Physiology Team 439

MED439
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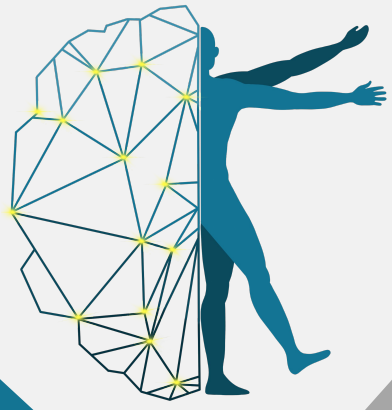

Spasticity and Increased Muscle Tone

Objectives:

- Define spasticity and rigidity .
- Describe the neurophysiology of spasticity
- Describe the causes of spasticity

Color index:

- Important.
- Girls slide only.
- Boys slide only.
- Dr's note.
- Extra information.

* = girls slides only
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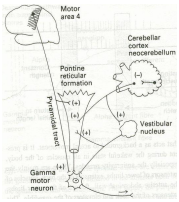
Editing File

Introduction



These facilitatory supra spinal centers excite gamma motor neurons , Increased Gamma efferent discharge is the main cause of increased muscle tone.*

But what is 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.*

What is hypertonia?

Refers to increase resistance to passive stretch (passive lengthening) of a muscle. This may mean increased stiffness of the muscle because of hyperactive stretch reflexes.

Hypertonicity could be due to a neural drive problem such as:

Spasticity or rigidity.

We have 2 types of motor neurons:

1-Upper motor neuron (UMN): Arises from higher levels either cerebral cortex or brain stem, and there are 2 types of UMN: pyramidal and Extrapyramidal

2- Lower motor neuron (UMN): Arises from anterior horn cell to skeletal muscle

Overview

	Spasticity	Rigidity
Character	❖ velocity dependent ❖ usually uni-directional (only in against) ❖ Involvement of the corticospinal tract ❖ is often associated with UMNL	❖ not velocity dependent ❖ present in both agonist and antagonist muscles. ❖ Rigidity is usually extra-pyramidal in origin ❖ It is often associated with basal ganglia disease such as Parkinson's disease
Type	❖ (UMNS) syndrome include : ➢ Cerebral palsy ➢ Stroke ➢ Spinal cord injury ➢ Multiple Sclerosis ➢ Acquired brain injury (trauma , etc)	❖ Rigidity in Parkinsonism: ➢ Lead-pipe rigidity ➢ Cog-wheel rigidity ❖ Decerbrate rigidity ❖ Decorticate rigidity

Rigidity

Usually due to Extrapyramidal lesion

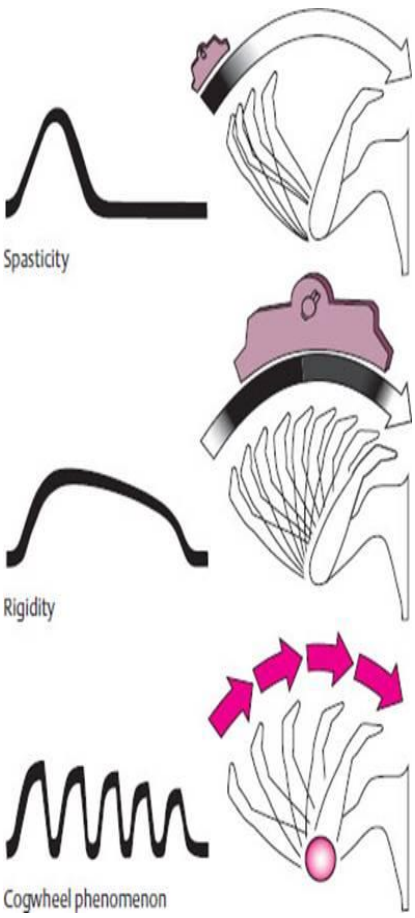
L23

Definition

Rigidity is increased resistance or neural activity throughout the range of muscle movement and not related to the speed of muscle movement (not velocity dependent).

Characteristics:

1. In rigidity resistance is present in both agonist and antagonist (bi-directional).
2. Rigidity is usually extra-pyramidal in origin & rigidity includes other features of increased muscle tone
3. Stiffness is different from rigidity. Stiffness is a principle symptom of the patient (complain).
4. Rigidity is often associated with basal ganglia disease such as Parkinson's disease.



Causes of rigidity:

1. Parkinsonism
2. Decerebrate
3. decorticate

Rigidity

L23

Causes of Rigidity:

1 Parkinsonism rigidity is of two types:

-Lead-pipe rigidity:

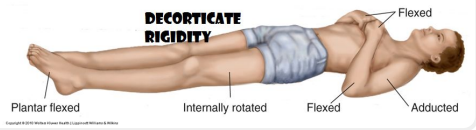
- Passive movement of an extremity meets with a constant dead feeling resistance like a lead pipe throughout the range of movement.
- Lead pipe rigidity describes a constant resistance when moving a joint.

-Cog-wheel rigidity: Lead-pipe + tremors = Cog-wheel rigidity

- In cogwheel rigidity one feels that resistance varies rhythmically (catches) when applying a passive movement,
  it's because of an underlying resting tremor which is masked by the rigidity but can be felt on passive movement.

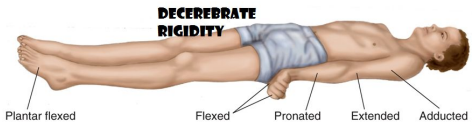
2 Decorticate rigidity:

- Extensor rigidity in legs & moderate flexion of arms if head unturned



3 Decerbrate rigidity:

- Extension of head & 4 limbs extensors



Testing for Rigidity: Girls slide only.

passively move the joint in both direction:

If there is a relatively uniform rigidity in both agonist and antagonist muscle group this is known as. **lead-pipe rigidity**.

if there is tremor superimposed with background increase of tone is **Cogwheel rigidity**.These rigidity is commonly seen in Parkinson's disease

Spasticity

Usually due to a lesion in pyramidal tract

L23

Definition

As described by lance (1980): "It's a motor disorder, characterised by increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex as one component of the upper motor neuron (UMN) syndrome.
Clinically it can be defined as increased resistance to passive stretch.

Characteristics:

1

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. (proportional with speed of flexion)
- The faster you stretch the muscle the greater the resistance.

2

Neural in nature:

- Spasticity is clearly neural in nature (can't be caused by muscle diseases) and is associated with (UMNL) due to involvement of the corticospinal tract.

3

Usually Uni-directional: Either affecting the agonist or antagonist.

- Flexor spasticity in the upper limb & extensor spasticity in the lower limb.

(no spasticity while extending arms or flexing legs)

In the beginning of flexion or extension (depending on the patient presenting problem) the patient will feel resistance, but as the movement continue the resistance disappears.due to activation of Golgi tendon reflex

4

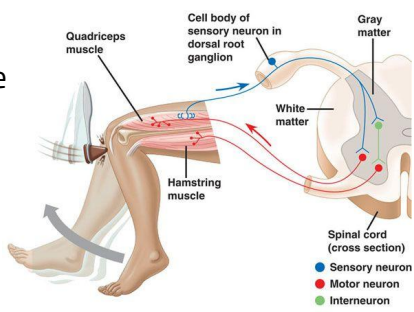
clinical features:

1. Hyperreflexia.
2. UMN lesions spasticity is of **clasp knife type.**  (مثل كسارة البندق) 
3. Spasticity with the increased muscle tone together cause a contraction and deformity of a limb.

5

hyper-excitability of both types of stretch reflex:

- Increase in tonic static (while resting) stretch reflexes (muscle tone) as one component of the upper motor neuron (UMN) syndrome.
- Exaggerated tendon jerks, resulting from hyper-excitability of the dynamic stretch reflex as one of the upper motor neuron (UMN) syndrome.



Spasticity

A simple way to assess spasticity : Girls slide only.

- Fast flexion or extension of selected joint, typically the elbow or knee, to elicit a sudden increase in tone. & demonstrate the velocity dependent nature of spasticity.
- Clasp-knife spasticity in UMNL, describe a sudden release of resistance after an initial hypertonia of selected joint movement.

- Spasticity & hypertonia is a feature of altered muscle performance.
- Usually in Upper Motor Neuron Syndrome (UMNS).
- Patients complain of stiffness & inability to relax.
- Muscle become permanently “tight” or spastic.
- The condition can interfere with walking, movement, or speach.

Spasticity is caused by UMN syndrome which includes:

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

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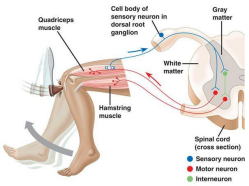
Features of UMN Syndrome:

01 Weakness and decrease muscle control.

02 No remarkable muscle wasting, but disuse atrophy.

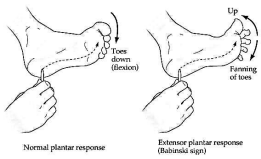
03 Spasticity & hypertonia, frequently called "Clasp-knife spasticity"= increased resistance at the beginning of muscle stretch due to increased extensor muscle tone then a sudden collapse in resistance due to inhibition of extensor motor neuron by GTOs (golgi tendon organs) when I raise and flex the arm at elbow joint I will stretch triceps muscle which is already hyperactive or hypertone so the tone inside the muscle will increase more and more during passive stretches which will activate Golgi tendon reflex>>> relaxation

04 Clonus repetitive jerky motions (clonus), especially when limb moved & stretched suddenly. Clonus is a series of involuntary, rhythmic, muscular contractions and relaxations.



05 Exaggerated tendon jerks.

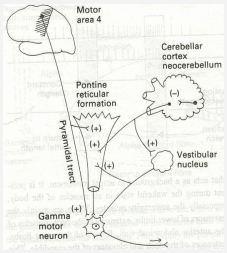
06 Extensor plantar reflex = **Babinski sign**
dorsiflexion of the big toe and **fanning** out of the other toes.
(Stimulation of plantar side result in dorsiflexion instead of plantarflexion)



07 Absent abdominal muscle reflexes.

Mechanism of spasticity in UMN lesions:

- In UMN syndrome there's a loss of descending inhibition from the brain higher motor-inhibitory centers (Medullary RF ,red nucleus,
- basal ganglia & suppressor area 4) resulting in un-antagonized excitatory input (to make balance) from brain stem excitatory centers As (Pontine RF & vestibular N) through vestibulospinal & reticulospinal excitatory tracts to gamma motor neurons causing hypertonia & spasticity of muscle.



This will result in:

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

Causes of spasticity

1 Cerebral palsy:

- **Caused by:** brain damage due to lack of oxygen (as near drowning or near suffocation) which causes damage to the motor control centers of the developing brain
- **It's can occur during:**
  - Pregnancy
  - during stressed childbirth or after birth up to about age three by meningitis
- **Spastic Cerebral Palsy:**
  - There are 3 major forms of CP, but spastic CP is the most common (70%-80% of all affected).
  - it is a form in which there is an increase in muscle tone, tense and contracted muscle.
- **Signs of Spastic Cerebral Palsy:**
  - Stiff, jerky and awkward movements.
  - limbs areusually Underdeveloped.
  - Increased deep tendon reflexes.

2 Multiple Sclerosis:

- Autoimmune 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 sheaths (demyelination) preventa axons from saltatory conduction of action potentials causing muscle weakness and wasting.
- Disease onset usually occurs in young adults, and it's 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 and spinal cord, the subject develos spasticity and sings of UMNS.
- The disease frequently remits and relapses because of remyelination & restore of function.
- During acute attacks IV corticosteroids can improve symptoms .

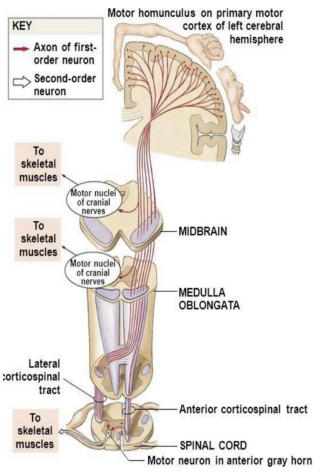
3 Stroke:

Causes:

- 1-Hemorrhagic stroke as in cerebral hemorrhage
- 2-Ischaemic stroke as in thrombosis or embolism in the brain blood Vessels.

1 and 2 can cause death of brain tissue which 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 .



Causes of spasticity:

4 Complete Transection of Spinal Cord:

The higher the level of the section, the more serious are the consequences because it affect respiration.**e.g. following tumor or trauma**

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)**

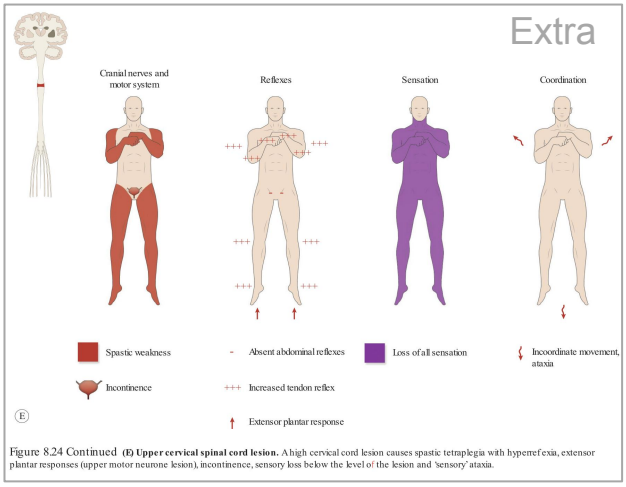


Figure 8.24 Continued **(E) Upper cervical spinal cord lesion.** A high cervical cord lesion causes spastic tetraplegia with hyperreflexia, extensor plantar responses (upper motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia.

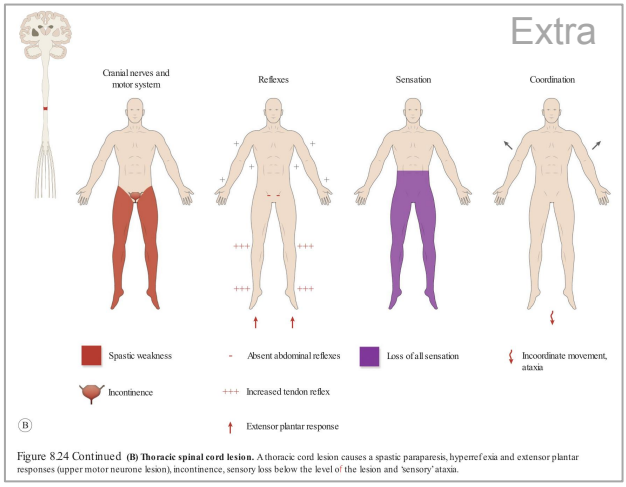


Figure 8.24 Continued **(B) Thoracic spinal cord lesion.** A thoracic cord lesion causes a spastic paraparesis, hyperreflexia and extensor plantar responses (upper motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia.

Stages of Complete Transection of Spinal Cord

Spinal shock
(2-6 weeks)

Recovery of reflex activity

Paraplegia in extension

*there is spasticity in the first two stages, and no rigidity

Stages of Complete Transection of Spinal Cord:

A-Spinal shock

This stage varies in duration but usually lasts a maximum of 2-6 weeks, after which some reflex activity recovers. in animas it last for a fewer weeks, because they have a less developed brain.

In the immediate period following transection there is :

- **Loss of all sensations** (anesthesia) and loss of voluntary movements (paralysis) below the level of lesion, due to interruption of all sensory and motor tracts.

- **Loss of tendon reflexes and superficial reflexes** such as: abdominal, planter and withdrawal reflexes, (which means complete loss of spinal reflexes below the level of the lesion)

- **Loss of muscle tone (flaccidity)** and loss of any muscle activity (muscle pump) which leads to decreased venous return causing the lower limb to become cold and blue in cold weather.

- **Loss of visceral reflexes such as:** micturition, defecation and erection reflexes.
  the wall of the urinary bladder becomes paralyzed the urine is retained until the pressure in the bladder overcomes the resistance offered by tone of the sphincters, as a result dribbling occurs which is know as retention with overflow

- **Loss of vasomotor tone** due to interruption of fibres that connect the vasomotor centres in the medulla oblongata with the lateral horn cells of the spinal Cord of 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

- Bedsores due to pressure of body weight against underlying support

Stages of Complete Transection of Spinal Cord:

B-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 , because of disinhibition of motoneurons due to absence of inhibitory impulses from higher motor centres
- Sprouting (new branches) of fibers from remaining neurons undamaged neurons will send collaterals.
- Denervation hypersensitivity to excitatory neurotransmitters

Features of the stage of recovery of reflex activity:

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 in absent, the blood pressure is not stable.and vasoconstriction tone in arterioles and venules, improve the circulation through the limbs

Return of spinal reflexes:

- Flexor reflexes(e.x. Flexor withdrawal reflex) return earlier than extensor ones.
- Positive Babinsiki sign (extensor plantar reflex) is one of the earliest signs of this stage +/- flexion reflex
- Tendon reflexes also recover in flexors
- As a result, flexor spastic tone causes the lower limb to take a posotion of slight flexion, a state referred as to as paraplegia in flexion
- The return of the stretch reflex &(consequently muscle tone)

Recovery of visceral reflexes:

- Return of micturition (automatic evacuation), defecation & erection reflexes
- Voluntary control over micturition and defecation, and the sensation of bladder and rectal fullness are permanently lost (automatic micturition).*while in first stage there is dribbling

Sexual reflexes:

- Consisting of erection or ejaculation on genital manipulation, recover (mentioned by girls Dr)

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.

Stages of Complete Transection of Spinal Cord:

C-Stage of Extensor Paraplegia:

1. During this stage the tone of the extensors returns gradually to exceed the tone of the flexors.
   - ➢ The lower limb become spastically extended
   - ➢ Extensor reflexes become exaggerated as shown by tendon jerks and appearance of **clonus**
   - ➢ The patient can stand on his feet with appropriate support as the positive supportive reaction becomes well developed

2. During this stage the flexor withdrawal reflex which showed in the earlier stage is instead associated the crossed extensor reflex

Summary

- Spasticity is resistance to passive stretch + an involuntary + velocity- dependent + unidirectional - - - leads to----> resistance to movement
- Rigidity is resistance to passive movement + an involuntary + not velocity-dependent + bidirectional - - - -- leads to- --> resistance to movement

Hemisection of the Spinal Cord (Brown- Sequard syndrome)

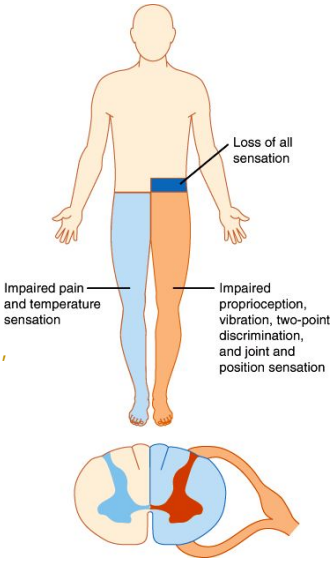
Girls slide only.

Results of unilateral lesion or hemisection of the spinal cord (e.g due to Stab injury, bullet, car accident TB, Gunshot , 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 :

1 At the level of the lesion :

All manifestations occur on the same side

1. Paralysis of lower motor neuron involving only the muscle supplied by the damaged segments
2. Loss of all sensations in the areas supplied By the afferent fibers that enter the spinal cord in the damaged segments +/- band of hyperesthesia or anaesthesia
3. Vasodilation of the vessels that receive Vasoconstrictor fibers from the damaged segments



2 Ipsilaterally below the level of the lesion:

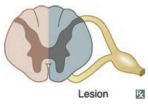
1. Paralysis of UMN due to interruption of Pyramidal and extrapyramidal tracts
2. UMNL/spastic lower limb (spasticity) &CLONUS
3. Loss of fine touch, two-point discrimination, Position and vibration sensations **why?** (Below the level of the lesion, there are tracts from the spinal cord (dorsal column/gracile and cuneate tracts) carry the sensation of the same side and ascend to the brain, which will be cut off)
4. Vasodilation

3 Contralaterally below the level of the lesion:

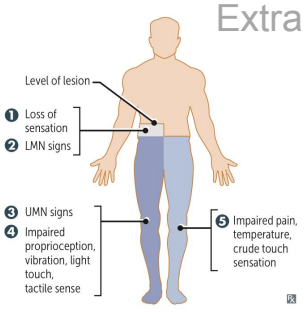
- Loss of temperature and pain sensations **Why?** (The lateral and ventral spinocortical tracts that cross below the level of the lesion and ascends to the opposite side of the brain will be cut off)

Extra

Brown-Séquard syndrome



Hemisection of spinal cord. Findings:
❶ Ipsilateral loss of all sensation **at** level of lesion
❷ Ipsilateral LMN signs (eg, flaccid paralysis) **at** level of lesion
❸ Ipsilateral UMN signs **below** level of lesion (due to corticospinal tract damage)
❹ Ipsilateral loss of proprioception, vibration, light (2-point discrimination) touch, and tactile sense **below** level of lesion (due to dorsal column damage)
❺ Contralateral loss of pain, temperature, and crude (non-discriminative) touch **below** level of lesion (due to spinothalamic tract damage)
If lesion occurs above T1, patient may present with ipsilateral Horner syndrome due to damage of oculosympathetic pathway.



MCQ & SAQ:

Q1: Which one of the following is a characteristic of the spasticity:

A. Bi-directional resistance
B.Associated with parkinson's disease
C. Extra-pyramidal in origin
D. Corticospinal involvement

Q2: Constant resistance when moving the joint describes:

A.Clasp knife type
B. Decrerbrate rigidity
C. Cogwheel rigidity
D. Lead pipe rigidity

Q3: Which one of the following is not associated with UMN syndrome:

A.Clasp knife spasticity
B. Babinski sign
C. Disuse atrophy
D. Muscle wasting

Q4: Spinal shock causes:

A.loss of all sensations
B. Loss of tendon reflexes and superficial reflexes
C.loss of muscle tone
D.all above

Q5: Loss of pain and temperature sensation caused by lesion at which level?

A.at the level of lesion
B.ipsilaterally below the level of lesion
C.contralaterally below the level of lesion
D.none of the above

Q6: Cerebral palsy can occur during which of the following:

A. Pregnancy
B. Normal childbirth
C. Stressed childbirth
D. A & C

answer key:
1: D
2: D
3: D
4: D
5: C
6: D

1- Describe briefly the difference between Lead-pipe rigidity and Cog-wheel rigidity?

2- What is the mechanism of spasticity in UMN lesions?

3-What are the stages of complete transection of spinal cord? What is duration of the first stage?

4- what causes the cerebral palsy?

A1: Lead-pipe rigidity: constant resistance when moving the joint.
Cog-wheel rigidity: resistance varies rhythmically when applying a passive movement.

A2: loss of descending inhibition from the brain higher motor-inhibitory centers resulting in un-antagonized excitatory input from brain stem excitatory centers to gamma motor neurons causing hypertonia & spasticity.

A3: Spinal shock, return of reflex activity, , paraplegia in extension / the duration of spinal shock is 2-6 weeks

A4: brain damage due to lack of oxygen which causes damage to the motor control centers of the developing brain

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