23

Spasticity and increased Muscle Tone

PHYSIOLOGY
CNS BLOCK
TEAM 443

MED 443
King Saud University

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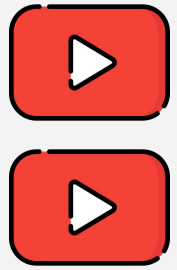
Editing File

Objectives:

1. Define **spasticity**, **rigidity** & hypertonia
2. Know main /describe causes of **spasticity** & rigidity
3. Describe the neurophysiology of spasticity

Highly recommend!!



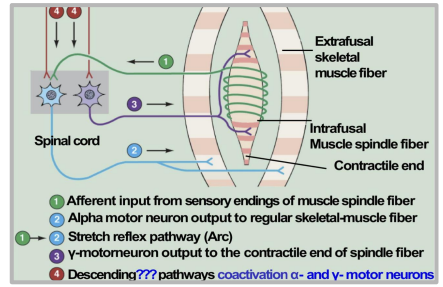
*Means=Male slides
*Means=Female slides

*Due to the huge difference between Female and Male slides it is better for the eye

Dr Faten said I wrote questions from this lecture :)

Introduction

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)*
(The main cause of increased muscle tone is: Increased Gamma efferent discharge How?) *



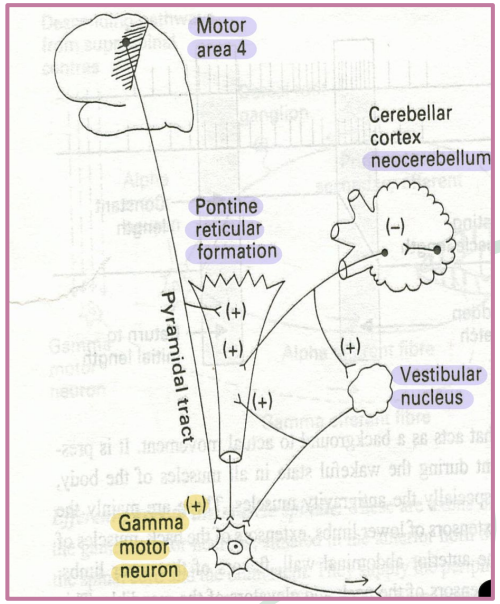
**it is a balance between excitatory and inhibitory centers of the brain which affect the stretch reflex predominance of excitation will cause hypertonia, and predominance of inhibition will cause hypotonia

Hypertonia

-(Refers to **increased resistance** to **passive** stretch (passive lengthening) of a muscle, This may mean increased stiffness of the muscle).*/ (A hypertonic muscle is one in which the resistance to stretch is high because of hyperactive stretch reflexes).*
Hypertonicity is of two types/could be due to a **neural** drive problem such as:

1	Spasticity
2	Rigidity

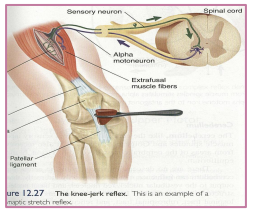
(These are facilitatory **supraspinal** centers to gamma motor neurons)*



Spasticity

Definition | Testing | Characteristics | Causes

What is the difference between UMNL &UMNS:
Upper motor neuron lesion describes the physical damage but upper motor neuron syndrome describes the clinical condition



Clinically it can be defined as **Increased resistance to passive stretch/ movement of the muscle due to abnormally high muscle tone (hypertonia) which varies with the speed of displacement of a joint.**

(As described by Lance (1980): "it is a motor disorder, characterised by increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks(dynamic reflex), resulting from hyper-excitability of the stretch reflex as one component of the upper motor neuron (UMN) syndrome). *

Important

Characteristics

Velocity dependent, the faster you stretch the muscle the greater the resistance.

clearly Neural in nature and is a associated with the UMNL due to Involvement of the corticospinal tract (Pyramidal tract){1}

Usually uni-directional, The effect :Flexor spasticity in the upper limb & extensor spasticity in the lower limb. {2}

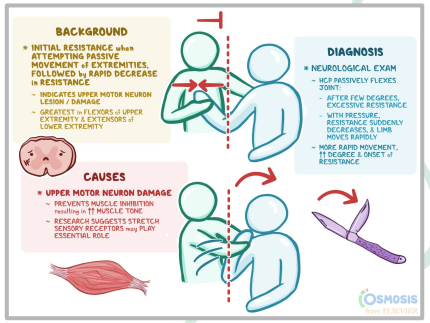
hyper-Excitability of both types of stretch reflex:
1- Increase in tonic static stretch reflexes (muscle tone) as one component of the upper motor neuron (UMN) syndrome.
2- Exaggerated tendon jerks, resulting from hyper-excitability of the dynamic stretch reflex as one component of the upper motor neuron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

{1}: UMNL is damage to the pathway connecting the upper motor neuron to the spinal cord,usually it is due to hemorrhage or stroke).
{2}: Either agonist or antagonist group of muscles)

Spasticity cont..

Definition | Testing | Characteristics | Causes

- (A simple way to assess spasticity, is by 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)*
- (There are a number of clinical features that are associated with spasticity :-
  -hyperreflexia and clonus)*
- **Clasp-knife spasticity in UMNL** (describe a sudden release of resistance after an initial hypertonia of selected joint movement)*. (مثل كسارة البندق بالبدايه مقاومه عاليه لانه البندقه ناشفه بعدها المقاومه تفك و البندقه تتكسر,باختصار بيكون فيه مقاومة في نقطة معينه من الحركة بالبدايه فيه **resistance** لانه الـ**stretch reflex** يشتغل بعدين لما يزيد مره يشتغل الـ **inverse stretch reflex**.
- **Spasticity & hypertonia:** (is a feature of altered muscle performance, Usually in UMNS)*.
  - (together cause a contraction and deformity of a limb.)*
  - (Patients complain of stiffness & inability to relax.)*
  - (Muscles become permanently "tight" or spastic.)*
  - (The condition can interfere with walking, movement, or speech.)*



Causes of spasticity

(UMNS) syndrome include:

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 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

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).(هنا الفايبرز تكون متفرقه فـ عشان كذا الشلل يصيب طرف واحد)
-A lesion in the internal capsule on one side may cause hemiplegia or hemiparesis on the contralateral side (with/gives the picture of upper motor neuron syndrome)(هنا الفايبرز تجمعت فـ عشان كذا الشلل صار نصفي)

Cerebral palsy

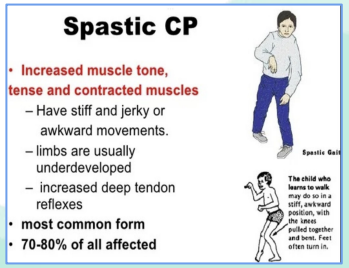
Caused by brain damage due to lack of oxygen (near drowning or near suffocation), that cause damage to the motor control centers of the developing brain.
It can occur during pregnancy , during stressed childbirth (or after birth up to about age three by meningitis or encephalitis).

Spinal cord injury

Complete transection of spinal cord.(will be discussed later)

Acquired brain injury (trauma, etc)

Cerebral palsy



Rigidity

Definition | Characteristics | Testing | Causes

Important

- Increased neural activity/ resistance to the passive movement which is constant throughout the range of muscle movement.
- (Stiffness is different from rigidity. Stiffness is a principal symptom of the patient (complain).)*

(يجي العيان يقول انا حاسس بstiffness مايعرف يشرح لك انها rigidity)

Characteristics

- Not velocity dependent, not related to the speed of movement.
- Usually extra-pyramidal in origin & Rigidity includes other features of increased muscle tone .
- Bi-directional, resistance is present in both agonist and antagonist muscle.
- Often associated with basal ganglia disease such as Parkinson's disease.
(كان في محمد علي كلاي)

Rigidity cont..

Definition | Characteristics | Testing | Causes

(To test for rigidity, passively move the joint in both direction:)*

Causes of Rigidity

1-Parkinsonism rigidity is of two types

a-Cog-wheel rigidity

In cogwheel rigidity /one feels the resistance in rhythms (catches)/varies when applying a passive movement. It is due to an underlying resting tremor which is masked by the/associated with rigidity but can be felt on passive movement .

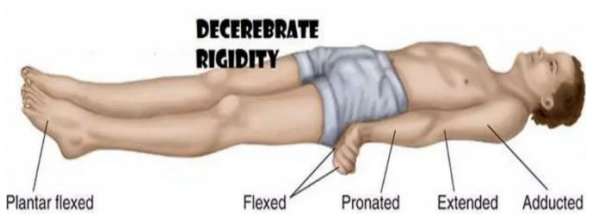
b-Lead-pipe rigidity .

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

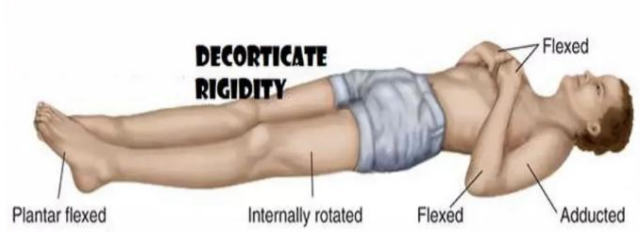
Female slides

lead-pipe rigidity	Cogwheel rigidity
a relatively uniform rigidity in both agonist and antagonist muscle group. (Rigidity only)	if there is tremor superimposed with background increase of tone. (Rigidity +tremor)

2- Decerebrate rigidity (extension of head & 4 limbs extensors)



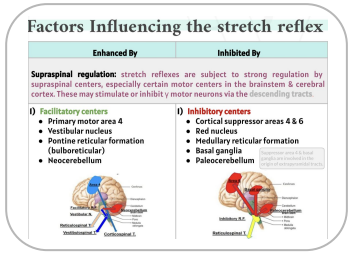
3- Decorticate rigidity(extensor rigidity in legs & moderate flexion of arms if head unturned)



UMNS (Upper Motor Neuron Syndrome)

Mechanism of spasticity in UMN lesions:

In UMN syndrome there is a loss of descending inhibition from the brain higher motor controlling -inhibitory-centers (medullary RF, red nucleus , basal ganglia, suppressor area 4) resulting in un antagonized excitatory input from brain stem excitatory centers As (pontine RF + vestibular N) through Vestibulospinal & reticulospinal excitatory tracts to gamma motor neurons causing hypertonia & spasticity, (State of ongoing (unremitting)contraction of muscles,decreased ability to control movement, increased resistance felt on passive stretch)*



حنا قلنا ان الـ tone is balance between excitatory and inhibitory, في حال UMNL بنفقد 4 من الـ5 inhibitory centers, وفي الـ excitatory centers بنفقد 2 من اصل 4 , فـ المحصلة عندي
2 excitatory centers > I inhibitory center, so we will have predominance of excitation

Features of UMNS:

Click on the reflex for a video

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 beginning 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. Hyperreflexia, Exaggerated tendon jerks.
6. (Extensor plantar reflex = Babinski sign (dorsiflexion of the big toe and fanning out of the other toes))*
   (Physiological sign in infants, deep sleep & under anesthesia). Normal reflex is plantarflexion of all the toes
7. (Absent abdominal reflexes)*.

Complete transection of Spinal cord

Complete transection of spinal cord:- e.g. following tumor or trauma [1]

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

Dr.Nagi 🌟Min 28:55

1- If the transaction is in the upper cervical region:

immediate death follows, due to paralysis of all respiratory muscles [2]

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

Spinal shock (2-6 weeks) → Recovery of reflex activity → Paraplegia in extension

I: (The whole segment from the two sides)

2:(phrenic nerve damage), (Remember: phrenic Roots 3,4,5C 'keeps you alive!')

A/ Spinal shock

In the immediate period following transection there is :

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

1. **Loss of all sensations (anaesthesia)**

and voluntary movement, (complete paralysis) below the level of the lesion , due to **interruption** of all **sensory** and **motor** tracts.

2. **Loss of tendon reflexes and superficial reflexes**

(abdominal , plantar & withdrawal reflexes) =(Complete loss of spinal reflex activity below the level of the lesion)* [1]

3. **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 [2].

1: (All deep+Superficial reflexes will be lost)

2: In cardiovascular block we used to say that limb muscles (specifically Lower limbs) is peripheral hearts!

A/ Spinal shock cont..

4 (Loss of visceral reflexes (micturition ,defecation & erection reflexes)*

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) [1]

5 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 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)*.[2]

6 (Bed-Sores)*

(due to pressure of body-weight against underlining support)*.

1: Retention with overflow/ Overflow incontinence / Dribbling (Same meaning)
2: (if there is good care the BP will return in 6 hours)

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 motor neurons due to absence of inhibitory impulses from higher motor centres)*
2-**sprouting** of fibres from remaining neurons [1]
3-**Denervation** super/hyper sensitivity to excitatory neurotransmitters . [2]

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(i.e. flexor withdrawal reflex)return earlier than extensor ones.
- However positive Babiniski sign (extensor plantar reflex) is one of the earliest signs of this stage +/- flexion reflex .
-(Tendon reflexes also recover earlier in flexors.)*
- as resulte Flexor spastic tone causes the lower limbs to take a position of slight flexion, a state referred to as paraplegia in flexion. [3]
- The return of the stretch reflex (muscle tone)

1 : كأنها تتبرعم، تطلع فايبرز Collaterals تنشط بعضها
2 :(receptors for neurotransmitters will be more sensitive)
3 : العيان نايم على السرير بس ساحب رجله عند بطنه

B/ Stage of return of reflex activity

(3) Recovery of visceral reflexes: return of micturition(automatic evacuation), defecation & erection reflexes.
- However voluntary control over micturition and defecation , and the sensation of bladder and rectal fullness are permanently lost(automatic micturition)

(4)(Sexual reflexes, consisting of <u>erection</u> or <u>ejaculation</u> 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 <u>irradiation</u>) to many autonomic centres. 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.)*

<u>Voluntary</u> movements and **<u>sensations</u>** are permanently **<u>lost</u>**, however; patients who are rehabilitated and properly managed may enter into a more advanced stage of recovery.

الإحساسات والـVolantry movevent تحت Lesion عمرها ما ترجع !
ربط من دكتور ناجي: (ودّع هواك وانساه وانساني، عمر الـsensations والـmotor activity ما هترجع تاني :)

C/ Stage of extensor paraplegia

(I) During this stage the tone in extensor muscles returns gradually to exceed that in the flexors. The lower limbs become spastically extended. (المريض لو وقفته على رجله في هذي المرحله بيوقف)

-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 , car accident,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

I.Paralysis of the lower motor neuron type

involving only the muscle supplied by the damaged segments.

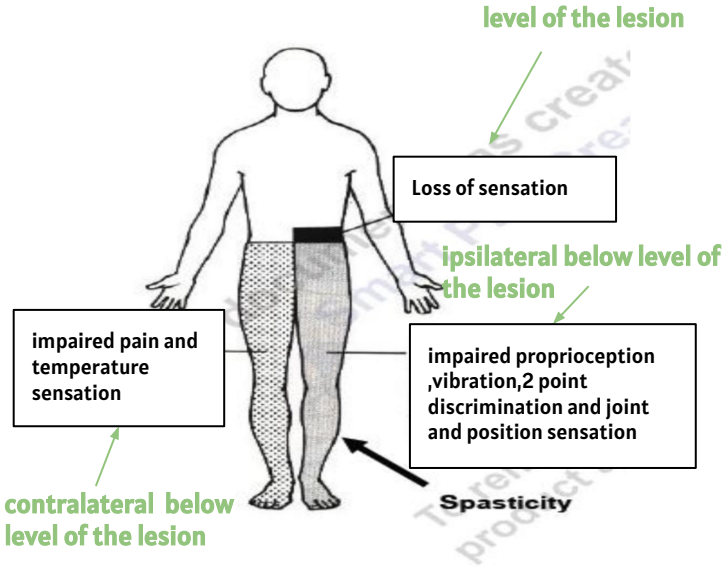
2. Loss of all sensations

In the areas supplied by the afferent fibres that enter the spinal cord in the damaged segments +/- band of hyperesthesia

عند بعض المرضى ممكن يحصل Irradiation فينتج عن ذلك hyperthesia

Hemisection of the Spinal Cord (Brown-Sequard syndrome)

At the level of the lesion: **all manifestations occur on the same side**	I. 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
Ipsilaterally below the level of the lesion :	I.Paralysis of UMN type due to interruption of pyramidal and extrapyramidal tracts.(ipsilateral because the lesion is below the crossing level) 2. (UMNL/spastic lower limb (spasticity) &CLONUS,spastic paresis below lesion"LatCorticospinal")* 3. Fine touch, two-point discrimination, position and vibration sense are lost. why?(loss of dorsal column, injury below the crossing level, so it is ipsilateral) (Leg Ataxia "Dorsal Spinocerebellar")* 4.(Vasodilatation)*
Contralaterally below the level of the lesion :	Pain and temperature sensations are lost, Why ? (injury in Lateral spinothalamic tract cause loss of pain and temperature, why is it contralaterally? because the crossing in the spinal cord) (loss of crude touch and pressure (vent spinothalamic))* (minor muscle weakness (vent cortciospinal))* (Leg Ataxia (Vent Spinocerebellar))*

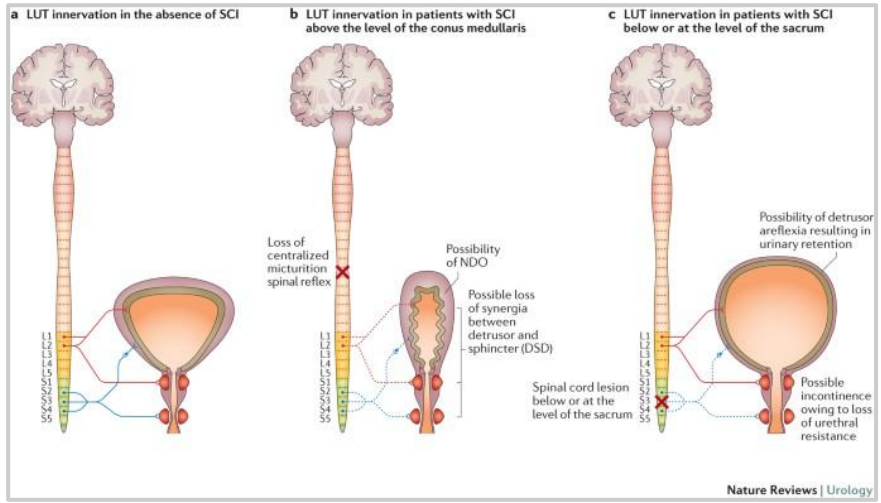


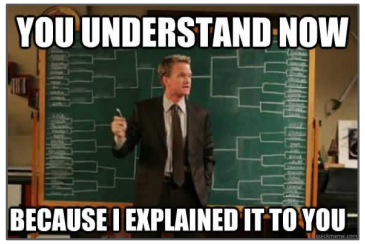
Rigidity vs spasticity

	Rigidity	Spasticity
Definition	Increased resistance to the passive movement of a muscle which is constant throughout the movement.	Increased resistance to passive movement of the muscle due to abnormally high muscle tone (hypertonia)
Characteristics	Not velocity dependent Bi-directional extrapyramidal in origin	Velocity dependent Corticospinal tract (Pyramidal tract) origin Uni-directional Excitability of both types of stretch reflex
Types	lead-pipe rigidity Cogwheel rigidity	Clasp-knife spasticity
Causes	Parkinson's disease Decerebrate rigidity Decorticate rigidity	(UMNS) include: Cerebral palsy Stroke Spinal cord injury Multiple Sclerosis Acquired brain injury (trauma, etc)

Extra slide..

Renal Block Recall, Overflow incontinence vs Automatic bladder?





إذا كان injury فوق sacral nerves يعني انقطع التواصل بين الـ reflex والـ high brain centers (مثل واحد صار له حادث وجاه كسر في العمود الفقري فوق sacrum، مثلاً بالـlumbar) بتكون على مرحلتين (بس خلونا نتفق ان reflex is intact يعني الاعصاب اللي هناك كلها تمام بس بالبداية مارح يشتغل بس زي اللي انصدم skock لأن كانت ياخذ الأذن من فوق اذا
appropriate to urinate or not !
اما الحين لا،
(Lost the facilatotry impulses)
موجود الريفلكس وتمام لكن مارح يستمر ويصير urination لأن مايقدر هو تعود على ان فيه اشارة تسمح له (اللي انقطعت الحين)

🌟اللي هي المرحلة الاولى acute/spinal shock:
تستمر ست اسابيع تقريباً،
تمتلي الـbladder وتروح الـsignal على الـ Afferent ولكن will be inhibited ليه؟ قلنا فقد facilitatory signal اللي كان متعود عليها، الـbladder راح تمتلي urine الين يصير الضغط مره عالي واحنا نعرف ان الـ fluide moves from high pressure to the low)
فـ بيصير فيه leaking
وعشان كذا نسميه overflow incotninace
وبهالحاله يحتاج قسطرة (حتى تتحسن حالته)

🌟المرحلة الثانيه recovary phase:
(بعد ست اسابيع أو أكثر حسب الـseverity) من اسمها, يبدأ يشتغل الـreflex (يستوعب انه intact ومافيه شيء) ولكن هل بيرجع ارادي مثل اول؟لا، ليه؟ لأن راحت الـfacultatiry/inhibtory centers

ومايحتاج قسطرة، بيصير مثل الأطفال بالضبط بيكون عنده automatic bladder

TEST YOURSELF !

1.What is the main cause of increased muscle tone?

- A)Decreased gamma efferent discharge
- B)Increased gamma efferent discharge
- C)Decreased alpha motor neuron activity
- D)Increased alpha motor neuron activity

2. Spasticity is usually observed in which type of lesion:

- A)Upper motor neuron lesion
- B)Lower motor neuron lesion
- C) Peripheral nerve lesion
- D)Neuromuscular junction lesion

3.Spinal shock causes:

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

4-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

Answers:B,A,D,C

SAQ

Describe the difference between spasticity and rigidity in terms of their characteristics and causes.

Click here to go to the related slide

What is the mechanism of spasticity in UMN lesions?

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.

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

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

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Abdullah alzamil
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