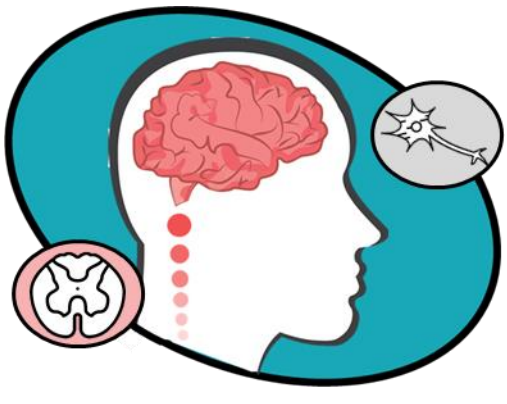


CNS

Physiology

Sheet Slide

Number

11

Done by:

مها أبو عجيمية

Corrected by:

معاذ درويش

Doctor:

لؤي

CNS – Physiology

- **The doctor hasn't sent the slides yet, please refer to them when they are uploaded**
- **You can skip the extras and sorry for any mistake.**
- **Section1 Record:** **https://onedrive.live.com/?authkey=%21AA0pK3GOrL7yTak&cid=E95ED7C5E3BDA749&id=E95ED7C5E3BDA749%21165&parId=E95ED7C5E3BDA749%21112&o=OneUp**

1. **NREM**: In the non-REM sleep, the cortex becomes **less active**, it is turned off, this **decreases** vital signs, breathing and heart rate.

2. **REM**: Deep sleep or REMs (stands for Rapid Eye Movements sleep) is the **most important** stage, in REM sleep, the demand of oxygen and blood supply **increases** which leads to increasing the vital signs, heat rate and breathing, this happens to supply the cortex.

Sleep Disorders

1) **Insomnia**: unable to sleep or inadequate sleeping hours, but more specifically poor sleeping quality, like persistent problems in falling asleep, staying asleep, or awakening too early, one of the most popular cases of insomnia is **Sleep Apnea,** it is defined as repeated interruption of breathing during sleep.
2) **Parasomnia**: abnormal movements, behaviors, perception or emotions during sleep or as a result of interrupted sleep, people with parasomnia, might unintentionally harm themselves because of these movements, or wake up crying real tears, or simply feeling profoundly sad and not able to decide whether what they have seen, was in real life or during sleep.
3) **Narcolepsy**: excessive daytime sleepiness, it is characterized by sudden and irresistible onsets of sleep during normal waking hours, too much sleep or sleeping in an inappropriate times, conditions or situations!
4) **Nightmares**: anxiety-arousing dreams occurring near the end of sleep, during REM sleep.
5) **Night Terrors**: abrupt/sudden **awakenings** from NREM sleep accompanied by intense physiological arousal and feelings of panic.
6) **Somnambulism**…sleepwalking
   - 40% of children will have an episode, peaking at between 11-12 years of age.
   - It can be induced if arouse children during NREM
   - It is associated with complete amnesia.

- It occurs within 2 hours of falling asleep
- EEG, reveals both waking and sleep signals.
- It is considered **benign**.

INSOMNIA

If there was **not enough oxyge**n, there would be a risk of cortex's damage, this is why **REM is suppressed (Remember that in REM stage the demand of oxygen increases)**, returning back to stage 2 and 3, in this case, we want to help the patient breathe by positioning the patient in bed and changing his face position.

If it was a **chronic** breathing problem, this would happen 2-4 times every night, so no matter how **many hours** the patient slept, he would **not** get **enough REM** because it was interrupted, this is why he would feel exhausted and suffer from headache and chronic fatigue after waking up, he might develop cognitive problems and not be able to concentrate. (one similar example that may last about a week, is when you've got a cold.)

PARASOMNIA

In both **wakefulness** or in **REM sleep**, the cortex should be **active** (cortex arousal) to process the information, this can be done by activating **RAS system** (reticular activating system) and increasing Acetylcholine (Ach), Norepinephrine (NE), Serotonin (5-HT), Histamine (HA) and Dopamine (DA), this will eventually activate the cortex.

Their functions during REM are similar to their functions during the day:

a- Serotonin: although it is complex, it modulates thinking and processing by playing a role in many areas of the brain.
b- Norepinephrine: **selective** attention (playing a role in directing prefrontal cortex at focusing on a certain task).
c- Acetylcholine: **sustained** attention.

To **sleep**, (especially stages 2, 3 and 4 of NERM) the **RAS** and **cortex** should be turned **off** by increasing **GABA**.

(RECORD 7:07-8:24), In REM episodes, there will be regulation of prefrontal cortex by re-arranging (certain cortical circuits, probably) and activating certain connections and networks (via certain pathways, probably), and make them more active than others. That's why your concentration would be on nothing more than CNS such as those

implicated in memory, (there is nothing more important to be done in the meantime), this is why your prefrontal cortex needs:

- NE to select what to focus on (e.g: memory retention)
- ACh is needed to re-arrange CNS information for 15 mins.

During REM sleep, as we previously said, RAS should be activated, ACh, NE and serotonin play important roles in REM sleep:

- activation in REM sleep is sustained mainly **ACh**, which remains highly active during **most** stages of REM,
- **NE** is also needed to enable the prefrontal cortex to select, this is why NE levels increase at **the beginning and end of REM**,
- **Serotonin** also increases, but its action is complex.

If there were **disturbances** in these **neurotransmitters**, or if they were not working right, the person would have **interrupted** initiation and termination of **REM**, it might be longer or shorter than normal even if the person gets enough sleeping hours, the patient will have **symptoms** related to **parasomnia**.

SLEEP DISORDERS AND HEALTH

The neurotransmitter levels might be altered because of either having a health problem OR taking certain medications or drugs

1. ***Health Problems***:
    - patients with ***ADHD,*** will have ***norepinephrine deficiency,*** *this is why they will have sleep problems.*
    - *patients with **Anxiety** will have **increased norepinephrine** levels, which leads to parasomnia.*
    - *those with **depression** will have **less**, **interrupted** or **un-rhythmic** sleep.*
2. ***Drugs**, In addition to health problems, any treatment that will alter these neurotransmitters will cause sleep disorders*
    - one of **β-blockers'** side effects is **nightmares** that are associated with REM sleep, because these blockers alter norepinephrine levels.
    - **Drugs** used in **ADHD,** we give them noradrenaline treating drugs which can cause **parasomnia.** (extra: examples of these drugs are Amphetamine or Desipramine)
    - When using **SSRIs** (selective serotonin reuptake inhibitors), SSRIs are antidepressants that can cause:

- **insomnia**, but the problem is that depressed people will suffer from severe insomnia, because they already have insomnia (and you are increasing serotonin levels), meaning that you are trying to fix their mood but sleep disturbances will turn up.
- **parasomnia** Also could occur as a side effect for SSRI

- **Anti-cholinergic** medications for **Parkinson's** or (cholinesterase inhibitors) for **Alzheimer's**, will also affect the sleep, leads to a group of many symptoms, parasomnia is one of them.

SLEEP TALKING OR SLEEP WALKING - SOMNAMBULISM

At stage 3 (NREM), the person might move and change his position, if the motor remained active, this would lead to sleep talking or walking

It is normal during l**ate childhood** or **early teenage**, BUT might persist in late adulthood, still it doesn't need treatment unless it was life-threatening (e.g. cooking, driving or other risky actions during sleep), the person is usually unaware of that unless someone's watching him during sleep

(a typical example, an 8-year old boy has slept in his bed, but found himself in his parents' room in the morning)

NIGHTMARES

Dreaming takes place during **REM**. if you remember, REM give us a type of dream which is not realistic. We don't usually remember all our dreams but nightmares are usually remembered, the doctor has defined the nightmare as an intense dream, in which the body and brain will feel afraid, so the brain will **end the REM**, the person will **wake up** suddenly and he will be able to remember what he was dreaming before few seconds. If the dream's intensity was exceeding the limit, it could be called PARASOMNIA.

NIGHT TERROR (or Sleep Paralysis)

Similar to a nightmare where the dream is more intense, as a result, the brain will act rapidly, by waking the person up **without** the presence **cortical output**, the person is awake, conscious, and the effect of this bad dream hasn't gone yet but the person is unable to move any part of his body because the muscles are **completely relaxed**, this is so terrifying (could be considered a subtype of parasomnia).

(e.g. something is following you but you feel that you are paralyzed and unable to escape.)

SOMATOSENSATION

Most important sensations come from **skin** and **muscles**, but to a lesser extent, the sensations that come from visceral organs.

There are **different** types of **receptors** (mainly mechanoreceptors) that receive different sensations, so any mechanical change that stimulates the muscle or skin will end up in the CNS (you know! to be sensed.)

Depends on the **processing**, one can have two types of sensation or the perception of this sensation will be amplified.

More pathways mean that there will be **more processing**, there will be more accuracy (acuity) and the brain will be able to feel different types of sensations (e.g. two different sensations). In Other Words, there are many receptors in skin and muscles, and there will be different processing along the pathway, even though **touch** and **pressure** are **mechanical** forces that affect mechano-receptors, but they are sensed **differently**, why? because of different **processing**.

There are also **different mechano-receptors** for the *same* mechanical **force** (e.g.: touch), you can find receptors for Discriminative Touch (two-point discrimination) and other mechanical receptors, that are responsible for Crude Touch (non-discriminative touch), (such as rubbing and itching), in the latter, you feel that there is something touching you, but you are not able to tell what exactly it is, or you simply cannot feel more than one stimulus at different points.

The same receptor can make you feel two different types of sensations, but how? The **same receptor** might have **two fibers**, each fiber is connected to a different pathway, the result will be feeling **two types of sensations**.

Somatosensory Pathways: (the ascending tracts: PCML and ALS.)

Pathway	3-NEURON SYSTEM
PCML	1st order neuron 2nd order neuron decussates
ALS	3rd neuron: thalamus (VPL -> CORTEX)

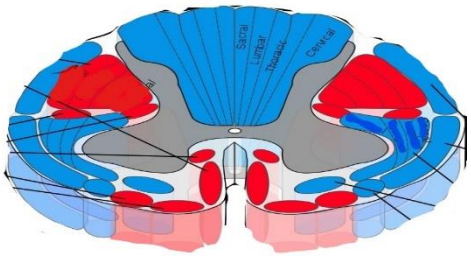
PCML: (posterior column- medial lemniscal system)

- Modalities: two-point discrimination (fine/ discriminative touch), vibration and proprio-sensation (proprioception) the latter means the sensations coming from muscles, including muscle length, muscle tension, joint position and joint movement, PCML enables the brain to do more than one function.
- The Pathway: (go to anatomy lectures to learn it in detail)

The PCML system carries sensory information (for discriminative touch/ proprioception …) from peripheral nervous system, the axons of the **first-order neurons** will enter the spinal cord, the axons will ascend ipsilaterally in **posterior columns** (extra: also known as dorsal columns), then from there, they continue their journey to the lower part of the **brainstem** to synapse with **second-order neurons** in what we call **posterior nuclei** (I think he means gracilis and cuneatus), then after second-order neurons **decussate** (extra: in medulla), they'll ascend contralaterally to terminate on **thalamus** (the thalamic ventral posterolateral nucleus (**VPL**))

(extra: VPL is responsible for the whole body except head&neck) From VPL, the fibers project to the sensory **cortex**, physiologically known as: the primary somatosensory cortex, anatomically: postcentral gyrus, Brodmann: 3.1.2.

PCML will have fibers from all over the body, but the first part of **the spinal cord** will receive fibers from the **lower extremities**, this is why, they would be closer to the midline (more **medially**), then **lateral** to them, we'll find the fibers of the trunk and then the **upper extremities** and face.



(easy way to remember how the posterior/ dorsal column is organized, they are organized as you are, with hands at sides "Arms outside, Legs inside)- a useful extra.

At the level of **brainstem** (medulla), **graci<u>L</u>is** is closest to the midline and it receives input from **<u>L</u>ower** extremities medially and **c<u>U</u>neatus** is more lateral &receives input from **<u>U</u>**pper extremities, at this point, crossing/ decussation occurs, the fibers re-arrange, the upper extremities will become posterior and the lower extremities will become anterior.

from the level of the upper part of brainstem to the level of the thalamus, the fibers keep twisting *counterclockwise*, as a result, they become almost lateral and medial, then they become almost anterior and posterior again and so forth, EVENTUALLY, when they reach the **thalamus**, they will be almost lateral and medial.

At the level of the **cortex**, the area would be represented in the postcentral gyrus, more **medially** we find the **lower** extremities, and more **laterally** the **upper** extremities and face.

Why it is important to know the somatotopic organization at different levels? To be able to localize and identify the consequences of different lesions, tumors …

e.g: if there was a damage in the central area of the postcentral gyrus, the person would lose body (trunk) sensation but the upper and lower extremities would be spared/ intact, and brainstem hemorrhage in one area might lead to sensation loss in the lower part of the body but not the upper, a tumor that compresses from posterior to anterior, will affect one part and the other will not be affected and BLAH BLAH BLAH) – I think there would be more specific and clear examples in the anatomy lectures.

- PCML Functions and Possible Lesions.

A- **Stereognosis** : if a person closed his eyes and was given an object, he would be able to **identify** (its shape, form, consistency..) according to the touch, and that's because the accurate touch he received from PCML. Also, because of receiving information that comes from muscles and joint ,like muscles tone and length, as we as joints position and movement

B- **Graphesthesia** the person can identify any letter or number **drawn** onto his **skin** (at 24:50 without knowing its direction).

C- **Barognosis**: the ability to evaluate the **weight** of the objects and exerting the **proper** amount of **force** (movements) based on that, meaning that the muscle tension, the amount of force needed, the angle of your arm will differ according to different orders/ actions, e.g: taking a pencil from a table does not require as much force as when taking something heavier, and you will automatically know at what height you need to raise your cup of tea &the force needed without spilling it on yourself, everything's calculated! Subhan ALLAH!

A great part of this, occurs mainly in the **cerebellum** where the sensation and movement are coordinated, this is why **abarognosis** is usually associated with **ataxia**; incoordination between movement and sensation, if it was because of cerebellar damage, we'd call it cerebellar ataxia BUT if there was no sensation, sensation would not reach the cerebellum obviously, we'd call it sensory ataxia.
Note: the most important sensations at which the movement will depend are the pressure and touch in fingers and the tension and movement of the arm…

If there was a damage in PCML, the person would lose the previous functions, and would **not** be **able** to depend on **touch purely** to identify the shape, size, weight of objects, this will lead to **a**sterogenosis (is written also sterogenosi**a)** ,**a**graphthesia ,**a**barognosis and sensory ataxia.

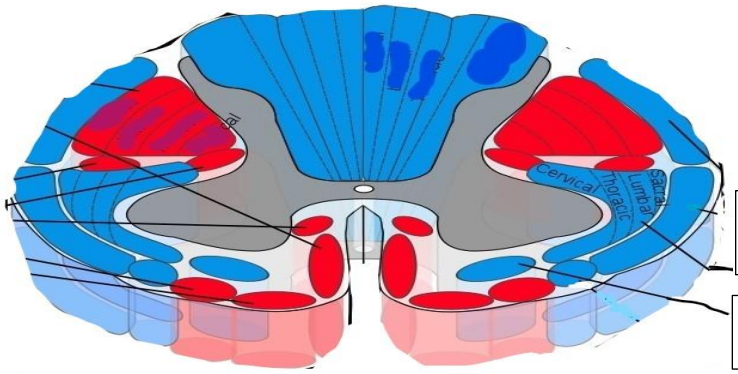
If there was **a complete damage** of PCML pathway (e.g.: there was a complete cut of posterior column at the level of C2 of spinal cord), the patient would lose the previous functions, this is why he would **not** be **able** to identify the objects' shape and their other characteristics but he would be able to know that there is something that touches his skin, but WHY? Because there is **another pathway**, ALS (which is responsible for crude touch).

ALS: (Anterolateral System, aka Spinothalamic)

- Modalities: Crude Touch, (such as rubbing and itching), pain and thermal sensation.
- Pathway:

This system carries pain, temperature, crude touch sensations from periphery, the **first-order neuron** travel from periphery (where we find the receptors) to the spinal cord, it directly synapses with **second-order neuron** in the (extra: ipsilateral) **gray matter** of the spinal cord, then axons of the second-order neuron **decussate** (extra: at anterior white commissure) and coalesce (to form the spinothalamic tracts), and ascends contralaterally. In other words, forming another pathway in the anterior and lateral sides of spinal cord (it is thus called the antero-lateral system), then axons ascend to third-neuron neuron in the **thalamus** (the **VPL** nucleus), then continues its journey to the primary somatosensory **cortex**.

(extra: the **lateral** (spinothalamic tract) is responsible for sending **pain** and **temperature**, the **anterior** (spinothalamic tract) is responsible for **crude** touch and **pressure**, Both PCML and ALS have a role in sensing Pressure)



ALS

Lateral spinothalamic tract

Anterior spinothalamic tract

- Lesions:

If ALS was cut, the person will have the ability of two-point discrimination touch, with good stereognosis, graphesthesia, with good motor, no ataxia and applying the proper force, but he will **lose** pain and thermal sensation.

NOTES:

ALS and PCML, have different pathways at the level of spinal cord and brainstem, but the **same cortical** area.

PCML fibers enter the spinal cord ipsilaterally, at the level of brainstem they will cross/ decussate. On the other hand, **ALS** fibers will directly decussate at the level of spinal cord.

If there was a *damage* at the **right side** of spinal cord, the patient would lose the **PCML** modalities at the **right** side and the **ALS** modalities at the **left** side, this condition is called **Dissociated Sensory** loss.

(at 33:00) If there was damage in the spinal cord at level of T5, the patient would **not lose** PCML **completely**, he would lose PCML below level of lesion (T5), which means he will have ipsilateral loss of proprioception, vibration, 2-point discrimination below T5 AND he would **not lose** ALS **completely** as well, we would think that he would lose ALS at the level of T5 and below on the other side, which means there will be a contralateral loss of pain, temperature, crude touch below the lesion, but it's found that he would lose it at level of T7 and bellow, why?

- Because PCML directly enters, so what is at level T5 stays at level of T5.
- Because ALS enters, then synapses, but while crossing, it doesn't ascend at right angles, meaning that if it entered at the level of T5, it would be found

at higher level, in other words, if ALS entered at level X, it would not be found at the same level in the midline, it would be at level X+1 in the midline, and would be found a higher level when it reaches to the fiber bundles of the other side (at level X+2) , e.g.: if it enters at T5 level, it will be at the level of T4 in the midline, and at level T3 in the fiber bundles??!

I honestly have no idea, why the person would lose sensation at the level of T7, it does not make any sense according to what the doctor has said, but to cut a long story short, he concluded that when a patient has lesions in ALS, **loss of sensation (analgesia) begins 2 segments below the lesion and includes everything below it,** feel free to contact me if you have an explanation, thank you.

Dedicated to Lolo, YQueen, Salsal, Fatoom, Hyundai and Others x'D

"When you want to succeed as bad as you want to breathe then you WILL be successful,"

Don't Let any Pressure Dictate your Options, It's too Early to Quit, Work as Hard as you CAN.