

# **Spasticity and Increased Muscle Tone**

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# OBJECTIVES

- At the end of this lecture you should be able to:
- Define spasticity and rigidity .
- Describe the neurophysiology of spasticity
- Describe the causes of spasticity

# MUSCLE TONE

- Resistance of a muscle to stretch is often referred to as its tone or tonus.
- Muscle tone is static component of stretch reflex .It is a continuous mild muscle contraction that acts as background to actual movement.
- A hypertonic muscle is one in which the resistance to stretch is high because of hyperactive stretch reflexes

- Hypertonia is of two types:

1. Spasticity

2. Rigidity

## **Spasticity:**

As described by Lance (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

■ Clinically it can be defined as increased resistance to passive stretch.

-Spasticity is velocity dependent increased resistance to passive movement of the muscle due to abnormally high muscle tone (hypertonia) which varies with the speed of displacement of a joint.



- The faster you stretch the muscle the greater the resistance.

-- Spasticity is clearly neural in nature and is associated with the - UMNL

-Involvement of the corticospinal tract is often associated with UMNL and spasticity. There are a number of clinical features that are spasticity associated reactions:-

-hyperreflexia, flexor spasticity in the upper limb & extensor spasticity in the lower limb.

I-UMN lesions Spasticity is of Clasp Knife Type

# Spasticity

- - Spasticity is characterized by hyper-excitability of both types of stretch reflex:-
- 1- increase in tonic static stretch reflexes (muscle tone) as one component of the upper motor neuron (UMN) syndrome
- 2- Exaggerated tendon jerks, resulting from hyper-excitability of the dynamic stretch reflex as one component of the upper motor neuron (UMN) syndrome

# Rigidity

- Rigidity is increased neural activity throughout the range of muscle movement and is not velocity dependent.
- Rigidity is present in both agonist and antagonist muscles. It is often associated with basal ganglia disease such as Parkinson's disease

## Rigidity in Parkinsonism:

- 1. Lead-pipe rigidity :  
Passive movement of an extremity meets with a constant dead feeling resistance like a lead pipe throughout the range of movement.



# Rigidity-2

## 2. Cog-wheel rigidity :

- In cogwheel rigidity one feels that resistance varies rhythmically when applying a passive movement.
- It is because of an underlying resting tremor associated with rigidity.



# Other types of Rigidity

- - **Decerebrate rigidity :**  
(extension of head & 4 limbs extensors )
- - **Decorticate rigidity:**
- (extensor rigidity in legs & moderate flexion of arms if head unturned )

Decerebrate posture results from damage to the upper brain stem. In this posture, the arms are adducted and extended, with the wrists pronated and the fingers flexed. The legs are stiffly extended, with plantar flexion of the feet.

## DECEREBRATE



Decorticate posture results from damage to one or both corticospinal tracts. In this posture, the arms are adducted and flexed, with the wrists and fingers flexed on the chest. The legs are stiffly extended and internally rotated, with plantar flexion of the feet.

## DECORTICATE



## Features of UMN Syndrome

- (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
- (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

## ■ Mechanism of spasticity in UMN lesions:

In UMN syndrome the motor neurones are free from the descending inhibitory influence of the Higher Motor-Controlling centers ( medullary RF, red nucleus , basal ganglia)resulting in un antagonized excitatory input ( pontine RF, vestibulo-spinal) to gamma motor neurones 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:-

**A-(UMNS) syndrome include :**

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

**B-Parkinsonism**

**C-Decerebrate & decorticate rigidity**

# Spastic CP



Spastic Gait

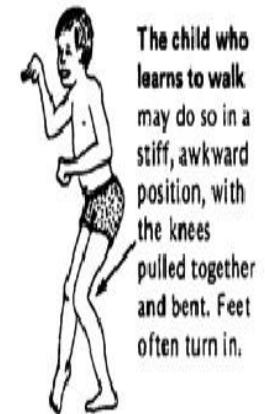
## (1) Cerebral palsy

CP includes disorders apparent at birth or in childhood due to intrauterine or neonatal brain damage

### Causes:

- hypoxia in utero and/or during parturition
- neonatal cerebral haemorrhage and/or infarction
- trauma, neonatal or during parturition
- prolonged seizures - status epilepticus
- Hypoglycemia
- kernicterus

- Increased muscle tone, tense and contracted muscles
  - Have stiff and jerky or awkward movements.
  - limbs are usually underdeveloped
  - increased deep tendon reflexes
- most common form
- 70-80% of all affected



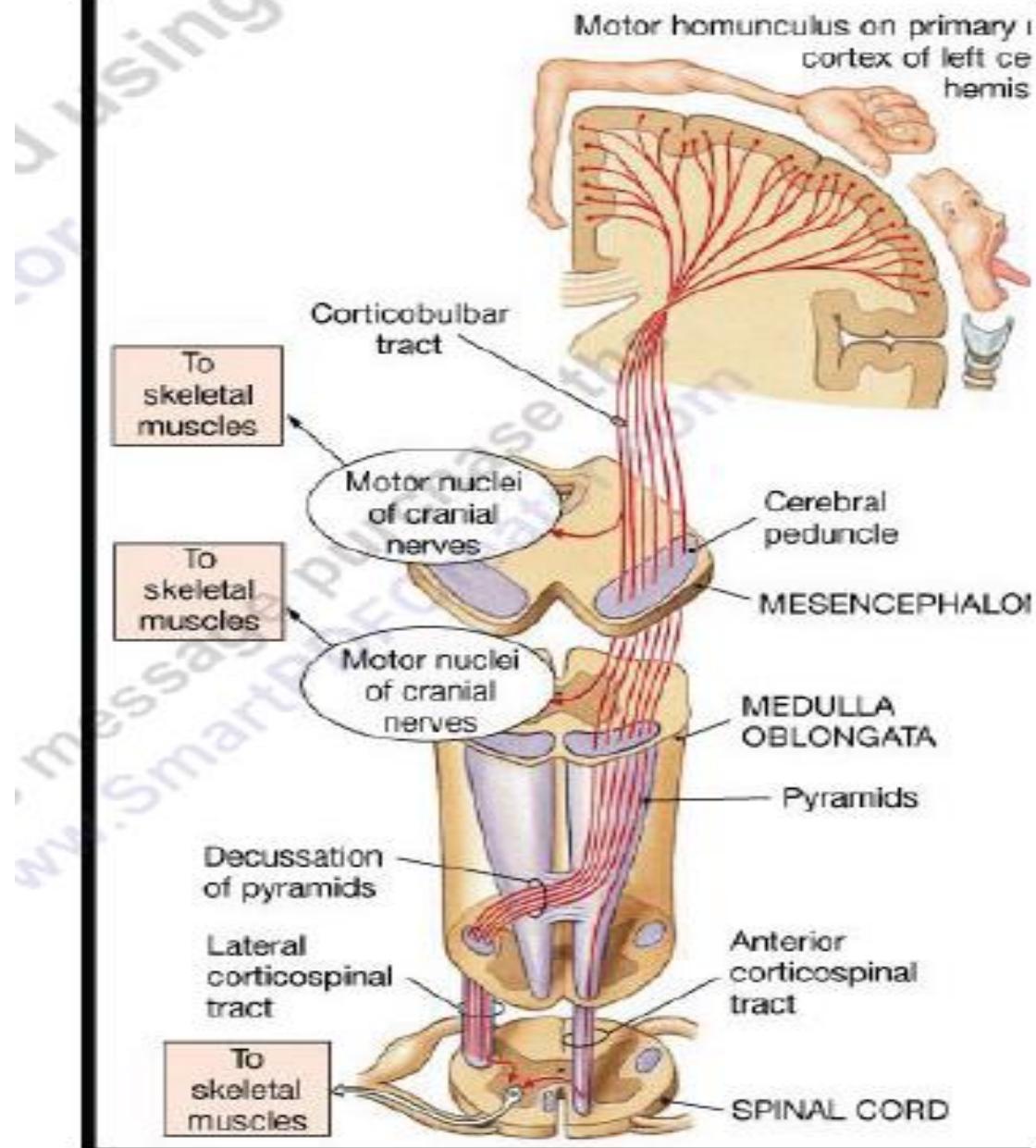
The child who learns to walk may do so in a stiff, awkward position, with the knees pulled together and bent. Feet often turn in.

## (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 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 remyelination & restore of function and during acute attacks intravenous corticosteroids can improve symptoms

## 3-STROKE:-

- a-Haemorrhagic stroke as in cerebral haemorrhage  
b- 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 (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 ).



## 4. Complete transection of spinal cord- 1

- 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<sup>th</sup> 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**)

# Complete transection of spinal cord-2

- **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 (**anaesthesia**) 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 and body temperature to decrease.

## Complete transection of spinal cord-3

(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**.

(7) Loss of vasomotor tone occurs, due to interruption of fibers 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.

(8) Bedsores where pressure of body-weight against underlining support exerts enough pressure to hinder the circulation.

- **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 , to make up for the loss of supraspinal facilitatory influences. It may also be due to sprouting of fibers from remaining other inputs.

### 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.
- ✓ Babinski sign ( extensor plantar reflex) is one of the earliest signs of this stage.
  - 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 (& consequently **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) Sexual reflexes, consisting of erection or ejaculation on genital manipulation , recover.

## ■ (5) **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. So 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, 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 ( Brown-Sequard syndrome)**

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

## A/ 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
3. Vasodilatation of the blood vessels that receive vasoconstrictor fibers from the damaged segment

## B/ Ipsilaterally below the level of the lesion :

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

## C/ Contralaterally below the level of the lesion :

- Crude touch , pain and temperature sensations are lost, Why ?

