LECTURE 28

SPASTICITY & INCREASED MUSCLE TONE

DONE BY: Ahlam Almutairi

REVIEWED BY: Leena Al-Yahya & Hamad Al-Mohsen

Physiology Team

OBJECTIVES

At the end of this lecture, student should be able to describe:

Not given

■ Slides ■ Important ■ Doctor's Notes ■ Explanation ■ Boy's Slides

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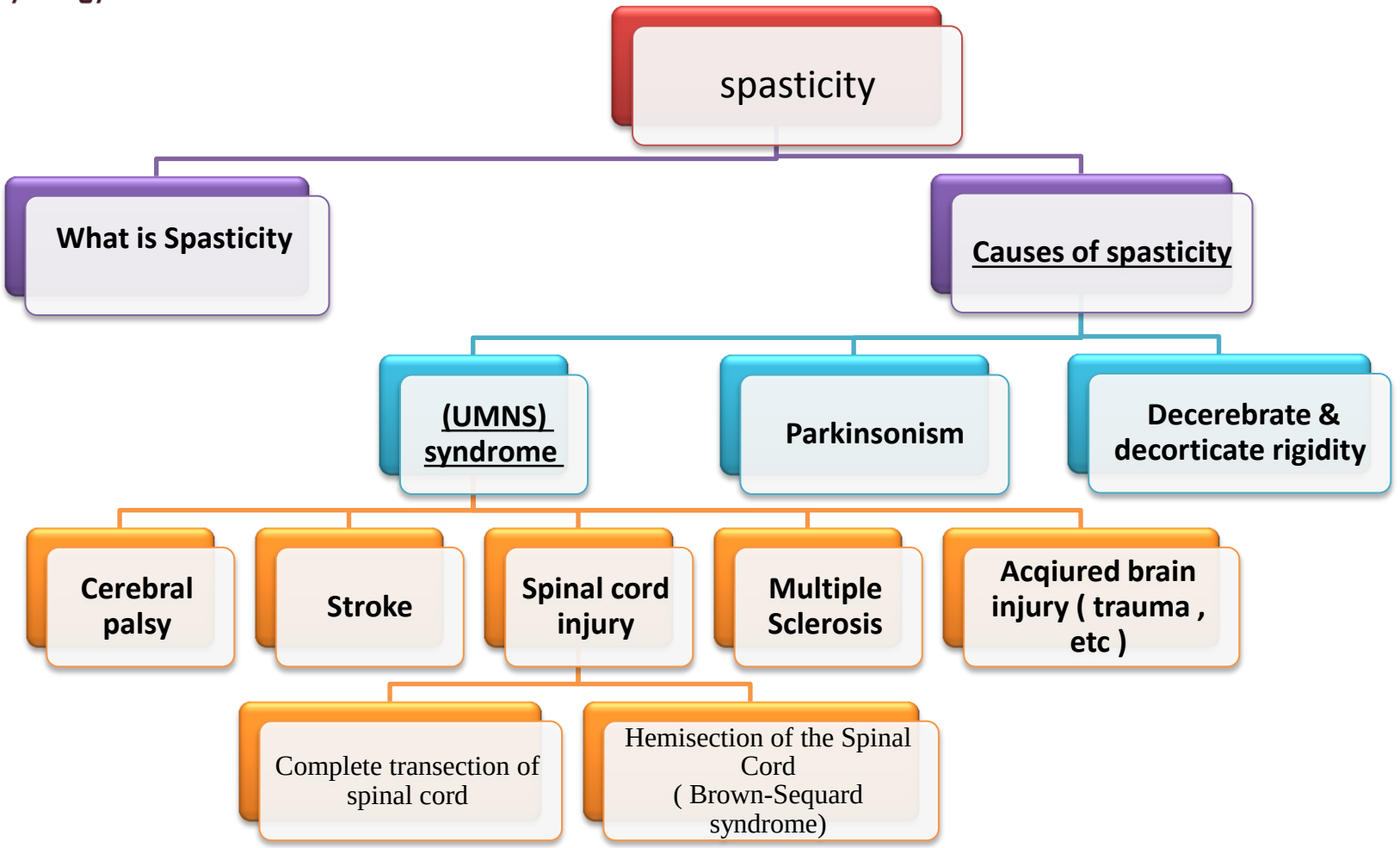
MIND MAP



Slides | Important | Doctor's Notes | Explanation | Boy's Slides

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Increased Gamma efferent discharge is the main cause of increased muscle tone(hypertonia).
how?

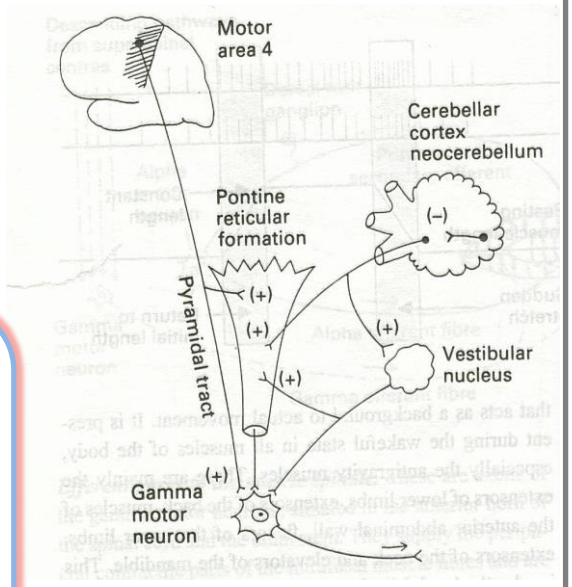
- if there is high input from Facilitatory supra spinal centers to gamma motor neurons through descending motor tracts gamma motor neuron hyper stimulate them to produce hyperexcitability in muscle spindle to produce hypertonia.

-when there is lost of descending inhibitory signals- to the gamma motor neurons (excitatory will take the upper hand)

in Both cases muscle tone will be increased

Remember!
Enhances of Suprspinal
-Primary motor area4
-Vestibular N
-Pontine RF
-Neocerebellum

Remember!
1-Supraspinal
-Cortical (suppressor area4&Area 6)
-Basal ganglia
-MedullaryRF
-Red nucleus
-paleocerebellum



Facilitatory supra spinal centers to gamma motor neurons

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Spasticity

-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

Spasticity is a motor disorder, characterised by:-

1- increase in tonic static stretch reflexes (muscle tone) as one component of the upper motor neurone (UMN) syndrome

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

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Features of UMN Syndrome

(1) Weakness and decreased muscle control

- because of the rigidity muscles are weaker

(2) No remarkable muscle wasting , but disuse atrophy

- Wasting occur if I cut the nerve supply but the nerve is still there so no wasting .)
- No use of the muscle will make it atrophic

(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

- Clonus: is a series of involuntary,rhythmic, muscular contractions and relaxations

(5) Exaggerated (Brisk) tendon jerks

6) Extensor plantar reflex = Babinski sign (dorsiflexion of the big toe and fanning out of the other toes)

- it can occur in normal people e.g.: children under 2 year, under sever anesthesia and coma

(7) Absent abdominal reflexes (Superficial reflexes)

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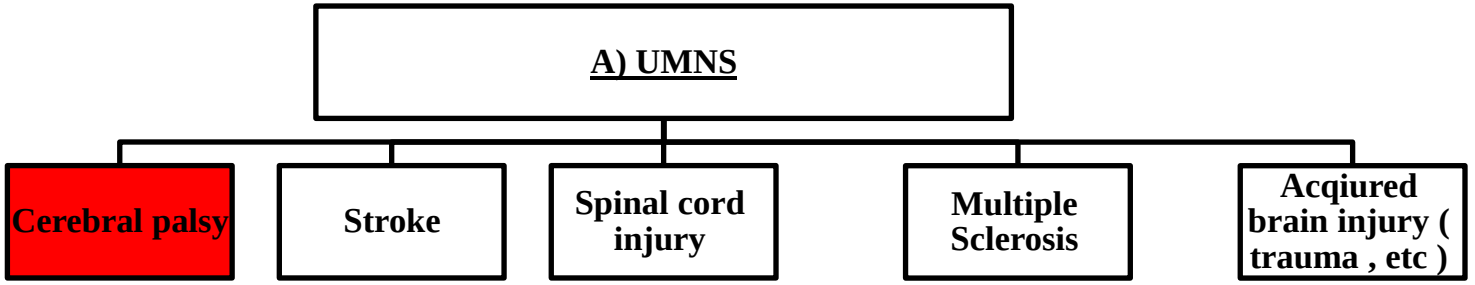
Causes of spasticity



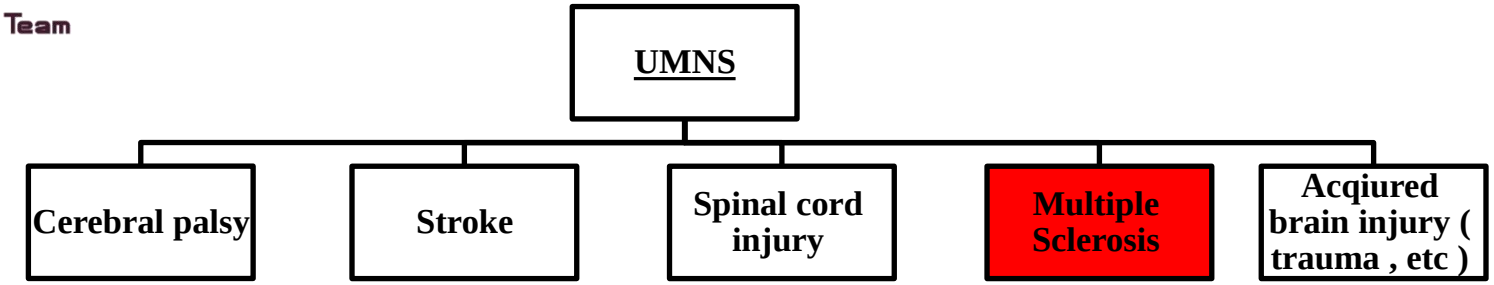
(1) Cerebral palsy -

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 cause physical disability in human development

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

Causes of spasticity



(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

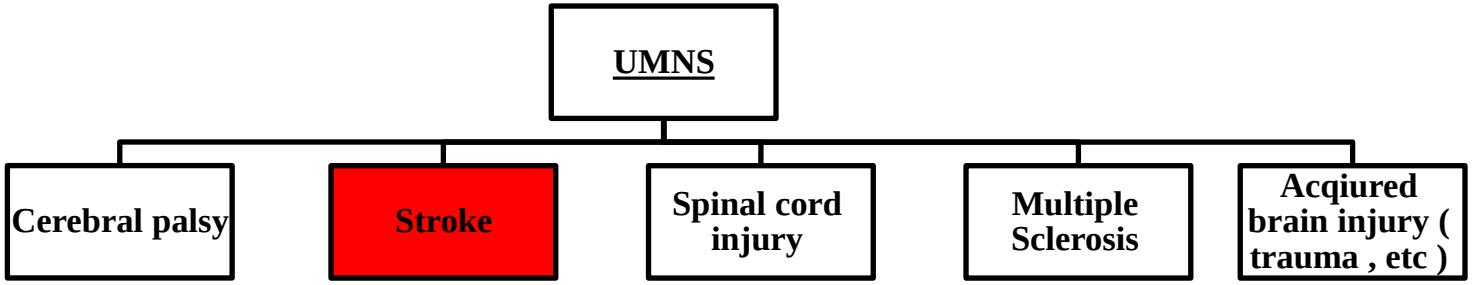
• Loss of myelin sheath (demyelination) prevents axons
from saltatory conduction of action potentials causing muscle weakness& wasting.

• 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, the subject develops spasticity and other signs of UMNS .

• The disease frequently remits and relapses because of remylination & restore of function

Causes of spasticity



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 .

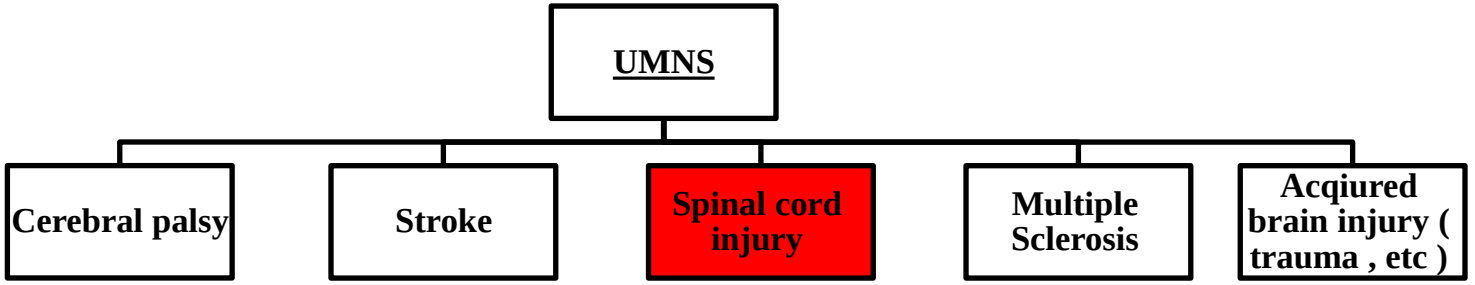
- A lesion in Corona Radiata on one side can cause Monoplegia in a contralateral limb (UL or LL ,according to site).
- A lesion in the Internal Capsule on one side may cause Hemiplegia or Hemiparesis on the contralateral side.
- with the picture of upper motor neuron syndrome UMNL

monoplegia is a paralysis of a single limb.

Hemiplegia is paralysis of the arm, leg, and trunk on the same side of the body.

Hemiparesis is weakness on one side of the body. including the legs

Causes of spasticity



4-Complete transection of spinal cord:- **e.g. following tumor or trauma.**

Depend where the lesion occur

•1- **If the transection is in the upper cervical region immediate death follows. Why?**
Due to damage of the respiratory centers and 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 of (quadriplegia).**

• 3-**Transection lower down in the thoracic region allows normal respiration but the patient ends up with (paraplegia)--**

Quadriplegia: paralysis of all four limbs
paraplegia :paralysis of the lower part of the body, including the legs

- Spasticity does not occur immediately following a spinal cord injury. When an injury occurs to the spinal cord, the body goes into spinal shock, and this may last several weeks. During this time changes take place to the nerve cells which control muscle activity.
- Once spinal shock wears off, the natural reflex which is present in everyone reappears.
- Spasticity is an exaggeration of the normal reflexes that occur when the body is stimulated in certain ways

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stages follow spinal cord transaction: Spinal shock → Recovery of reflex activity → Paraplegia in extension

A/ Spinal shock (2-6 weeks)

In the immediate period following transection there is :

(1) Loss of all sensations (anaesthesia) and voluntary movement (paralysis) below the level of the lesion
(2) Loss of tendon reflexes and superficial reflexes.
(3) The loss of muscle tone (flaccidity) and absence of any muscle activity(muscle pump)
muscle pump important in increasing venous return to the heart thus the patient will have edimitous,cold and blu extremetes
(4) 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 &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 (branching of SC neurons to excite nerve supply to muscles)

-supersensitivity to excitatory neurotransmitters .

• Features of the stage of recovery of reflex activity

• **(1) Gradual rise of arterial blood pressure** (unstable BP) due to return of spinal vasomotor activity in the lateral horn cells**.** - vasoconstrictor tone in arterioles and venules

2) Return of spinal reflexes:

- Flexor tendon reflexes return earlier than extensor ones.(e.g. biceps &quadriceps femoris)

-Babiniski sign

- Flexor spastic tone & paraplegia in flexion. (paraplegia in flexion is the fixation of the paralyzed legs in a flexed posture)

(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**.(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**;**

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- C/ **Stage of extensor paraplegia**

• (1) During this stage the tone in extensor muscles returns gradually to exceed that in the flexors. The The lower limb become spastic and 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.

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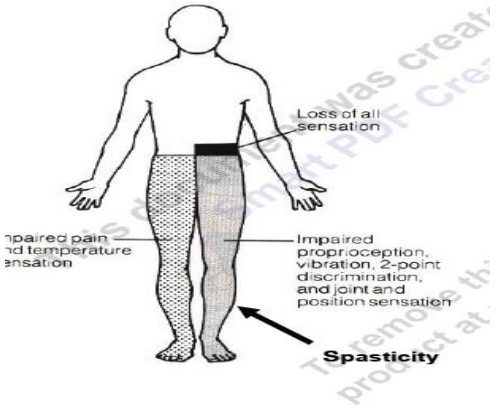
Hemisection of the Spinal Cord (Brown-Sequard syndrome)



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

A/<u>On the same side at the level of lesion</u>

1.Paralysis of the lower motor neuron type(involving only muscles supplied by damaged segment= LMNL there is Atonia, wasting and absence of reflexes on the same side of the lesion)

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

<u>B/ Ipsilaterally below the level of the lesion :</u>

1. UMNL/spastic lower limb (<u>spasticity</u>)&CLONUS

2. Fine touch, two-point discrimination, position and vibration sense are lost. why?

Due to cut of Dorsal column tracts.

C/ <u>Contralaterally</u> below the level of the lesion :

Pain and temperature sensations are lost, Why ?

due to cut of lateral spinothalamic tract (which crossed to the opposite side).

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Boy's slide

Tetanus

- This is a medical condition characterized by a prolonged contraction of skeletal muscle fibers.
- The primary symptoms are caused by a neurotoxin produced by the Gram-positive anaerobic bacterium Clostridium Tetani .
- Infection generally occurs through wound contamination
- As the infection progresses, the patient develops spastic muscle spasms in the jaw (thus the name "lockjaw") , face (Sardonic Smile, Risus Sardonicus) and elsewhere in the body (e.g. opisthotonus)

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B-Parkinsonism rigidity is of two types:-

-Cog-wheel rigidity

tension in a muscle which gives way in little jerks when the muscle is passively stretched.

-Lead-pipe rigidity

stiffness and inflexibility that remains uniform throughout the range of passive movement.

C- Decerebrate & decorticate rigidity

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SUMMARY

•Spasticity =hypertonia = rigidity: feature of altered muscle performance
•occurring in disorders of the CNS which give rise to the (UMNS).
•Spasticity characterised by: increase in tonic static stretch reflexes and Exaggerated tendon jerks.
•**Features of UMN Syndrome**
•(1) Weakness and decreased muscle control,(2) No remarkable muscle wasting , but disuse atrophy
•(3) Spasticity, 4) Clonus Repetitive jerky motions (clonus), 5) Exaggerated tendon jerks
•(6) Extensor plantar reflex, (7) Absent abdominal reflexes
•

Causes of spasticity:-

A-(UMNS) syndrome , B-Parkinsonism C- Decerebrate& decorticate
•A-(UMNS) syndrome include
•• **(1) Cerebral palsy:** Caused by brain damage due to lack of oxygen.
• **(2) Stroke:** Both Haemorrhagic and Ischaemicstroke cause death of brain tissues results in
• paralysis in the opposite half of the body
A lesion in Corona Radiata on one side ➔Monoplegia in a contralateral limb
A lesion in the Internal Capsule on one side➔ Hemiplegia or Hemiparesis on the contralateral side
• **(3) Multiple Sclerosis**: - is an auto-immune demyelinating disease
• **(4) Acqiured brain injury** (trauma , etc)

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SUMMARY

(5) Spinal cord injury

-Complete transection of spinal cord

If the transection is in the upper cervical region ➔ immediate death

In the lower cervical region below the 5th ➔ quadriplegia

In the lower down in the thoracic region ➔ paraplegia

stages follow spinal cord transaction:

A/ Spinal shock (2-6 weeks):

anaesthesia, paralysis, loss of muscle tone and muscle pump, loss of tendon reflexes, loss of vasomotor tone and Retention with overflow of the urinary bladder

B/ Recovery of reflex activity

Gradual rise of arterial blood pressure, Return of spinal reflexes such as Flexor tendon reflexes, Babiniski sign and Flexor spastic tone & paraplegia in flexion. Recovery of visceral reflexes, Mass reflex.

C/ Paraplegia in extension:

Extensor reflexes become exaggerated

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

-On the same side at the level of lesion

-Ipsilaterally below the level of the lesion :

-Contralaterally below the level of the lesion

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QUESTIONS

Q1/ all of the following are Features of UMN Syndrome except :

a) Exaggerated tendon jerks
b) Spasticity
c) abdominal reflexes are remarkably found
d) Babinski sign

Q2/ transection of one of the following area will cause quadriplegia ?

a)thoracic region
b)lumbosacral region
c)upper cervical region
d)lower cervical region below the 5th

Q3/ Mass reflex appears in which one of the following stages:

a) Spinal shock
b)Stage of return of reflex activity
c)Stage of extensor paraplegia
d) Non of the above

Q4/ Pain and temperature are lost in case of Brown-Sequard syndrome :

a)On the same side at the level of lesion
b) Ipsilaterally below the level of the lesion :
c) Contralaterally below the level of the lesion

1)C
2)D
3)B
4)C

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THE END

If there are any Problems or Suggestions,
Feel free to contact:

Physiology Team Leaders

Mohammed Jameel & Shaimaa Al-Refaie

432100187@student.ksu.edu.sa
432200643@student.ksu.edu.sa

THANK YOU