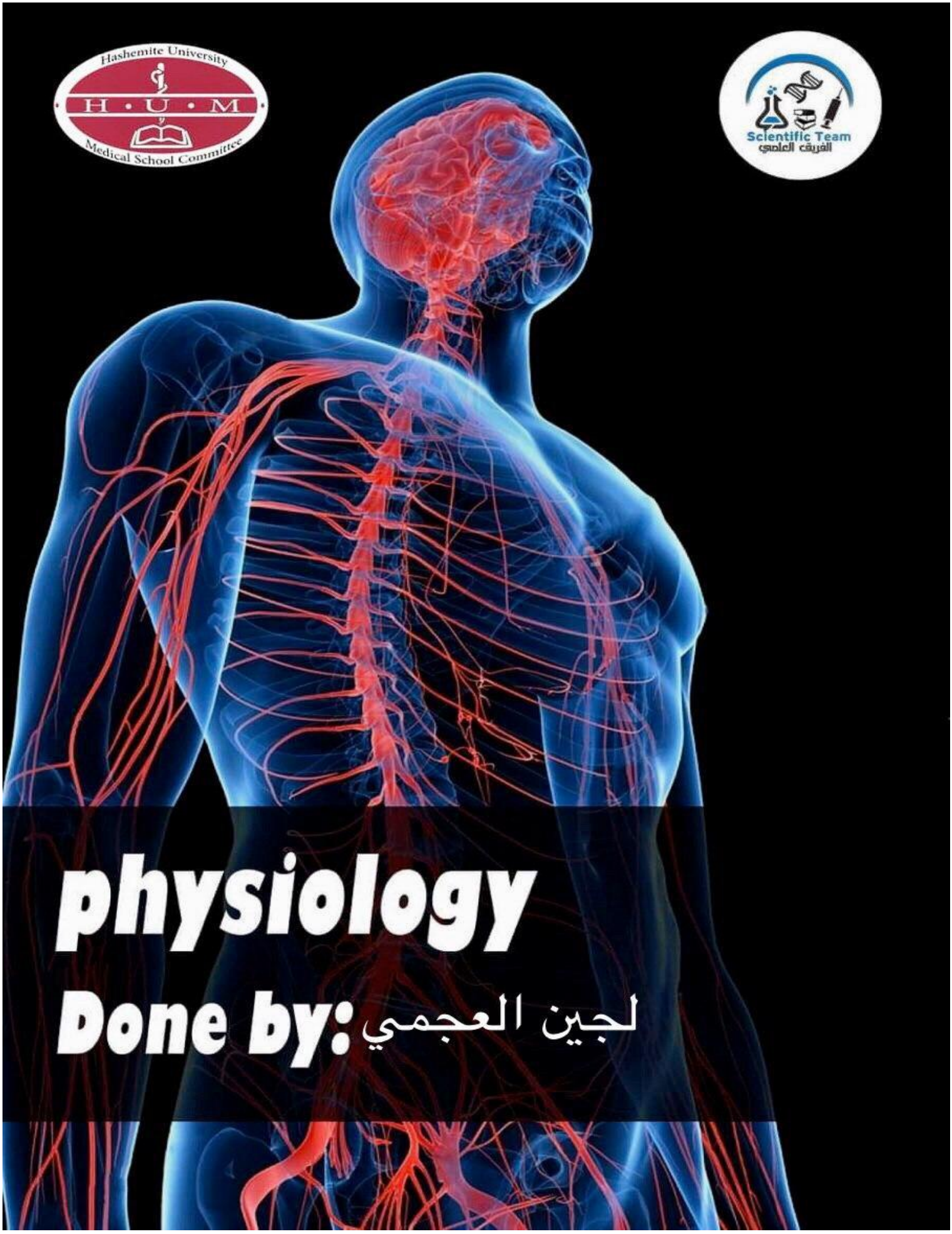

Hashemite University
H · U · M
Medical School Committee
Scientific Team
الفريق العلمي
physiology
Done by: لجين العجمي

بسم الله الرحمن الرحيم

(وَفِي السَّمَاءِ رِزْقُكُمْ وَمَا تُوعَدُونَ)

"اللهم ارزقنا بركة في الوقت والجهد والعمر وارزقنا سعه الصدر وابعد عنا الضيق ويسر لنا امورنا يارب" ♡

بدايه هالمحاضره عباره عن تكمله للفروقات ما بين ال lesions of UMN & LMN وتأثير كل وحده عالجسم

The differences in clinical manifestations of upper motor neuron lesions (UMNL) & Lower motor neuron lesions (LMNL):

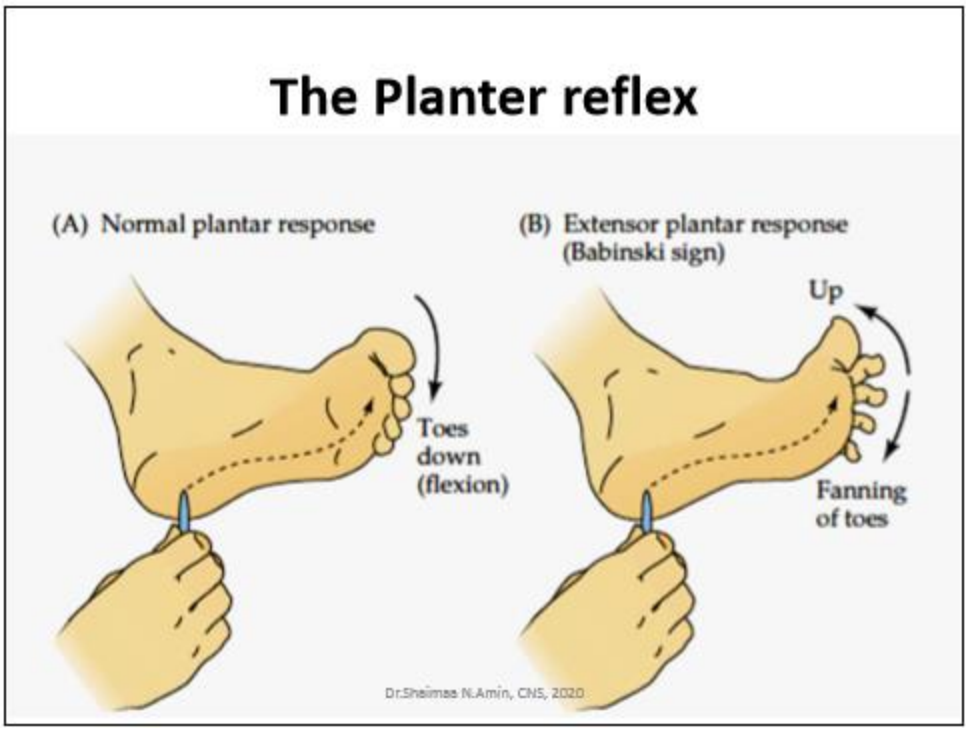
A) Loss of superficial reflexes IN UMNL & LMNL will cause the following manifestations:

IN UMNL:

1) The loss of superficial reflexes occurs on the affected side, due to loss of supra-spinal facilitation

*Note that in UMNL the effect of the loss of superficial reflexes is more generalized & affects all segments of affected side, while in LMNL its more localized

2) Positive Babinski sign, which is a modified form of planter reflex, the planter reflex will become extensor, look at the picture below to understand better. (normally plantar reflex causes flexion of toes, but in UMNL it will cause extension)



IN LMNL:

1) Seen in the affected segments only, so its more localized

2) In UMNL the plantar reflex is always modified to become extensor (positive Babinski sign), while in LMNL **there are two scenarios:**

a) if the lesion happens in the center of plantar reflex, the plantar reflex will disappear as the reflex's circuit has been disrupted

b) if the lesion happens away from the center of plantar reflex, the reflex will remain & won't disappear because there was no disruption of its circuit

So we conclude that LMNL is more localized depending on which segment is affected…..

لأن في حاله الLMNL :

ال lesion لو ما صارت بال center مرح يأثر عال plantar reflex فعشان هيك بنتعبره more localized

How can we perform the planter reflex test?

Testing plantar reflex is done by bringing a blunt object & stroking it on the sole of the foot (lateral edge of the foot) as we can see in the picture above

When you stroke the sole of the foot with a blunt object the response will be:

1) In NORMAL people the toes should flex (bend down)and that is known as plantar reflex

2) But if you stroke the sole of the foot and the toes extend up that could be **either because:**

a) the patient has positive Babinski sign because of neurological lesion

b) the patient is an infant(: and this is a physiological condition & not pathological

Babinski sign isn't always pathological, there are some physiological variants like:

Infants, patients under effect of anesthesia or when a person is in deep sleep

Note that plantar reflex appears only a year after birth, so in infants extensor plantar reflex is normal

يعني الانسان اول ما ينولد بكون عنده positive Babinski sign بعد سنه من الولاده رح يختفي ويجي مكانه plantar reflex واللي هو الطبيعي...

What is the reason for disappearance of physiological Babinski sign a year after birth?

Pyramidal tracts during infancy period are not well myelinated so they can't do proper inhibition of plantar extension reflex which is responsible for appearance of physiological Babinski sign in infancy

A year after birth these pyramidal tracts will be well myelinated and thus inhibition of Babinski sign will occur & planter flexion reflex takes over.

And based on that, the reappearance of Babinski sign in adults or even babies above one year old means the inhibitor of this sign was disrupted causing it to appear again, this happens in the 3 cases we have previously mentioned:

1) in UMNL 2) Deep sleep 3) Anesthesia

We refer to the reappearance of Babinski sign as ''release phenomenon''

Because the inhibited area was "released" or "freed" from its inhibitor which is the pyramidal tract

يعني الطفل بس ينولد ال pyramidal tract اصلا ما بكون شغال لانه لسا not well myelinated

وعشان هيك مرح يقدر يعمل inhibition of Babinski sign or plantar extension reflex

بعدها بسنه رح يصير في well myelination ورح يشتغل عادي ال pyramidal tract ومن وظائفه انه يمنع ظهور Babinski sign و يستبدلها ب normal plantar reflex

في حال صار في خلل في وظيفه ال pyramidal tract رح ينشال ال inhibition effect ورح تظهر ال Babinski sign وهالشي بيصير ب3 حالات اللي ذكرناهم فوق: التخدير , النوم العميق و الUMNL

B) Muscle wasting:

Muscle wasting (or muscle atrophy) is seen in both UMNL & LMNL but the times of appearance differ in both lesions

IN LMNL:

Muscle wasting & atrophy happens early & it will be markedly severe because:

a) The muscle is completely paralyzed so there's no contractions or reflexes as the reflex arc has been disrupted, so the muscle atrophies faster due to inability to contract voluntarily or even reflexly

b) Also because the final common pathway has been damaged.

Recall that final common pathway is responsible for receiving signals for reflexes or volunteer movements & sends them to skeletal muscles, in LMNL this pathway is damaged so both types of movement are absent

وكونه العضله انشلت ومش قادره تتحرك طبيعي يصيرلها ضمور وتموت الخلايا

Also, in LMNL the muscles suffer from degeneration reaction which is a condition caused by a change in the electrical **response** of the muscles due to muscle denervation.

Ya3ni the muscle lost its source of innervation & thus when we perform electrical stimulation test on the muscle it won't respond normally due to pathological electrical changes (degeneration reaction)

IN UMNL:

Voluntary movement is lost, but reflexes are still present

وعشان هيك بكون ضمور العضلات اخف وبيصير بعد فتره اطول بكثير من الLMNL لأن العضله لسا قادره انها تتحرك بسبب استمرار ال stretch reflex movementبس طبعا

المريض مش كل شوي رح يقدر يعمل stretch reflex ف اخرتها العضله رح يصيرلها ضمور بسبب disuse atrophy

Neurologists use electrical stimulation of skeletal muscles to test their response, we have two types of electrical stimuli…

(first we will discuss their effect on NORMAL muscles and then move into discussing the effect on injured muscles)

a) Faradic stimulus: a normal muscle will present with tetanic response (tetany), because under the influence of this stimulus there will be summation of twitches & continuous contractions

b) Galvanic stimulus: a normal muscle will respond by contraction at the beginning of the stimulus (when the circuit opens) & the end of the stimulus when its removed (when the circuit closes)

so the muscle contracts twice, once the electrical stimulus touches it & once its removed, unlike faradic which produces continuous contractions….

Once the muscle goes through galvanic stimulation, there will be 4 contraction states:

يعني بس نعرض العضله لل galvanic stimulation رح ينقسم الانقباض ل4 حالات..
طيب ليش اصلا بيصير في 4؟

Remember that when testing the patient, we use TWO types of electrodes….

> Anodal electrode & cathodal electrode, the contraction state of the muscle differs depending on which electrode we use….

> Also we said that the muscle contracts when the circuit opens & closes (the anodal electrode will produce 2 contractions, first contraction when circuit opens & second when the circuit closes, same

applies to cathodal electrode, so we have 4 various states of contraction of muscles)

The 4 states of contractions predicted to happen:

1) If we use anodal electrode, the first contraction happens when the circuit opens & its called (anodal opening contraction or AOP)

2) The second anodal electrode contraction happens when the circuit of closes, the contraction state will be (anodal closing contraction or ACC)

3) If we use cathodal electrode, the first contraction state will be when the circuit opens & its called (cathodal opening contraction COC)

4) if the circuit closes it will be cathodal closing contraction (CCC)

As we can see, galvanic stimulation gives 4 states of contraction while faradic stimulation only produces one state of contraction which is tetany

All the effects of electrical stimulation we previously mentioned were for NORMAL muscles, now how will these electrical stimulations affect the muscle if it was injured?

In case of LMNL:

If the muscle was suffering from LMN lesion & was subjected to Faradic stimulation there will be no response

If it was subjected to Galvanic stimulation, the strength of 4 states of contraction will differ

يعني بالعضله الطبيعيه لو عرضتها ل galvanic stimulation قوه انقباض العضله بتكون ضمن range معين وطبيعي, اما في حاله ال LMN لو تعرضت العضله ل Galvanic stimulation رح يصير في خلل في ترتيب strength of contraction

مثال توضيحي من الدكتوره: حكينا ال galvanic stimulation بعطي 4 حالات, لو فرضا بالوضع الطبيعي ال AOP تنصف كأقوى انقباض بعدين ACC بعدين COCبعدها اضعف شي CCC ... في حال ال LMN رح يختلف هالترتيب ورح يصير في فرق بقوه الانقباض وخلل ب normal range of the strength of muscle contraction

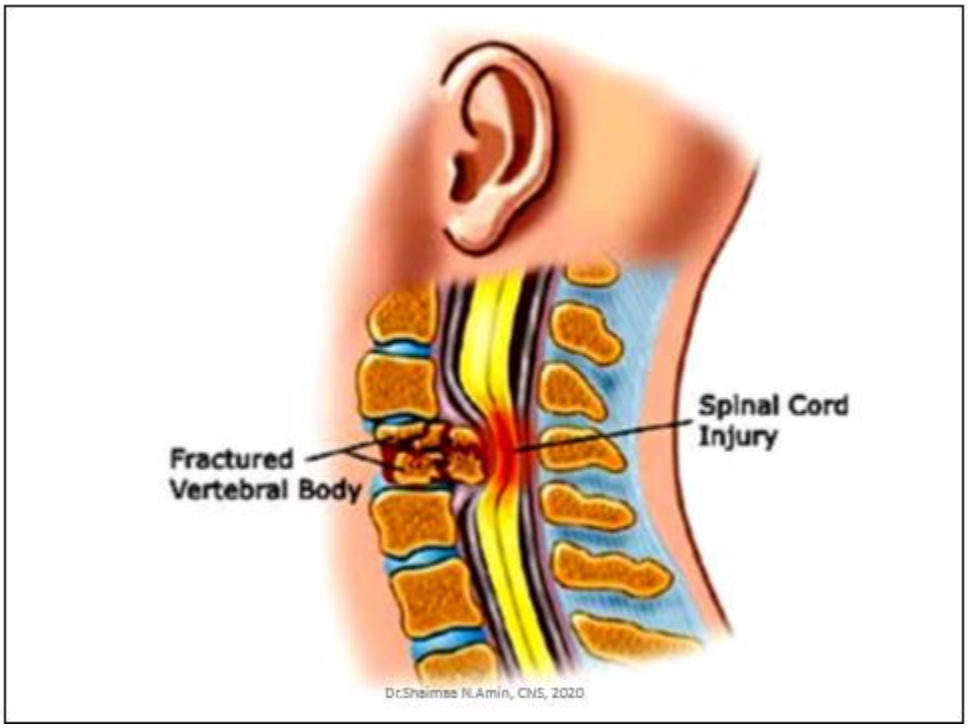
The excitability of the muscle will decrease & chronaxies will increase

All these electrical changes of muscles in LMN are known as ''reaction of degeneration''

While in changes that occur in UMN lesions are mostly vascular with hemorrhages in internal capsule

Note: you are not required to know any details about chronaxies, that's why I didn't mention any extra information regarding it ^-^ so just memorize the sentence above as it is :3

Trauma of spinal cord: 1)hemisection or 2)complete SC transection):

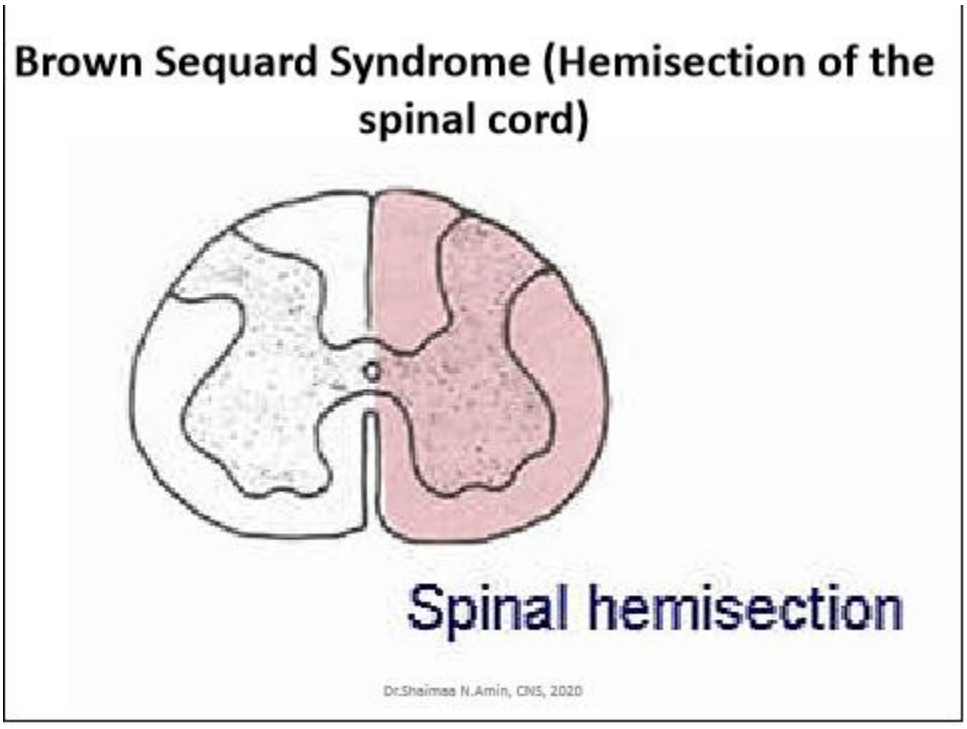


As you can see in the picture above, the spinal cord
is formed of very soft structures surrounded by meninges & vertebra

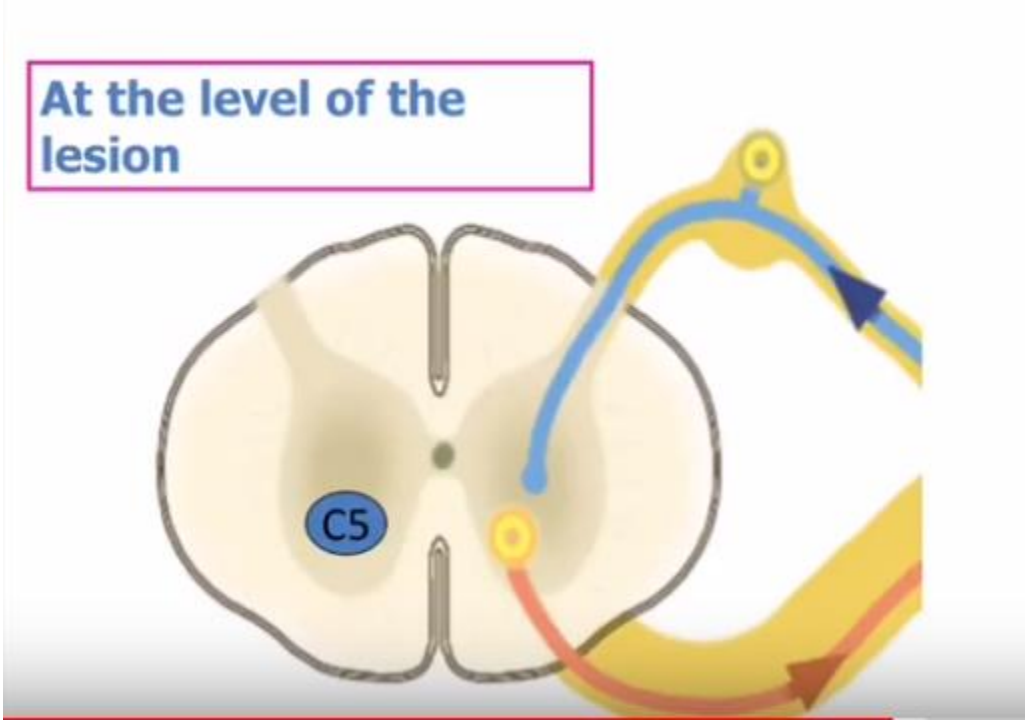
If the vertebral body was fractured, the edges of it will turn into sharp structure which will injure the spinal cord

The causes of spinal cord injury vary from minor to major with injury of the entire spinal cord

Hemisection of spinal cord (Brown-Séquard syndrome- the second name is important for the exam)



This lesion will produce different manifestations according to the level of the injury… (In this lesion only part of spinal cord is injured, not all of the spinal cord) … we will discuss manifestations in two levels:



Manifestations at the level of the lesion:

<u>In normal people:</u>

The nerve colored in orange is exiting through the anterior root to join spinal nerve & finally reaches certain skeletal muscle to innervate it

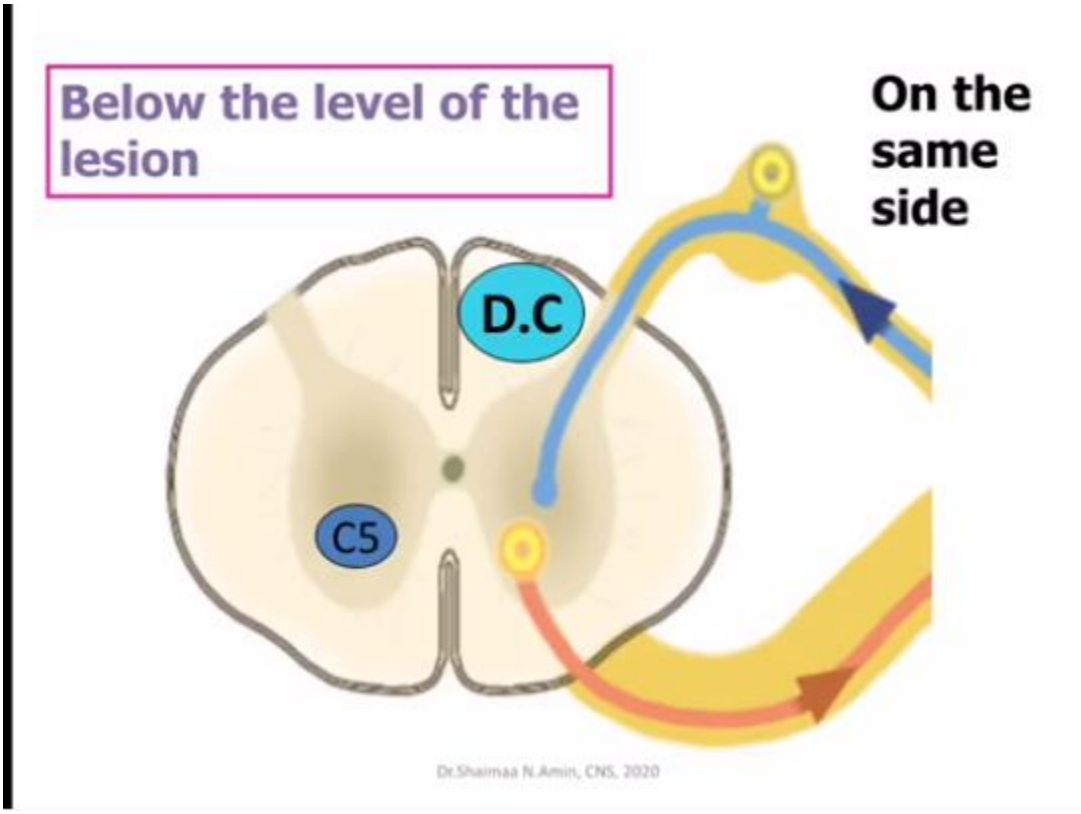
<u>In case of Brown Sequard syndrome:</u>

The nerve will be torn apart & a lower motor neuron lesion at the same level will be produced

يعني لو صار هذا ال syndromeعاليمين, الشلل او الاعراض تبعت ال LMNL رح تطلع على جهه اليمين

***so the injury happens on one side & LMNL manifestations appear on the same side of injury

Manifestations below the level of lesion:



Below the level of the lesion:

There will be sensory & motor manifestations. And it is a case of UMNL

Recall that in normal people the crossing (or decussation) of fibers carrying sensations of dorsal column happens in the medulla, so the sensations decussate to the opposite side in the medulla

Which means below the level of lesion there will be loss of dorsal column sensations on the same side

عشان تفهمو احسن... بالوضع الطبيعي ال fibers اللي بتنقل dorsal column
sensations رح تعبر للجهه المقابله(decussate)بس توصل لل medulla .. بس هون
ما لحقت توصل اصلا يعني لسا موجوده بال spinal cord ما لحقت تتخطاه.. فعشان هيك

الاعراض رح تطلع بنفس الجهه.. بمعنى لو صارت الاصابه بجهه اليمين رح تفقد الاحاسيس لجهه اليمين...

While spinothalamic sensations will be lost from the opposite side

حكينا ال fibers اللي بتنقل ال spinothalamic sensations رح تعبر للجهه المقابله (decussation) بس توصل لل spinal cord ... فمعناته خلص صارو متجاوزين جهه اليمين ونقلو ال sensations لجهه اليسار.. فعشان هيك رح يصير في loss of sensations of the opposite side

Spinothalamic sensations like temperature & pain will be lost from the opposite side (spastic paralysis)

COMPLETE TRANSACTION OF SPINAL CORD:

The manifestations depend on which level of spinal cord has been effected, could be monoplegia, paraplegia, tetraplegia or quadriplegia

If the lesion happens above the origin of phrenic nerve (above C3) then its fatal as respiratory muscles are innervated by phrenic nerve so loss of this nerve causes respiratory destruction … prisoners which are set for execution are killed by this method

Stages of spinal cord lesion:

1) stage of spinal shock

2) stage of recovery of reflexes (not all movements are recovered, just reflexes, voluntary movements are lost)

3) failure of reflex activity (rare, often fatal)

All patients with spinal injuries go through the first 2 stages, and depending on how well the patient obeys the precautions will decide if his case will progress to failure of reflex activity

We will discuss the stages in more details…

A) STAGE OF SPINAL SHOCK:

Happens because of sudden withdrawal of the effect of supra-spinal tract

Supra-spinal tract has some effects on spinal cord segments

SO when any lesion to spinal cord happens this will detach the spinal cord segments from the supra-spinal tract and this sudden detachment will cause spinal shock stage

Duration of spinal shock varies depending on the agree of encephalization…. Humans have high degree of encephalization

Encephalization is a concept that implies an increase in brain or neocortex size relative to body size, size of lower brain areas, and/or evolutionary time.

يعني كل ما كان ال brain cortex متطور اكثر وله تأثير اكبر كل ما كان اسوء

يعني مثلا الضفدع مخه صغير وال cortex مش كثير مرتبط بال spinal cord و ما عنده any degree of encephalization فلو فصلت ال spinal cord segments عن supra-spinal tract مرح يحس ابدا وبدقيقه يرجع طبيعي

بينما الانسان مخه معقد اكثر ومرتبط بكثير شغلات بل spinal cord فعشان هيك فتره الشفاء بتكون اطول بكثير من الضفدع

Usually period of recovery occupies 2-6 weeks in human (human heal faster >then dogs>then cats>then rats>then frogs)

*في حال حبيت تتخصص بيطري احفظهم-.-

* الدكتوره حكت مثال توضيحي عشان تفهمو موضوع ال encephalization احسن..
لنفرض في شخصين راحو يكملو دراستهم برا بعيد عن اهلهم... واحد منهم متعلق ب اهله

بشكل مش طبيعي ومستحيل ياخذ اي قرار من غير لا يستشيرهم.. والثاني ولا عارف شي عن اهله فمش فارق معه لو بعد عنهم او لا..

نفس الشي الانسان بحتاج ال spinal cord reflexes & brain cortex بكل خطوه بحياته عشان يعيش ولو ال spinal cord انفصل عن ال brain cortex رح تصير حياه الانسان صعبه... اما الضفدع مش فارقه عنده لو مخه انفصل عن ال spinal cord لانه اصلا ما بحتاجه... بختصار وجود مخ الضفدع وعدمه واحد (سبحان من خلق بعض البشر زي الضفادع :))

The stage of spinal shock is characterized by the following manifestations:

1) Loss of all reflexes (superficial, deep & visceral) … but note that its not permanent

2) Permanent loss of all sensations

3) Permanent loss of all voluntary movements

الشي الوحيد اللي ممكن يرجع هو reflexes اما ال sensations & voluntary movements انفقدت تماما

Micturition reflex: … How is it going to be affected in this stage?

We took in GIT module that this reflex is a spinal reflex which depends on stretch receptors present in the bladder, it is also controlled by supra-spinal tracts

Supra-spinal tracts control micturition reflex through:

> Inhibiting it when its not the right time for it, like when you're in the middle of an exam, you cant just answer mother nature calls in exam hall

> Facilitating this reflex when it's the right time for it (like when taking urine sample)

In the beginning of the “spinal shock” stage, this micturition reflex will be inhibited.

But few days later, the muscle tone of internal urethral sphincter will recover quickly, the reason is unknown الحمد الله حمدا كثيرا مباركا

On the other hand, the external urethral sphincter will not recover at all because it is under voluntary control which has been permanently lost as we previously mentioned

ال external sphincter هي المسؤوله عن عمليه ال urination وانت اللي بتتحكم فيها اما ال internal sphincter بتكونinvoluntary reflex

وحكينا بل spinal shock stage ال reflex رح يظل بس ال voluntary movement رح تفقد

So at this stage of spinal injury the patient will have retention of urine with overflow

بكون تجمع البول عنده بس ما بقدر انه يتخلص منه لان ال external urethral sphincter تعرضت للشلل

This patient will continue to retain urine in the bladder until the amount of urine retained pressures the bladder to the point where it overcomes the tone of internal urethral sphincter & ability of to maintain urine so dribbling of urine happens

البول بتجمع بكميه كبيره جوا ال bladder ورح يصير عليها ضغط كبير لدرجه ان ال internal urethral sphincter مرح تقدر تظل مسكره ال bladder فرح يصير dribbling of urine

B) STAGE OF RECOVERY OF REFLEXES:

Two theories explain why this stage of recovery happens…

a) denervation hypersensitivity (most acceptable):

If we de-innervate a certain structure for some time, the receptors of this structure will be upregulated & become sensitive to the least amount of neurotransmitters

يعني لو قطعنا النيرف سبلاي من عضله لفتره طويله ال receptors اللي موجودين عليها رح يزيد عددهم (upregulation) وبالتعالي تزيد ال sensivity of response وبيصير يستجيب لأقل كميه من الneurotransmitters

b) regenerative sprouting:

The axons of the nerves may sprout & regenerate

The characteristics of this stage:

1) The spinal reflexes will return with positive Babinski sign

2)Return of visceral reflexes

Muscle tone recovers slowly & increases, it will recover first in flexor muscles before extensor muscles

In case the patient was suffering from paraplegia he will develop paraplegia inflexion, ya3ni muscle tone increases in flexor muscles so flexor muscles will always be bended

مثلا الرجل تكون دايما مثنيه

3) Mass reflex:

A minor painful stimulus to the skin, like pinching the patient's skin will stimulate this reflex which sends irradiations & nerve impulses to stimulate spinal tracts that cause:

1) Flexion withdrawal of the limb

يعني لو قرصته من ال limb رح يعمل flexion & withdrawal of his leg لا اراديا
مع انه فقد حاسه الالم بسبب ال mass reflex

2) Evacuation of rectum & bladder.

If you pinch the patient, his body sends signals to many spinal reflexes since it's a mass reflex, one of these reflexes is micturition reflex causing evacuation of rectum & bladder

3) Sweating of the skin due to autonomic disturbance because of the irradiations

4) Rise or decrease in blood pressure due to autonomic disturbances

The most important thing doctors focus on is monitoring micturition reflex

Because retention of urine in bladder & retention of stool in rectum has many complications on the patient's health so doctors provide patients with catheters to prevent retention of urine & we use enema to empty the rectum from stool

If we don't use the catheter as soon as possible, the patient will experience a condition called ''atonic bladder''

During this period of recovery, doctors ask patients to pinch themselves & stimulate mass reflex by themselves so they can empty their bladder from urine, this helps in avoiding the occurrence of atonic bladder

طبعا تذكرو ان المريض ما بقدر يعمل voluntary urination لازم من خلال reflex عشان هيك لازم يقرص نفسه عشان يحفز حدوث الmass reflex وبنفس الوقت بحمي حاله من atonic bladder development

We call this ‘’intentional mass reflex’’ where the patient intentionally pinches himself & stimulates urination & defecation reflexly

Advanced stage of recovery of reflexes:

1) We said the muscle tone increases in flexors initially then at more advanced stages of recovery it will increase in extensors, the muscle tone will keep increasing in extensors & becomes higher than in flexors & we refer to this as “paraplegia in extension”

2) Positive supporting reflex becomes well developed & the patient can stand without help, but he can never walk (remember only reflexes recover, voluntary movements don’t recover)

<u>Doctors should pay so much attention to:</u>

1) Nutritional state of the patient, especially protein diet because it may help in neuronal regeneration

2) Position of patient! Immobilization is associated with negative nitrogen balance which causes development of peptic bed sores

بالوضع الطبيعي لما تقعد لفتره طويله رح تبدا تحس بألم لأن قل تدفق الدم للانسجه فرح يصير في ischemia واللي رح يحفز ال pain sensations فرح تتحرك عشان تخفف الالم فلما تتحرك رح يصير في reflow of blood & ischemia vanishes ويختفي الالم... الناس اللي بيصير عندهم شلل بل spinal cord مرح يحسو ولا يتحركو فرح تطلع عندهم ulcers بسبب عدم الحركه وينتج ischemia وممكن تكون عميقه جدا وتوصل العظم

Doctors monitor development of peptic bed ulcers by re-positioning the patient in bed to help reflow of blood & prevent ischemia & ulcer development

3) Doctors must also ensure proper evacuation of rectum & bladder, if the urine & stool are retained for long time this will cause development of infection & patients may die from toxemia or septicemia

Most patients die from septicemia or toxemia from urine retention or from peptic bed sores

"Life of a medical student is tough, my darling, but so are you"

Good luck! ^-^