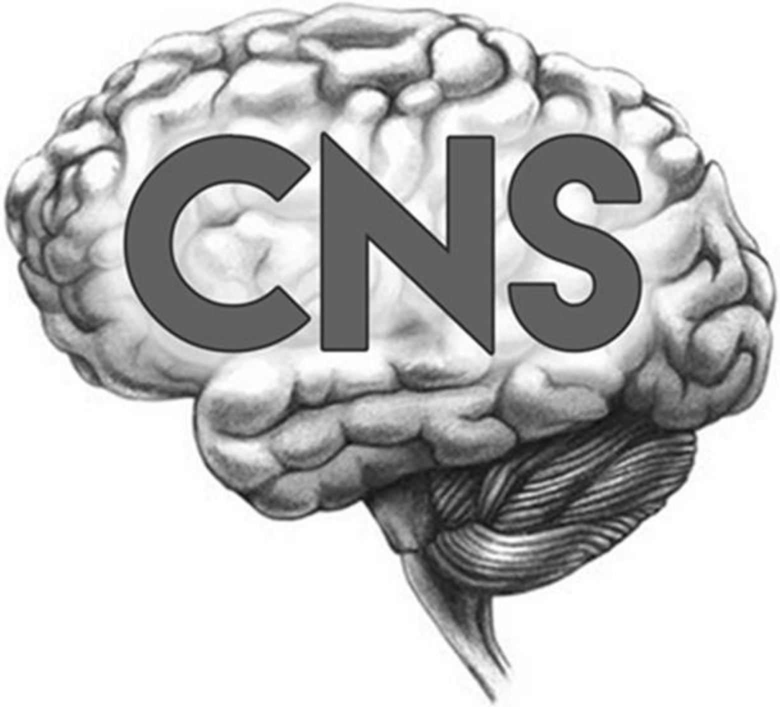

PHYSIOLOGY

☑ ~~Sheet~~ Summary ○ Slide ○ Handout

Number

∞

Subject

1-11

Done By

Saffar et al

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Date: 27/2/2017

Price:

بسم الله الرحمن الرحيم

- This enormous summary sheet contain all the information mentioned by Dr. Faisal and cannot be found or not explained well by Dr. Faraj
- In another words, if you finished studying anatomy, then you can only read this summary to consider yourself studied the 99% of the physiology lectures, ☺
- Enjoy & good luck to you all 🌷

Sheet 1

- Describe the anatomy of the functional unit of the nervous system:

The functional unite of the nervous system is the neuron, and it's composed of:

a) The cell body (soma); and it has almost every organelle of the cell except those needed for cell division "the centrosomes"; because the neurons don't "divide" regenerate.

Once the neurons die they don't regenerate and that's why they are located in a very hard tissue (brain in the skull, spinal cord in the verte bral column "the bone is the hardest tissue in our body").

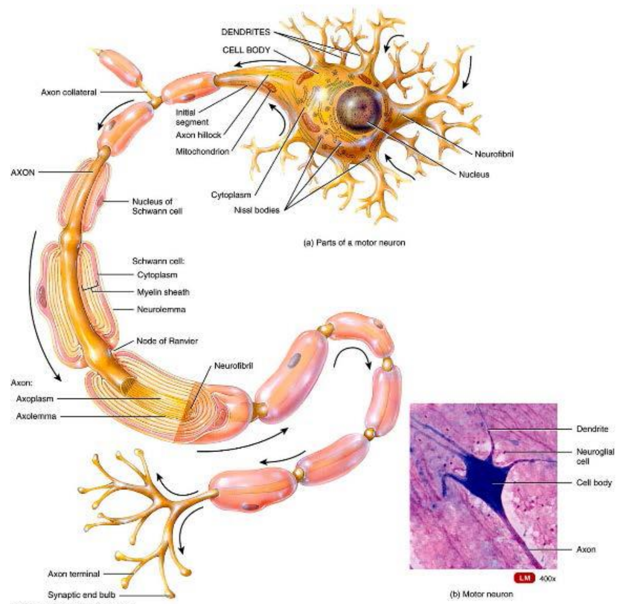


Figure 12.02 Tortora - PAP 12/e

Note: in the brain there are some cells that can regenerate but their function is support not transmission of action potential.

they also have specialized endoplasmic reticulum called Nissl bodies which forms the neuropeptides

b) Dendrites. c) Axon: Mylenated or non-Mylenated d) Axon terminal.

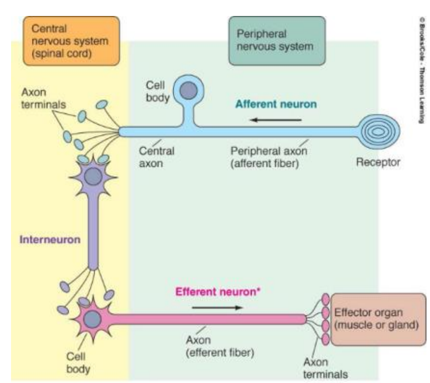
Remember: smooth muscles are supplied by ANS.

Interneurons:

- They connect the sensory to the motor neuron and they occupies most of neurons of the CNS.
- Found entirely within CNS and are responsible for:

a) Integrating afferent information and formulating an efferent response.

b) Higher mental functions associated with the "mind".



* Efferent autonomic nerve pathways consist of a two-neuron chain between the CNS and the effector organ.

- Most of these (sensory) tracts stop at the medulla like gracilis and cuneatus, or stop at the spinal cord, and by stopping it means that they make a synapse with other neurons, theses other neurons will transmit the signal and they are called the second order neurons and they crosses to the other side, which means that the sensation coming from the right will cross to the left side of the brain and vice versa.
- Higher level of organization are located in the Cerebral Cortex:
- The Cortex never functions alone!!!
- It always associates with the lower centers.
- It has a large memory store, essential for thoughts.

Sheet 2

- Premotor and supplementary areas :

Stimulation of their areas gives u a sequence of movements ,so stimulation of the primary motor area doesn't give you coordinated movements it rather gives , simple single muscle contraction. While stimulation of the premotor area gives you coordinated movements.

Quick reading:

Descending Spinal Pathways

- **pyramidal system :**
- Direct
- Control muscle tone and conscious skilled movements (other tracts control the subconscious and the unconscious)
- Direct synapse of upper motor neurons of cerebral cortex with lower motor neurons in brainstem or spinal cord
- **extrapyramidal system:**
- synapses in brain stem areas
- Indirect
- coordination of head & eye movements,
- coordinated function of trunk & extremity musculature to maintaining posture and balance n Synapse in some intermediate nucleus rather than directly with lower motor neurons

extra notes:

- Antigravity muscles : extensors + shoulder and hip joint muscles
- The lateral corticospinal tract controls the flexors for voluntary skilled movement.
- Rubro-spinal controls large muscles of the wrist and elbow.

Premotor Areas:

•Receive information from parietal and prefrontal areas "association"

•Project to primary motor cortex and spinal cord

•For planning and coordination of complex planned movements

Sheet 3

Lateral corticospinal tract controls the movement of distal flexor muscles of the hand(needed for skilled movements and manipulation).

NOTE: pyramidal and extrapyramidal tracts are examples on **corticofugal fibers**, that **Sharpen** the signal by lateral inhibition.

You should know that each motor and sensory signal coming from any part of the body is precisely represented on the thalamus.

***- Generally, afferent fibers to the cortex could be**:*

*1. **corticocortical fibers** :information and orders come to the cortex from the cortex itself. These include:*

a) Association fibers

b)commissural fibers

*2.**thalamo-cortical fibers(specific and non-specific)**: from thalamus to the cortex.*

*3.**extrathalamic subcortical fibers**.* **1.** subcortical fibers from adjacent areas of the cortex (the same cerebral
hemisphere) especially the **somatic sensory areas** from the **parietal cortex**, **visual (occipital**) and **auditory (temporal**) cortex. (point1)

2.subcortical fibers from the **opposite hemisphere** which pass through **corpus callosum**(point1)

3.**somatic sensory fibers** from the **ventrobasal complex** of the thalamus (i.e cutaneous and proprioception).(point2)

- The motor areas in the thalamus are VL and VA nuclei
- The sensory areas in the thalamus are the **ventrobasal complex**, these are subdivided into **VPL (ventropsterolateral**) and **VPM(ventroposteromedial)**

- The alpha motor neurons receive a group of signals from different sources**.** They receive directly from the corticospinal tract and indirectly through interneurons and rubrospinal tract. Some are excitatory signals (EPSP), others are inhibitory signals (IPSP).

All these potentials summate and we end up having the so-called **grand potential.**

Lesions of the primary motor cortex :

1- Primary motor cortex lesions : Loss of voluntary control of discrete movements of the distal segments of the limbs (corticospinal tract lesion)

- In contrary, if you lose the rubrospinal tract, you will still be able to move your distal muscles; you can write and type ..etc. however, you won't be able to flex your elbow and wrist joints (which are also involved in these actions).

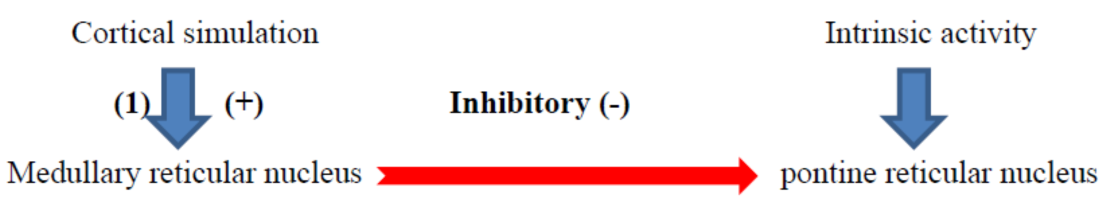
2- Broca's area lesion : expressive aphasia

3- hand skill area lesion: motor apraxia

The muscles of **spinal column** (axial muscles) and **extensor muscles of the leg** support the body against gravity.

- These muscles are under the influence of brainstem nuclei.
- The **pontine reticular nuclei excite** the anti-gravity muscles.
- The **medullary reticular nuclei inhibit** the anti-gravity muscle.

- The pontine reticulospinal tract is intrinsically active (inhibiting the flexor muscles and exciting the extensors).
- Medullary reticulospinal tract is inhibitory to the pontine reticulospinal tract (which is intrinsically active)
- The medullary reticulaospinal pathway rec by the cortex itself receives its stimulation from the cortex itself.



Sheet 4

Refer to pages from 1 to 8 in the sheet.

Functionally ; the cerebellum is divided by longitudinal axis not by lobes;

1- **Vermis**:

→ This is the central part of the cerebellum.

→ It is responsible for the axial muscles such as the neck, hip, shoulder and antigravity muscles…it has the representation of these muscles.

→ It receives feedback from the periphery.

2- **Intermediate zone:**

→ It controls the distal portion of upper and lower limps thus it has the representation of these muscles

→ It receives feedback from the periphery.

3- **The lateral zone:**

→ It controls the sequence, timing,
planning and coordination of the movements, especially for ballistic (i.e. very rapid) and programmed movements; like typing. These movements are very fast that they cannot be controlled by the cortex because it is going to take long time. Thus this part of the cerebellum programs such movements and tells the body to do them -like when you run a computer program to do a certain task and it does what it is supposed to do- .

→ In other words, if a person wants to type something, this part of the cerebellum makes something like a program for his typing movements. This program includes many actions like contraction of the agonist (flexor) and relaxation of the antagonist (extensor) and the opposite might take place within the perfect timing and sequence. So the cerebellum makes this
program and sends it to the body, it will run rapidly resulting in fast, professional typing movements. This is the concept of programming the movements.

→ Receives **No feedback** from the periphery thus there is no representation.

→ *Without this lateral zone, most discrete motor activities of the body lose their appropriate timing and sequencing and therefore become in coordinate*

❖ Cerebellar nuclei:

- All the deep cerebellar nuclei receive signals from two sources:
  (1) The cerebellar cortex
  (2) The deep sensory afferent tracts to the cerebellum
- All efferent fibers of the cerebellum originate from one of the nuclei.
- These deep nuclei are:
- → **Fastigial nucleus:**the deep nucleus of the vermis.
- → **Interposed nucleus:**the deep nucleus of the intermediate zonewhich is composed of globose nucleus and the emboliform nucleus.
- → **Dentate nucleus:**the deep nucleus of the lateral zone.

- **Very important note:** all the efferent fibers of the cerebellum originate from the deep cerebellar nuclei **except** some fibers that arise from the **lateral vestibular nucleus** of the medulla thus this nucleus can be considered **functionally**as a deep cerebellar nucleus. So you can notice that some afferent fibers go directly to the lateral vestibular nuclei and some efferent arise from it directly and go to the vestibulospinal tract.

Sheet 5

Read Quickly!

Longitudinal functional parts of the cerebellum:
1. Vermis (central area): works in conjugation with the flocculonodular lobe to control vestibular function,
Any disturbance in the areas of the vestibulocerebellum will result in **disequilibrium**, and abnormal eye movement which we call nystagmus.
In this area (vermis), cerebellar control of the axial muscles, shoulder and hip girdle takes place.

2. Intermediate zone/paravermis: immediately lateral to the vermis. Being part of the spinocerebellum, it is concerned with controlling motor activity of the distal parts of the upper and lower limbs.
Note: the spinocerebellum executes its motor control through a medial descending pathway in the vermis, and a lateral descending pathway in the paravermis.

3. Lateral zone: works along with cerebral cortex in the planning of coordinated motor movement.

Ø Deep nuclei of the cerebellum:
- Each functional part of the cerebellum has deep nuclei through which the efferent fibers pass, after having originated in the cortex of the corresponding functional part.
§ Vermis (and flocculonodular lobe): an efferent pathway that originates in the vermis passes through the fastigial nuclei
§ Intermediate zone: an efferent originating in this area then passes to the interposed nucleus (to both of its parts the globose and the emboliform)
§ Lateral zone: fibers pass to the dentate nucleus

Vestibulocerebellum and control of equilibrium:

The afferent vestibulocerebellar fibers originate from either:

- (1) the vestibular system (vestibular nuclei in the brain stem) or (2) the vestibular apparatus itself. Almost all of these terminate in the flocculonodular lobe and fastigial nucleus, delivering information concerning balance and position.
- Efferent fibers then leave from the fastigial nucleus to: (1) the reticular formation (fastigioreticular tract), (2) vestibular nuclei, and (3) motor nuclei of certain cranial nerve,,
- Now these three parts send their efferent as vestibulospinal and reticulospinal fibers that arise next

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Spinocerebellum and control of distal limb movements:

❖ The spinocerebellum receives afferents from: (1) peripheral body through the spinocerebellar tract, and (2) the cerebral cortex and brain stem telling the cerebellum about the intended movement through olivocerebellar, reticulocerebellar, vestibulocerebellar tracts and etc…

The job of the ventral spinocerebellar tract is to send a copy of this grand potential to the cerebellum.
This tract is bilateral (affects ipsilateral and contralateral areas.)

Cerebrocerebellum function in planning sequenced and timed movements:

❖ The cerebrocerebellum receives input from: (1) all the different parts of the cerebral cortex; motor areas, and primary and association somatosensory areas, which provides motor information as well as information concerning the posture of the body (from the somatosensory).

❖ The afferent fibers carrying this input are the cortico-ponto-cerebellar fibers. (2) the inferior olive, and even the vestibular nuclei -but are not exactly the same as the ones reaching the flocculonodular lobe-.

☆Important notes:

- Since the afferent fibers of the cortico-ponto-cerebellar tract transmit signals faster than the spinocerebellar fibers, a type of correction can take place even before receiving information about the actual movement.
- Meaning, correction of the commands can occur in two ways: through a long loop that is after receiving information about the actual movement through spinocerebellar tracts , OR through the upward corticoponto-cerebellar pathway (that is before receiving input from the periphery.)

So what are the functions of the cerebellum?
1. Regulation of equilibrium:
When the equilibrium is disturbed -either acceleration or deceleration-, this information is brought up by the vestibular system to the vestibulocerebellum, which initiates immediate corrected signals to the vestibular nuclei and reticular formation, those being part of the medial system pathways, adjust the antigravity muscles (posture) which helps to maintain equilibrium.

- Diseases of the inner ear (affecting the apparatus) disturb this equilibrium -that could manifest as a sense of dizziness-, as well as any damage to any part of the process mentioned above.

Within the context of equilibrium, it is important to mention that a coordination between the exposure to acceleration and the movement of the head must be maintained. This is accomplished through the association between the vestibular nuclei, the superior colliculus and the medial longitudinal bundle.

2. Regulation of posture (axial and antigravity muscles):
If there is any abnormality in this regulation, the body is going to sway (being unable to correct your posture), and to prevent the body from falling down the person would have a wide based gait also known as the drunken sailor gait.
The test performed by policemen to assess the balance of a drunk person is called the heel-to-toe test or tandem test.
3. Coordination of voluntary movement:
[1] working as a damping system.
The cerebellum is able to do this through the information it receives about the rate of change in muscle activity. ([2] Prediction of body position)
The correction is active and occurs very fast that it goes unnoticed.
[3] Planning of sequential movement, especially rapid movements (ballistic movements).
[1],[2] and [3] are mechanisms by which the cerebellum coordinates movement.
4. The role of cerebellum in learning of motor skills:

“The cerebellum learns by its mistakes –that is, if a movement does not occur exactly as intended, the cerebellar circuit learns to make a stronger or weaker movement the next time.”
5. The role of the cerebellum in ballistic movements:
“Ballistic movements, such as the movements of the fingers in typing, occur so rapidly that it is not possible to receive feedback information about it. These are called ballistic, meaning that the entire movement is preplanned and set into motion to go a specific distance and then stop.”

Sheet 6

Summary of the Afferents and Efferents

1. Afferent Pathways to the Cerebellum

A. From the brain

- They are coming mainly from 2 areas to the cerebellum either through the Cortico-ponto-cerebellar tract from the pons & from the motor, premotor and sensory areas of the cortex and it goes to the lateral hemisphere of the cerebellum.
- Or through vestibulocerebellar tract, reticulocerebellar tract and from the inferior olive through olivocerebellar tract.

B. From the periphery

- Dorsal spinocerebellar tract: it's uncrossed tract so bring information ipsilaterally. it receive signals from muscle spindle, Golgi tendon, all large tactile receptors around the joints to tell the person about the position of the joints is it flexion or extension, and how much flexion or extension and so on .
- Ventralspinocerebellar tract: it’s a bilateral tract. It receives the efference copy from the alpha motor neurons.

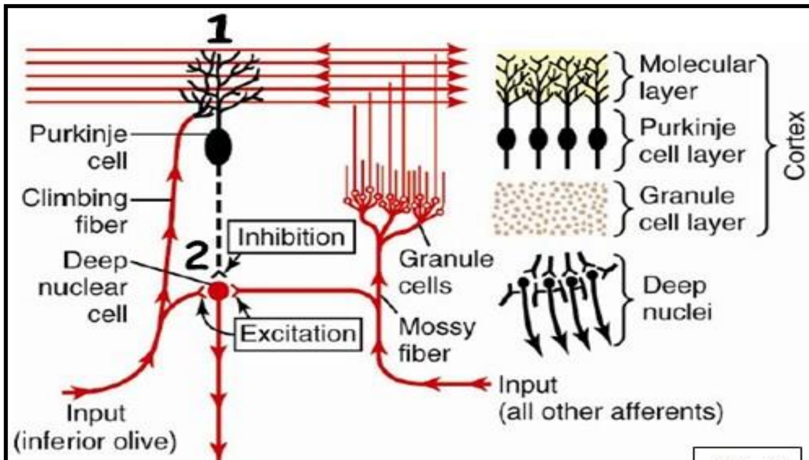
2. Efferent pathways from the cerebellum

- They have to go out from the deep cerebellar nuclei, remember:
- The deep cerebellar nucleus of the vermis and flocculonodular lobe
- fastigial nucleus
- The deep cerebellar nucleus of the intermediate zone
- The interposed nucleus (interpositus) with its 2 parts; Emboliform and Globose.
- The deep cerebellar nucleus of the lateral zone
- The dentate nucleus.

    - From the vermis: fastigioreticular tract and fastigiovestibular tract; for equilibrium control.

- ❖ From the intermediate zone: Interpositorubral tract to the red nucleus and interposito-thalamic tract to the thalamus.
- ❖ From the lateral hemisphere: dentatothalamocortical tract.

- ➢ Climbing fibers action potential is complex spiky potential, they send branches to the deep nuclear cells before they make extensive connections with the dendrites of the Purkinje cell.(follow the course of these fibers on fig- 3 to get this)



(Fig-3)

- ➢ Mossy fiber action potential is simple spiky potential. The granular cells are excitatory and they receive their inputs from mossy fibers; and these fibers also excite the deep cerebellar nuclei.
- ➢ The inferior olive is important for learning, why? Because when it goes and stimulate the deep cerebellar nuclei and stimulate the purkinje fibers it changes/alters the sensitivity of purkinje cells (their action potential or their excitatory postsynaptic potential), so the purkinje cells learn from the previous excitation that come from the climbing fibers, so the next time its sensitivity is changed or altered. So by this way the purkinje cells learn the next time.

» That's why the inferior olive is very important; also, because it receives feedback, so it works as if it's a cerebellum by itself it sends excitation to the deep cerebellar nuclei and it receives feedback, so it do comparison between the actual and the intended function; it receive Corticospinal and sensory from the muscles. So, if there is a mismatch; it corrects it, and send it through the climbing fibers. (Some signals pass with correction, other without it).

Cerebellar Disorders

1. Flocculonodular lobe (vestibulocerebellum) disorders: It is manifested by disequilibrium (sometimes with nystagmus); the patient is:

A. Swaying down during standing with a tendency to fall down, the patient is unable to balance and unable to maintain equilibrium.

B. Having Unsteady (staggering, drunken) gate, it's tested by the tandem test (heel to toe test). When the traffic police suspect an alcoholic driver, they test them by asking the driver to walk heal-to-toe in a straight line; he won't be able to do that if he's drunk.

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2. Vermal disorders: It is manifested by inability to maintain the upright standing posture due to failure to adjust the tone disequilibrium.

3. Neocerebellar syndrome

» It results from vascular strokes, degenerative disorders, or tumors/neoplasm.

» It is manifested by: hypotonia, asthenia, and ataxia.

A. Hypotonia:

- Remember: when the deep cerebellar nucleus is excited by the climbing fibers and mossy fibers, it sends these excitations back to the corticospinal tract to enhance the tone so, when the cerebellum is diseased decrease in the tone of skeletal muscles due to decreased facilitation of the **γ** motor neurons as a result of decreased supraspinal facilitation.
  - It's associated with hyporeflexia
  - decreased stretch reflex.
  - The knee jerk becomes pendular “like the pendulum”; it doesn't stop because the cerebellum is what does calculation to stop it.

B. Asthenia:

There is weakness because of enhancement loss, the muscles will fatigue more readily than normal, as a result of interruption of the activating effect of the cerebellum on the cerebral cortical motor areas.

When we take our physiology lab we will learn sensory and motor examinations, one of these examinations is reflexes.

We-as beginners- won't be able to tell if this is hypo-reflexia or hyper-reflexia, but by experience we will know that ++ is normal reflexia, + or zero is hypo reflexia and +++ is hyper reflexia (This is how the deep tendon reflexs are graded)

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C. Ataxia(or Asynergia):

Cerebellar ataxia can manifest itself in many ways:

1) Dysmetria

- Errors in the range and direction of the movement, because of improper measurement of the distance, i.e. abnormal calculations in the higher range or in the lower range. That will result in past pointing or hypermetria
- The patient will "overshoot" while doing the thumb – nose test because the movement is cortical, but if there is a cerebellum it will break it
- No overshot, it will stop at the intended position.
- So these errors result from failure of the "comparator" and "damping" functions of the cerebellum that normally adjust the course of the movement and bring it smoothly to the desired position.

2) Intention (action/ kinetic) tremor:

When the patient extends his arm, the cortex will think that the movement is very rapid so the patient will take his arm back, which is also seen to be very rapid then the patient will try to extend his arm again and so on.

- The cerebellar patient is normal without movement and he will have these tremors when doing a movement (i.e. they appear when the patient performs a voluntary motor act, and not seen when the muscles are at rest).
- Note: basal ganglia patient (e.g. Parkinson patient) will have resting tremors, it disappears while doing a movement.

3) Decomposition of complex movement

- The motor action is carried out as several fragmented steps rather than a smoothly progressing movement.
- For instance, in reaching for an object by the hand, the cerebellar patient may first move the shoulder joint, then the elbow, followed by the wrist and fingers → simulate movements of a "robot".
- Again, the cortical movement is slow (the patient moves the shoulder then the elbow then catch the cup for example), so the movement is decomposed/broken down (as the slow motion of a robot) because there is no cerebellar control.
- Talking is a rapid movement, (rapid contraction of agonists and antagonists), when it's decomposed the patient will have slurred speech
- dysarthria (dys: abnormal, arthria: articulation).–refer to point 7 next page-.

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4) Rebound phenomena:

- The cerebellar patient is unable to stop the ongoing movement rapidly due to failure of the predictive and damping functions of the cerebellum. This can be observed in what is called "rebound phenomenon".
- When there is a flexion of the forearm against resistance (provided by the examiner's hand), the cerebellar patient cannot stop the resultant inward movement of his limb in due time

following its release, and the forearm flexes forcibly and may strike his body with considerable violence.

)امسك يد المريض واحكيلو يشد , ولما تفلت يدك فجأة بترتد يد المريض عليه و بيضرب حاله لأنه ما قدر يوقف الحركة(.

5) Dysdiadochokinesia:

- The inability of the patient to perform rapid alternating opposite movements (e.g. rapid repetitive pronation and supination of forearm)
- The movements will be slow and irregular. But if he cannot do it at all
- Adiadochokinesia:

- It results from failure to adjust precisely the proper timing for the onset and termination (Inability to calculate the time).

6) Nystagmus

- Cerebellar nystagmus is a tremor of the eyeballs (rapid eye movement) as a result of "dysmetria" of the eye movement.

7) Scanning Speech (Dysarthria):

- It's the inability to connect the words together; each word is fragmented into several syllables, producing "scanning" or "staccato" speech, like someone trying to speak an obscure foreign language for the first time.

8) Unsteady Gait

- The gait is broad-based due to dysmetria and kinetic tremors of the lower limb muscles.

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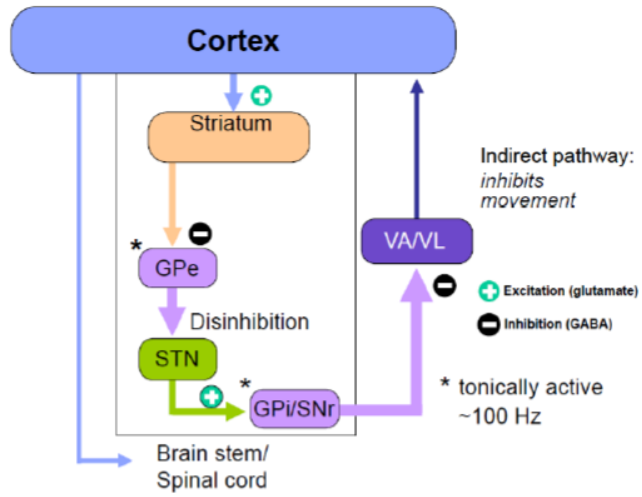
❖ **Summary of Clinical Abnormalities of the Cerebellum:**

- All signs of cerebellar diseases are ipsilateral since there is double crossing- from cortex to pons and back to cortex.
- Basal ganglia diseases are contralateral.
- Ataxia and intention tremor:
  - Failure to predict motor movement, patients will overshoot intended target, past pointing.
- Dysequilbrium- ataxic (staggering) gait (drunken gait)
- Dysdiadochokinesia (Adiadochokinesia):
  - failure of orderly progression of movement
- Dysarthria:
  - Failure of orderly progression in vocalization
- Cerebellar nystagmus:
  - Intention tremor of the eyes when trying to fix on object.

Sheet 7

The doctor only said that GPi and GPe are tonically active, and the striatum is tonically inactive

Indirect Pathway:



- The overall effect of the indirect pathway is to reduce the activity of neurons in motor areas of the cerebral cortex.

  Cortex → striatum → GPe → STN → GPi → thalamus (VA/VL) → Cortex

- "GPe → STN →": ☜ this is what makes the indirect pathway indirect.
- STN is excitatory to the GPi, This excitation is supposed to make the GPi more active → More inhibition of the thalamus → Reduction in movement. (This occurs without cortico-striatal signals).
- When there's movement, the cortex excites the striatum, that will inhibit GPe. So, the previously-inhibited STN, would be more active (i.e. disinhibition). Conclusion: Direct pathway: Disinhibition of the thalamus ⍰ enhancement of motor activity. Indirect pathway: Disinhibition of the STN ⍰ reduction of motor activity.

❖ <u>The Effect of Dopamine on the Direct and Indirect Pathways of the Basal Ganglia:</u>

- Dopamine is one of the neurotransmitters in the brain, and is produced by neurons of the pars compacta of the substantia nigra.

- Dopamine has an excitatory effect on the direct pathway, and an inhibitory effect on the indirect pathway (depending on the receptors, with which it binds).

- The striatal fibers that project into the GPi (those of the direct pathway) have D1 receptors, and dopamine here is excitatory.

- The striatal fibers that project into the GPe (those of the indirect pathway) have D2 receptors, and dopamine here is inhibitory.

- Dopamine has a modulatory, rather than a direct effect on the striatum (i.e. it modulates the action of other neurotransmitters).

Dopamine is excitatory = It makes other neurotransmitter more able to excite striatal neurons.

Dopamine is inhibitory = It makes other neurotransmitters more able to inhibit striatal neurons.

- Extra information: D1 and D2 receptors are capable of excitation and inhibition, because they are coupled to Gs and Gi proteins, respectively.

Important Note: In all basal ganglia disorders, the motor dysfunction is contralateral to the diseased component. This is understandable because the main final output of the basal ganglia to the body is mediated by the corticospinal tract. Cerebellar diseases cause ipsilateral motor dysfunction.

Sheet 8

the internal and external segments of globus pallidus are tonically active.

Refer to pages 2-3

patients with parkinson's disease are *monotonous* with monotone voice and show slow response and slow emotions .

Integration of motor control. *(1)Spinal cord level*: preprogramming of patterns of movement of all **muscles** (i.e., withdrawal reflex, walking movements, etc.). (2) *Brainstem level:* maintains equilibrium by adjusting **axial tone**. *(3) Cortical level:* **issues commands** to set into motion the patterns available in the spinal cord and controls the intensity and modifies the timing. *(4) Cerebellum:* functions with all levels of control to **adjust motor activity**, equilibrium, and planning of motor activity. *(5) Basal ganglia:* function to assist cortex in executing subconscious but **learned patterns of movement**, and to plan sequential patterns to accomplish a purposeful task. Refer to figure (8-2).

Blood brain barrier (BBB). It is a highly selective semipermeable membrane barrier that separates the circulating blood from the brain extracellular fluid in CNS, it is formed by the brain endothelial cells of microvasculature capillaries which are *continuous* by tight junctions, so patient with meningitis, it is more favorably to give the antibiotic *intrathecally* not orally to reach CSF since it is difficult to pass through BBB.

In the center of grey matter, there is *central canal* which is a continuation of the fourth ventricle thus **CSF** passes from the fourth ventricle to central canal. In **syringomyelia**, the central canal is enlarged compressing the surrounding neurons resulting in symptoms.

Note: the number of interneurons is much more than the number of motor and sensory neurons in the segment. (not important, wrote it just in case it was in the choices)

Even though that lateral (medullary) reticulospinal tract is located in the medial(anterior) column (anatomically), it is considered as a part of lateral system functionally. this is according to Dr.Faisal, but according to Dr.Faraj; he considers that lateral reticulospinal tract as a part of the medial system.

Interneurons and propriospinal fibers. Interneurons are 30 times as many as anterior motor neurons, small and very excitable and comprise the neural circuitry for the motor reflexes, while Propriospinal fibers travel up and down the cord for 1 - 2 segments especially for pain sensation providing pathways for multisegmental reflexes.

the peripheral contractile part of muscle spindle to contract outward(laterally) while the extrafusal fibers contract inward(medially) (not important)

Sheet 9

Note: Density of muscle spindles is more in the muscles that are used too much; these are axial and antigravity muscles.

Ia and II fibers have a basal rate of firing. This is very important for positive and negative control.

Clonus is an example on the sinusoidal stretch.

Note: As the dynamic fibers sense the change in rate, they help in the predictive function of the cerebellum. So they help us to stop the movement at the intended point. (Remember Ia fibers reach the cerebellum through spinocerebellar tracts).

Refer to pages 3-5

Important note before we continue: Remember that the Dorsal SpinoCerebellar tract receives its input from Muscle Spindles and Golgi Tendon Organs to correct the actual movement, so it has the same afferent fibers of the stretch reflex. So notice in the figure above that in the knee jerk some afferents will go to the brain through the dorsal column. Which means that there are no specific fibers that will do stretch reflex and other specific fibers for the cerebellum (Both are Ia). Other reflexes may use this circuit also, simply because all of them will reach the motor neurons of the muscles finally. So please don't get confused by this

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Deep tendons reflex circuit is at the level of the spinal cord (so they can be used to test the integrity of the spinal cord), so it's better to distract the patient when doing the reflex (by telling the patient to clench their teeth, flex both sets of fingers into a hook-like form and interlocks those sets of fingers together) so that the cerebral cortex won't affect the reflex consciously.
Deep tendon reflexes can be done with almost any muscle. Cortical lesions usually increase muscle stretch reflexes.

Characteristics of Deep Tendon Reflex: Monosynaptic reflex / Ipsilateral / Receptors are located in the same muscle stimulated by lengthening of muscle (stretch).
Characteristics of Tendon Reflex: Disynaptic (You can also say polysynaptic) **/ Control muscle tension by causing muscle relaxation when muscle tension is great.**

Flexor Reflex (Withdrawal Reflex):
The stimulus in this reflex is nociceptive (pain). So the afferents to this reflex are carried by **Aδ and C** fibers (that carry pain). Remember that these fibers are relatively slow.
The afferent fibers will synapse with **interneurons as well as propriospinal** tracts so it will travel to other segments of the spinal cord and the final effect will be contraction to a **whole group of muscle** (Flexors of the leg for example).
As always, there is reciprocal inhibition of antagonist group of muscles on the same side.
Characteristics of Withdrawal Reflex: Polysynaptic reflex / Ipsilateral / Pain Stimulus.
The Flexor reflex has another part, which is called the **Crossed Extensor Reflex**. When a person flexes his leg due to a pain stimulus, he will fall if the other leg didn't extend. So the function of the Crossed Extensor Reflex is to extend the other leg during the flexor reflex.
This happens by interneurons that cross the middle line to the other side, and do the exact opposite of the flexor reflex (i.e. stimulate extensors and inhibit flexors).
Note: Remember that the central canal is in the middle, so a problem in the central canal may affect this reflex.
Characteristics of Crossed Extensor Reflex: Polysynaptic reflex / Contralateral reflex / Multi-Segmented / Pain stimulus.

Sheet 10

Crossed extensor reflex:

- The crossed extensor reflex is slower than the flexor reflex, because of something called synaptic delay, the duration of which is around 0.5 milliseconds in every synapse, so it makes sense for the crossed extensor reflex to be slower since it has more synapses.
- A pain stimulus starts in the pain receptors in the skin and the impulse travels through the sensory neuron to the spinal cord, in the spinal cord it ascends or descends 1 or 2 segments via ascending and descending interneurons, the flexors are then excited and the extensors are inhibited ipsilaterally, while the opposite happens contralaterally, notice that there are too many synapses.
- The crossing from one side to the other happens anterior to the central canal, this is very important as the central canal might enlarge (a disease called **Syringomyelia**, enlargement of the central canal) and it might destroy these crossed fibers so you might <u>lose</u> the crossed extensor reflex.

- As you remember, signals from the golgi tendon organs and from the muscle spindle are transmitted to the spinocerebellum through the dorsal spinocerebellar tract via 1a and 1b fibers, respectively. This is very important for feedback control as their conduction speed might reach 120 m/s which is needed for this function.

After Discharge:

- In the synapse there is something called (after discharge) "other than the synaptic delay"
- synaptic delay is the time taken for the neurotransmitter to be released from the presynaptic end and travel to the post-synaptic membrane and then cause action potential , which takes around 0.5 millisecond
- action potential last around 0.1 ms “very fast”
- Excitatory post-synaptic potential (**EPSP**) last much longer than action potential, around 10 ms!
- In this 10 ms, it will still give Action potential because it will stay above **threshold** , this is what we call <u>after discharge</u>

- Now the cross extensor reflex is slower and takes longer time to react, due to the long After Discharge
- It begins 0.2-0.5s after the pain stimulus has started
- It serves to push the body away from the stimulus, also to shift weight to the opposite limb.

Babinski reflex:

- If there was an upper motor neuron lesion in an adult, we will get the Babinski reflex, but normally plantar reflex occurs when we stimulate the plantar surface of the foot
- Yet Babinski reflex occurs normally in children!
- Babinski sign is extension of the big toe and flexion of the others,

Reflexes that Cause Muscle Spasm:

- Rigid abdomen ➔ emergency case, occurs due to perforated appendix "visceral reflex" (muscle will spasm and become rigid)
  **pay attention to this as it's not caused by basal ganglia or decerebrate rigidity!!*

Sensory system

Transducer:

- a device that changes variety forms of energy "mechanical, chemical, etc.." to electric energy, or vice versa
- Receptors are transducers that change the received sensation into electric signal!
- they change the mechanical energy of touch and sound, electromagnetic energy of light, chemical energy of taste and smell, to electrical which generates Action potential,
- and the cortex only receives this action potential, yet it knows that this action potential is coming from pain, touch or sounds due to the (specificity of receptors) "labeled line principle"
- specific (special) pathway for these receptors
- if you stimulate this pathway anywhere you will get the same sensation of the receptor, *(it will reach the cortex as a normal stimulus)*
- every part of the body has a sensory representation in the cortex (just like the motor part), and that's how the cortex knows where every sensation is coming from
- these receptors are sensitive to "adequate stimulus"

in another words: the sensory receptors/neurons are sensitive to **quality** not quantity of the stimulus

refer to coding mechanism, Types of synapses, receptors & neurotransmitters in sheet 10, page 6-12

Sheet 11

- the **encapsulated** receptors are **rapidly** adapting receptors >>> the pacinian and meissner's corpuscles . these two types of receptors are different in the **frequency of vibration** they are able to sense depending **on their adaption { the pacinian corpuscles are faster than the meissner's corpuscle then they are able to sense higher frequency vibration (1000 HZ) }** .

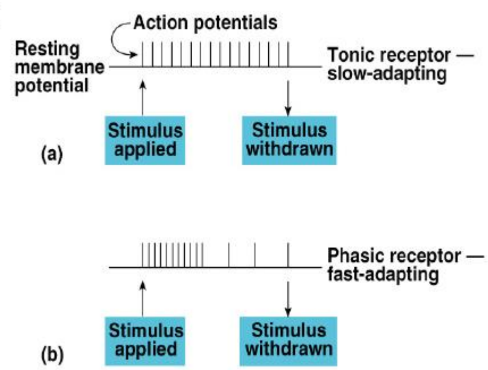
refer to "Transduction of sensory stimuli into nerve impulses" subject in sheet 11 page 2-4

Adaptation of the receptors:

- Another characteristic of all sensory receptors is that they adapt either partially or completely to any constant stimulus after a period of time .
- Two types of receptor in term of adaptation :

A. Rapidly adapting phasic receptors :

1. respond **only when change** is taking place
2. Rate and Strength of the response is related to the Rate and Intensity of the stimulus then it's important for **predicting the future position or condition of the body**
3. **Very good for vibration sense** .
4. Similar to dynamic response in the muscle spindle
5. very important for balance and movement types of rapidly adapting receptors: *pacinian corpuscle, semicircular canals* in the inner ear.



B. Slowly adapting ,tonic receptors :

1. *continue to transmit impulses to the brain for long periods of time while the stimulus is present* ; keep brain apprised of the status of the body with respect to its surroundings
2. will adapt to extinction as long as the stimulus is present, however, this may take hours or days
3. these receptors include: *muscle spindle, golgi tendon apparatus, Ruffini's endings, Merkelsdiscs, Macula, chemo- and baroreceptors*

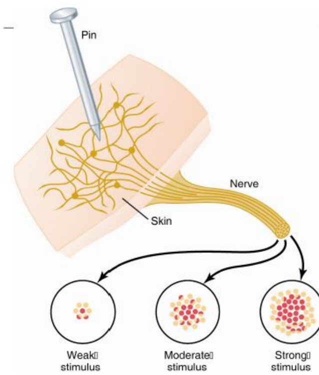
- **Sensory Adaptation**
  o **Tonic receptors**: Produce constant rate of firing as long as stimulus is applied .
  o **Phasic receptors**: Burst of activity but quickly reduce firing rate (adapt) if stimulus maintained.
- Pain receptors and proprioceptors **do not** exhibit adaptation
- **Mechanism of Adaptation varies with the type of receptor :**
  1. *photoreceptors* change the amount of light sensitive chemicals
  2. *mechanoreceptors* redistribute themselves to accommodate the distorting force (i.e., the pacinian corpuscle) , some mechanoreceptors adapt slowly, some adapt rapidly .

Nerve fibers that transmit signals

- Types of Nerve Fiber
  1) Myelinated fibers – Type A (types I, II and III)
  A α / A β / A γ / A δ { depending on their size }
  2) Umyelinated Fibers- Type C (type IV)
- Neurons attached to A β / A γ / A δ / A α {{ very little attached to A α **}}** **rapidly** transit the signals .
- Neurons attached to C **fibers slowly** transit the signals.
- the larger the nerve fiber diameter the faster the rate of transmission of the signal
- velocity of transmission can be as fast as 120m/sec or as slow as 0.5 m/sec
  The **rapid** transmission of the signals makes it **good oriented in term of time and place** >>> when the transmission is rapid then there is **no interconnection between the signal and the next one**

Transmission of signals of different intensity in nerve tract:

- Gradations in signal intensity can be achieved by:
  1) increasing the number of fibers stimulated, **spatial summation**
  2) increasing the rate of firing in a **limited number** of fibers ; increase the frequency of the nerve impulses in each fiber , **temporal summation**.
- **The Spatial Summation :**
  This figure shows us three section of the nerve bundle from skin are with different intensity of the stimuli .



An example of spatial summation

Figure 46-7; Guyton & Hall

  From the left :
  1st section >>> is the effect of weak stimulus >> only a single nerve fiber in the middle of the bundle stimulated strongly (red colored fiber) whereas rhe adjacent fibers are facilitated { that means their membrane potential is close but do not reach the threshold } .
  2nd section >>> the effect of moderate stimulus the number of strongly stimulated fibers increase { the fibers that were facilitated previously with this stronger stimulus become stimulated now } and the fibers that surround the stimulated become facilitated .
  3rd section >>> the effect of stronger stimulus {stronger than 2nd stimulus } >> progressively more fibers being stimulated
  From this example >>> the stronger signals spread to more and more fibers >> that is called spatial summation.

Le Fin.

"Don't let small minds convince you that your dreams are too big." – Uknown

Done by: ***Omar Saffar, Mohammed Sabbagh & Yousef Al-As3d***