Spasticity and Increased Muscle Tone

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At the end of this lecture you should be able to

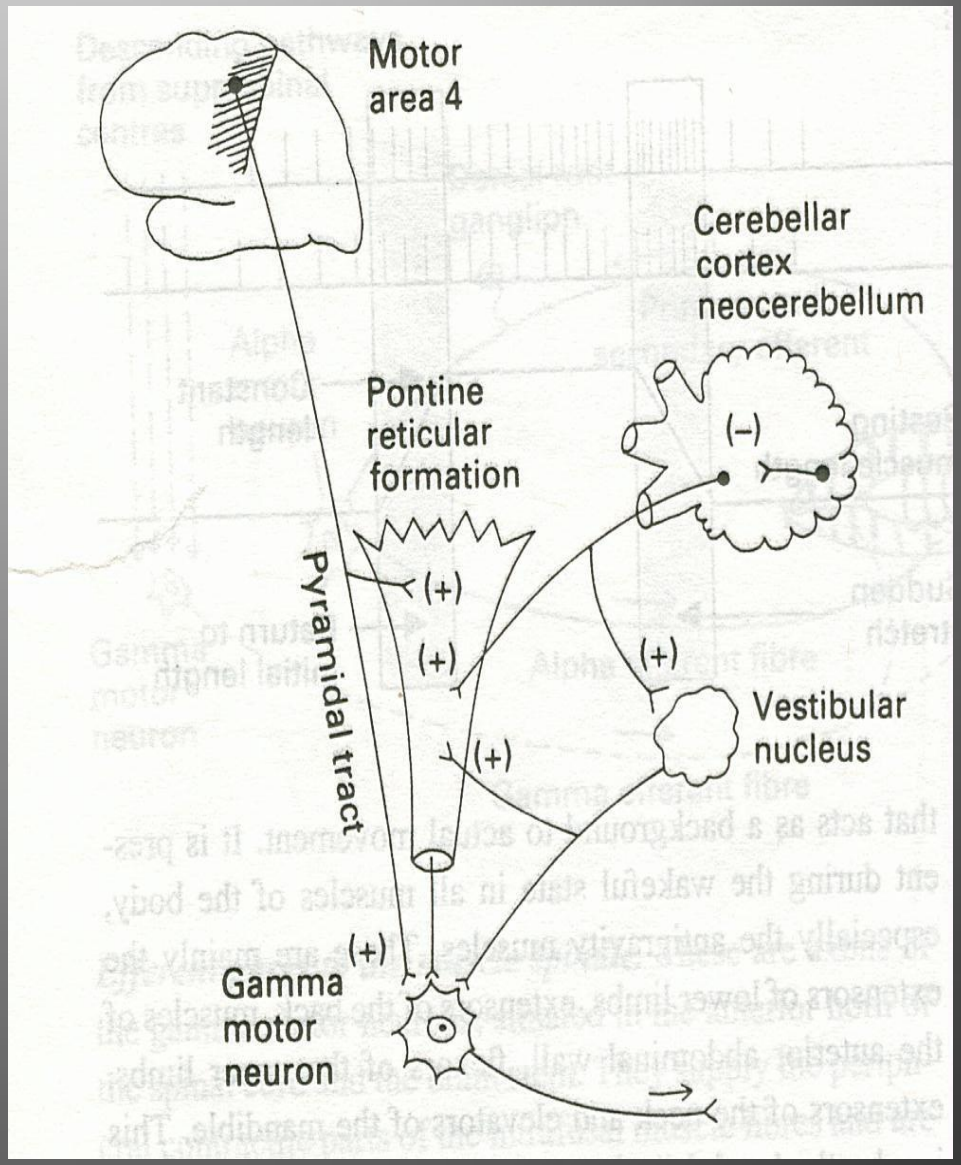
-Define spasticity,regididty& hypertonia

-Know main causes of spasticity&rigidity

Increased Gamma efferent discharge is the main cause of increased muscle tone.

how?

Facilitatory supra spinal centers to gamma motor neurons



Hypertonicity refers to increased resistance to passive lengthening (passive stretch)of a muscle or muscle group. This may mean increased viscosity or stiffness of the muscle. Hypertonicity could be due to a neural drive problem such as spasticity or rigidity

Spasticity is velocity dependent increased resistance to passive lengthening of the muscle. The faster you stretch the muscle the greater the resistance. Spasticity is clearly neural in nature and is a associated with the upper motor syndrome.
Involvement of the corticospinal tract is often associated with the upper motor unit syndrome and spasticity. There are a number of clinical features that are also associated with spasticity that are part of the upper motor syndrome and these features part of spasticity: associated reactions, hypereflexia, flexor synergy in the upper limb and extensor synergy in the lower limb.

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

-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).

- Spasticity can be defined as increased resistance to passive stretch.
-Patients complain of stiffness & inability to relax

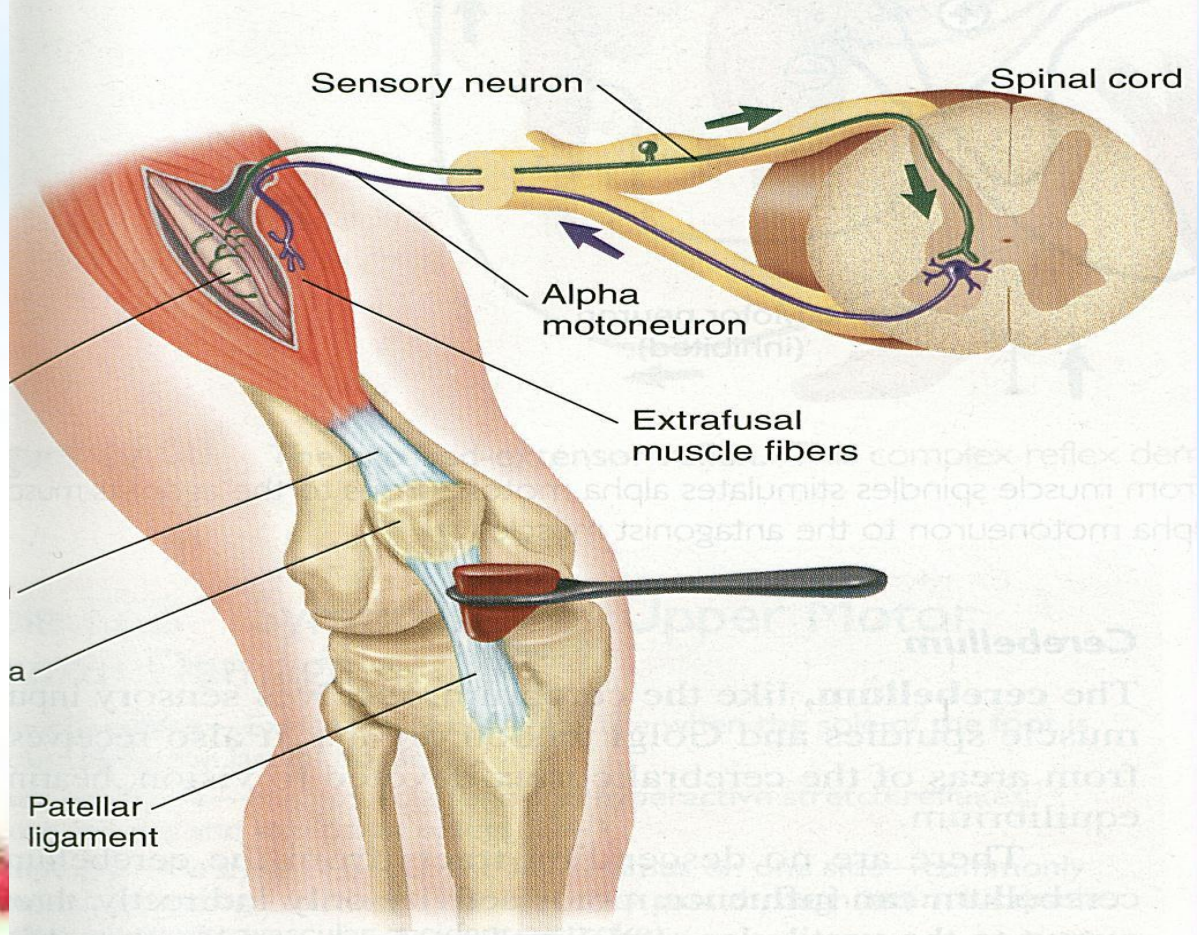
-Muscles become permanently "tight" or *spastic*.
- The condition can interfere with walking, movement, or speech.

- When there is a loss of descending inhibition from the brain to brain stem excitatory centers (pontine RF + vestibular N)
-Vestibulospinal & reticulospinal excitatory signals cause muscles to become overactive & spastic .

- Spasticity is a motor disorder, characterised 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 neurone (UMN) syndrome

2- Exaggerated tendon jerks, resulting from hyper-excitability of the dynamic stretch reflex as one component of the upper motor neurone (UMN) syndrome



ure 12.27 **The knee-jerk reflex.** This is an example of a
ynaptic stretch reflex.

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 resistance at the begining of muscle stretch due to 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

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

- 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) Acqiured brain injury (trauma , etc)**

B- Causes of rigidity

-Parkinsonism

- Decerebrate & decorticate rigidity

- **(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 centres of the developing brain**

- it can occur during pregnancy , during stressed childbirth (or after birth up to about age three by meningitis)

(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
- during acute attacks intravenous corticosteroids can improve symptoms

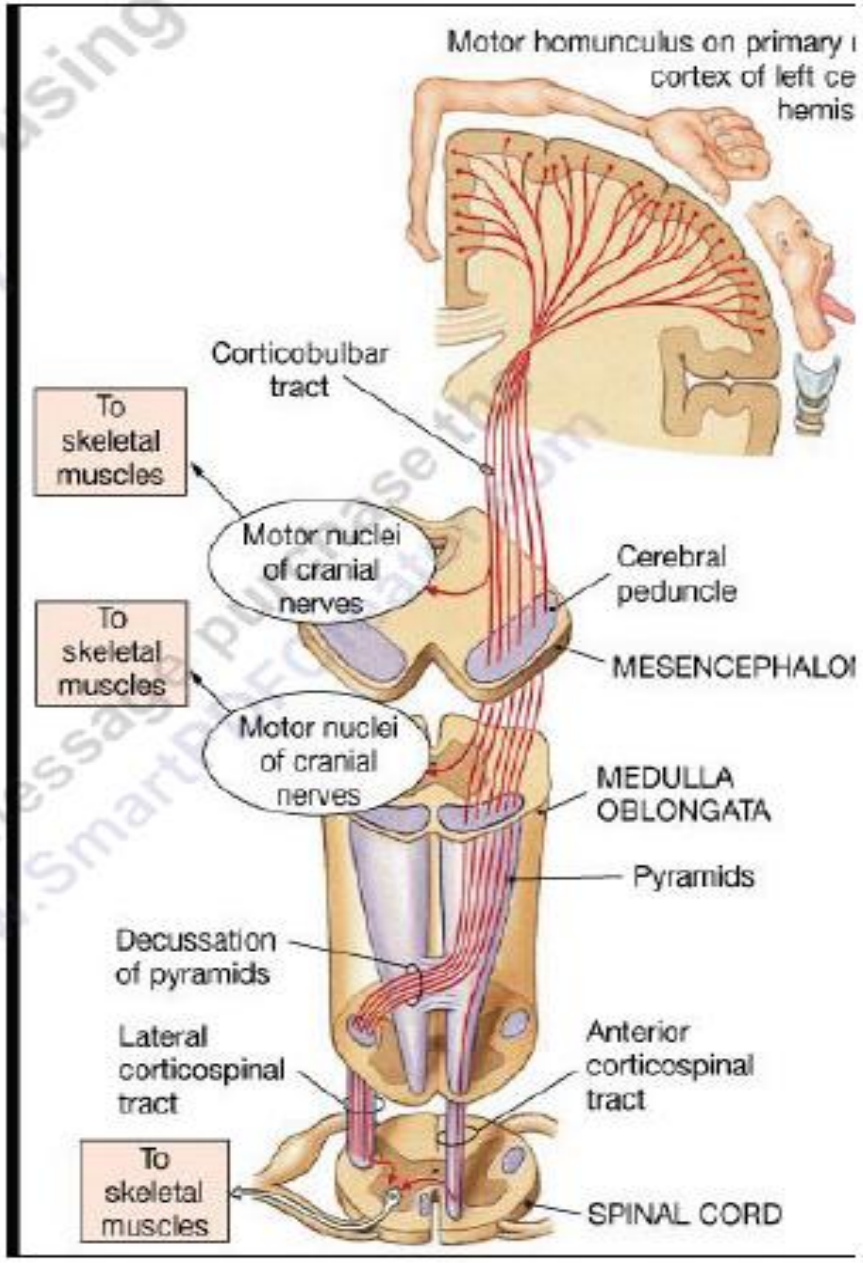
3-STROKE:-Causes :

- a-Haemorrhagic stroke as in cerebral hemorrhage
  b- Ischaemic stroke as in thrombosis or embolism in brain bl.v

-Both 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 UMNL .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Pyramids
Decussation
of pyramids
Lateral
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corticospinal
tract
To
skeletal
muscles
SPINAL CORD

4-Complete transection of spinal cord:-

e.g. following tumor or trauma

• The higher the level of the section, the more serious are the consequences.

•1- If the transection is in the upper cervical region immediate death follows, due to paralysis of all respiratory muscles;

•2- 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).

• 3-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) 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) =complete loss of spinal reflex activity below the level of the lesion .
(5) 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

(6) 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 fibres 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;

-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 degree of excitability of the spinal cord neurons below the level of the section ,

due to :_

1-disinhibition of motoneurons as a result of absence of inhibitory impulses from higher motor centres

-sprouting of fibres from remaining neurons

-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

- vasoconstrictor tone in arterioles and venules improve the circulation through the limbs.

2) Return of spinal reflexes:

- Flexor tendon reflexes return earlier than extensor ones.
- Babiniski sign (extensor plantar reflex) is one of the earliest signs of this stage +/- flexion reflex .

- **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 (**muscle tone**) ,
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(2) 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)

(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 centres. So the bladder and rectum will also empty, the skin will sweat, the blood pressure will rise**

-Voluntary movements and sensations are permanently lost;

-however , patients who are rehabilitated and properly managed may enter into a more advanced stage of recovery.

(Since effective regeneration never occurs in the human central nervous system, patients with complete transection never recover fully.)

• 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)

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

: The manifestations of the Brown-Sequard syndrome depend on the level of the lesion.(Let us take an example of such injury involving the thoracic spinal cord)

On the same side at the level of lesion

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

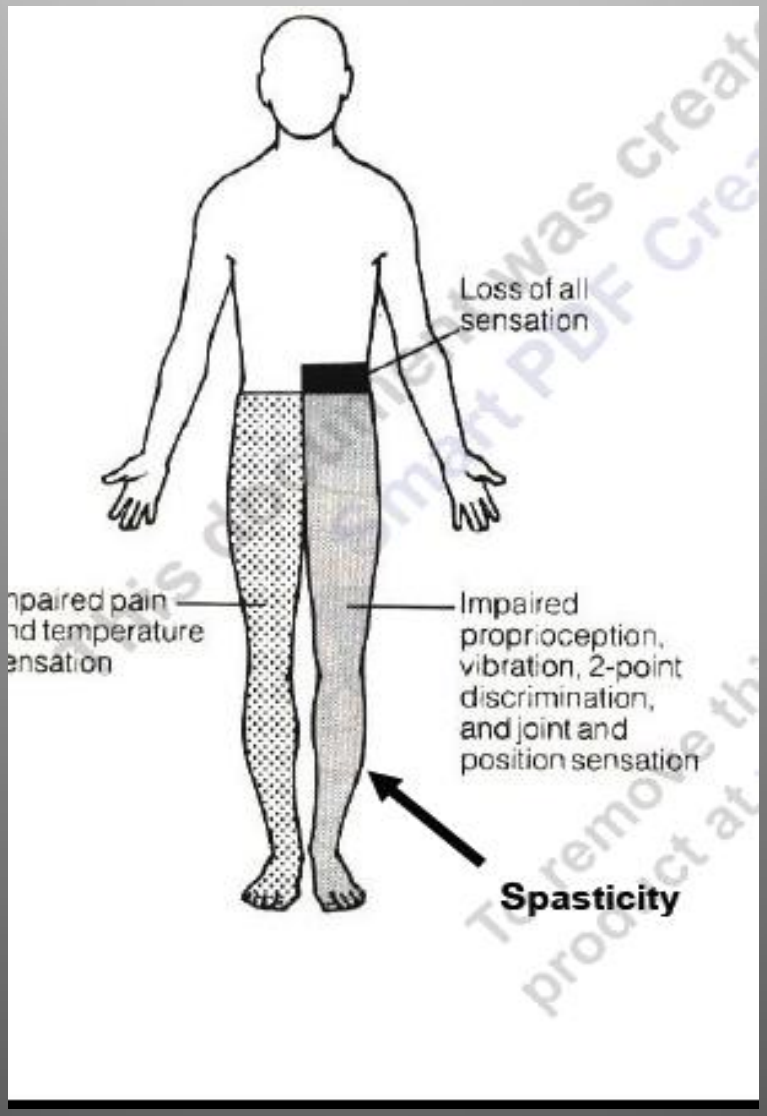
3. Loss of all sensations in the areas supplied by the afferent fibres that enter the spinal cord in the damaged segments +/- band of hyperesthesia

B/ Ipsilaterally below the level of the lesion :

1. UMNL/spastic lower limb (spasticity) &CLONUS
2. Fine touch, two-point discrimination, position and vibration sense are lost. why?

C/ Contralaterally below the level of the lesion :

Pain and temperature sensations are lost, Why ?


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

B-CAUSES OF RIGIDITY

-1-Parkinsonism rigidity is of two types:-

a-Cog-wheel rigidity

In cogwheel rigidity one feels the resistance rhythmically vary when applying a passive movement. It is thought to be the product of an underlying resting tremor which is masked by the rigidity but can be felt on passive movement .

b-Lead-pipe rigidity . Lead pipe rigidity describes a constant resistance where when moving a joint

C- Decerebrate (3-extension of head & 4 limbs extensors)& decorticate rigidity(extensor rigidity in legs & moderate flexion of arms if head unturned)


THANK
YOU