic*)

When does it occur???

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 & spastic** .

- The condition can interfere with walking, movement, or speech.

Causes of spasticity:-

A-(UMNS) syndrome include :

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

B-Parkinsonism

C-Decerebrate&decorticate rigidity

Features of the Upper Motor Neuron Syndrome (UMN):

(1) Weakness and decreased muscle control .

(2) No remarkable muscle wasting, but disuse atrophy
(atrophy due to lack of movement but not as obvious as in lower motor neurone lesion) .

(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) ←← over stretch .

- **(4) Clonus Repetitive jerky motions , especially when limb moved & stretched suddenly**

  (clonus: involuntary muscular contraction and relaxation in rapid succession)

- **(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**
  (abdomenal reflex: contraction of the muscles of the abdominal wall upon stimulation of the skin or upon tapping of neighboring bony structures)

In UMN syndrome the motorneurones are free from the descending inhibitory influence of the Higher Motor-Controlling centers (medullary RF,red nucleus,basal ganglia) resulting in unantagonized excitatory input (ponile RF,vestibular N) to gamma motoneurones causing hypertonia & Spasticity .

- This results in

(1) State of ongoing (unremitting)contraction of muscles .(due to hyperactive gamma activity)

(2) decreased ability to control movement

(3) increased resistance felt on passive stretch.

Causes of spasticity ⇨ 1 \ (UMNS) syndrome include :

(1) Cerebral palsy :-

- 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)**

Causes of spasticity ⇨ 1 \ (UMNS) syndrome include :

(2) 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 .

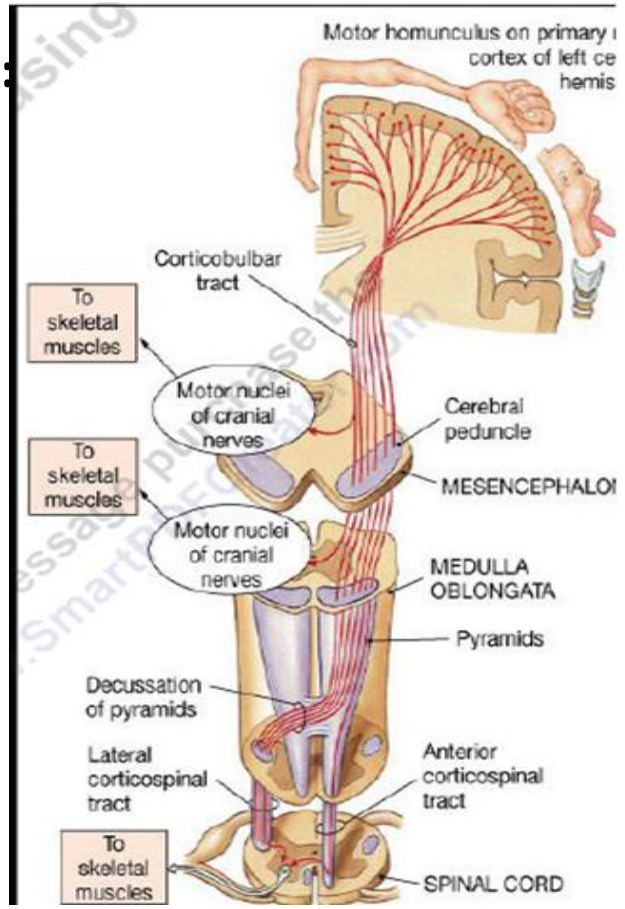
• Loss of myelin sheath (demyelination) prevents axons from saltatory conduction of action potentials causing muscle weakness& wasting.

• 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 descendindg motor tracts in the brainstem & spinal cord , the subject develops spasticity and other signs of UMNS .

• The disease frequently remits and relapses because of remylination & restore of function and during acute attacks intravenous corticosteroids can improve symptoms

Causes of spasticity ⇨ 1 \ (UMNS) syndrome include :



(3) STROKE :-

Causes :

A- Haemorrhagic stroke as in cerebral hemorrhage

B- Ischaemic stroke as in thrombosis or embolism

- All cause death of brain tissues because the brain tissue is deprived from oxygen and nutrients in both types , Results in paralysis in the oppsite half of the body .

- **A lesion in Corona Radiata on one side can cause Monoplegia (Complete paralysis of a single limb, muscle) in a contralateral limb (UL or LL ,according to site).**
- **A lesion in the Internal Capsule on one side may cause Hemiplegia or Hemiparesis on the contralateral side .**
- **(with the picture of upper motor neuron syndrome).**

Causes of spasticity ⇨ 1 \ (UMNS) syndrome include :

(4) Spinal injury Can be :

(I) Complete transecion of spinal cord

(II) Hemisection of the spinal cord

1-Complete transection of spinal cord:-

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 5^{th} cervical segment diaphragmatic respiration is still possible, but the patient suffers complete paralysis of all four limbs (quadriplegia).

• Transection lower down in the thoracic region allows normal respiration but the patient ends up with paralysis of both lower limbs (paraplegia)

Stages :-

A/ Spinal shock (2-6 weeks)
B/ Recovery of reflex activity
C/ Paraplegia in extension

A/ 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

(5) The wall of the urinary bladder becomes paralysed 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 connect the vasomotor centres 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.
-This stage varies in duration but usually lasts a maximum of 2-6 weeks, after which some reflex activity recovers.

B/ 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 the natural 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 fibrers 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.
- Babiniski sign (extensor plantar reflex) is one of the earliest signs of this stage +/- flexion reflex .
- Tendon reflexes also recover earlier in flexors.
- As a result, flexor spastic tone causes the lower limbs to take a position of slight flexion, a state referred to as paraplegia in flexion.
- The return of the stretch reflex & cosequently muscle tone , and vasoconstrictor tone in arterioles and venules improve the circulation through the limbs.

(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.

(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.**

The bladder and rectum will also empty, the skin will sweat, 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 .

C/ 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, 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.

2- Hemisection of the Spinal Cord(Brown-Sequard syndrome)

- **Occurs as a result of unilateral lesion or hemisection of the spinal cord (e.g. due to stab injury, bullet , car accident, or tumor).**

an example of such injury involving the thoracic spinal cord is the Brown-Sequard syndrome

The manifestations of the Brown-Sequard syndrome depend on the level of the lesion.

A \ At the level of the lesion (all the manifistations occur on the same side)

1.Paralysis of the lower motor neuron type, involving only the muscle supplied by the damaged segments.

2. Loss of all sensations in the areas supplied by the afferent fibres that enter the spinal cord in the damaged segments +/- band of hyperesthesia .

B/ Ipsilaterally (below the level of the lesion)

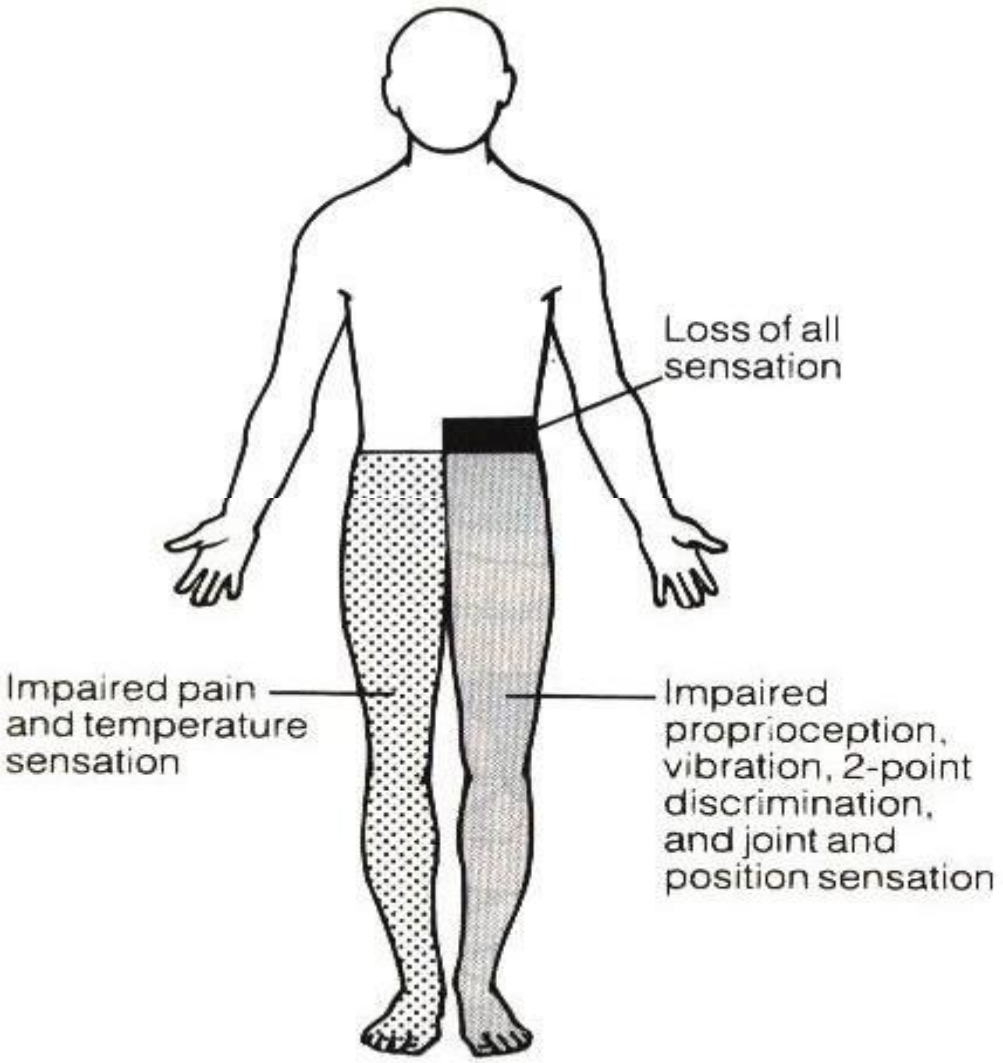
1. spastic lower limb, increased reflexes and clonus
2.Fine touch, position and vibration sense are lost WHY?

Because the Dorsal column tract decussate in the Medulla Oblongata

C/ Contralaterally (below the level of the lesion)

Pain and temperature sensations are lost, Why ?

Because the Lateral Spinothalamic Tract decussate at the same level (segment) of the spinal cord they enter


Loss of all sensation
Impaired pain and temperature sensation
Impaired proprioception, vibration, 2-point discrimination, and joint and position sensation

B- Parkinsonism:-

- Cog-wheel rigidity

a type of rigidity seen in parkinsonism in which the muscles respond with cogwheellike jerks to the use of constant force in bending the limb

- Lead-pipe rigidity

a state of stiffness and inflexibility that remains uniform throughout the range of passive movement, associated with diseases of the basal ganglia.