CENTRAL NERVOUS SYSTEM

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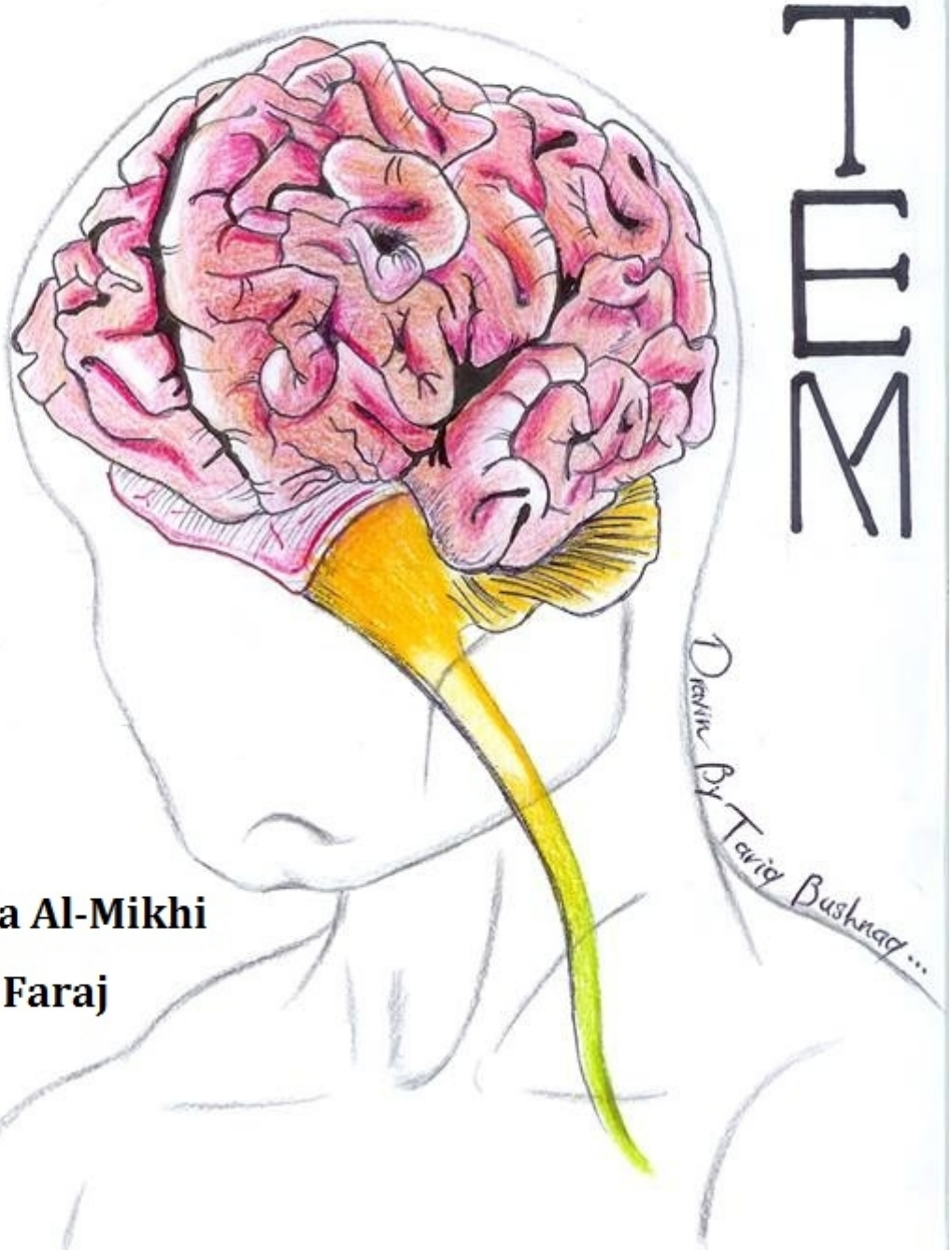

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Lec #: **4**

Muscle Spindle and Stretch reflex

Hello, I've written this sheet according to section's 1 recording and rearranged a couple of information. Let's begin:

- Any reflex has certain components; one of them is a receptor (which is considered the beginning of the reflex). The receptor for the stretch reflex of skeletal muscles is found inside them and is named as: muscle spindle.

NOTE: *Stimulation of any receptor causes the formation of receptor potential (partial depolarization) that is converted by a certain mechanism into action potential.*

Skeletal muscles can be divided into:
(1) **Intrafusal:**This is the muscle spindle (stretch receptor).
*structure of spindle: modified skeletal muscle fibers, 4-10 small muscle fibers surrounded by a connective tissue capsule which is fusiform-shaped (hence the word fusal). They are modified by having a central non-contractile part and an outer contracting part that contains sarcomeres.
(2) **Extrafusal:** the majority of the skeletal muscle and it is outside the spindle.

How do we stimulate the muscle spindle?
1- **Stretching** a muscle for example passively whch activates muscle spindle and as we said any receptor upon stimulation would generate receptor potential and convert it into action potential. Once the action potential is generated we consider the receptor (spindle) active, and it begins to discharge and send impulses through the dorsal root of spinal nerve and synapse with alpha directly.

➡ **This is called Monosynaptic reflex.**

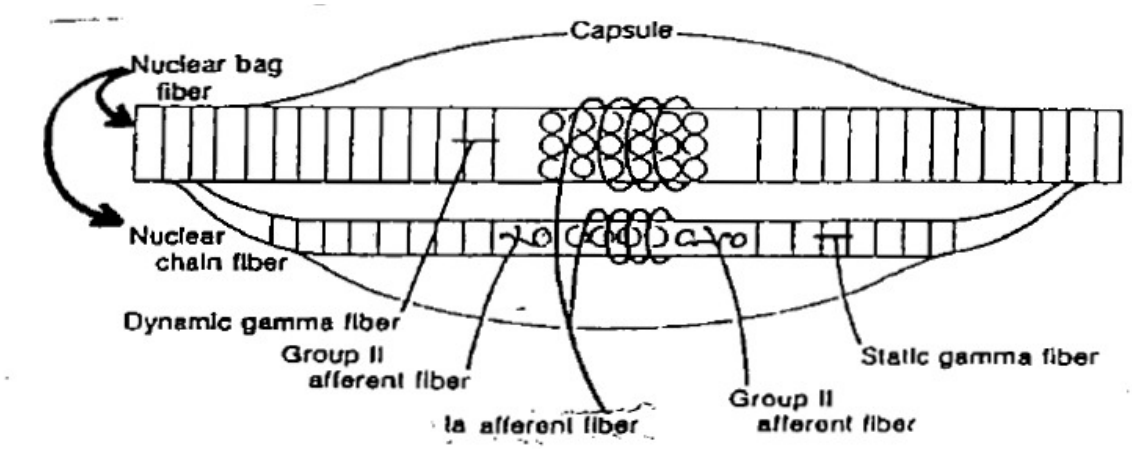
Note*: this is the ONLY monosynaptic reflex in the body.*

When alpha is activated, it will send impulses through its efferent fibers to the extrafusal muscles causing their partial contraction.
** This partial contraction is named **muscle tone,** and it is important for posture, standing up, and sitting down. In order for ANY movement to happen we need

minimum contraction (muscle tone) since it can NOT be started from zero. Also, we need action potential for any movement.
In order to generate an action potential the membrane must be in the resting membrane potential.

In a Nutshell: whenever a skeletal muscle is passively stretched, the spindle is activated and eventually leads to partial muscle contraction reflexly to bring back its length back to normal.

2- **Gamma** motor neuron, this will be discussed later on in this lecture.



Now, looking at the figure above, we classify intrafusal muscle fibers(you can notice the capsule surrounding it) into two types:

(A) Nuclear Bag Fibers: These have many nuclei, which are groupedtogether forming a dilated bag in the central part of the receptor area
(B) Nuclear chain fibers: These have a smaller number of nuclei,forming a chain throughout the receptor area. These fibers are thinnerand shorter than the nuclear bag fibers, and their ends are connectedto the sides of nuclear bag fibers.

-Both Nuclear bag and chain are connected to the extrafusal muscles.

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Also notice in the figure the nerve fibers:
*What are the afferent (sensory) nerve fibers coming out from the spindle?
(1) Primary afferent: type Ia, winds around both nuclear bag and chainfibers
(2) Secondary afferent: type II, found only in nuclear chain fibers.

NOTE: afferent sensory fibers run with dorsal root of any spinal nerve.

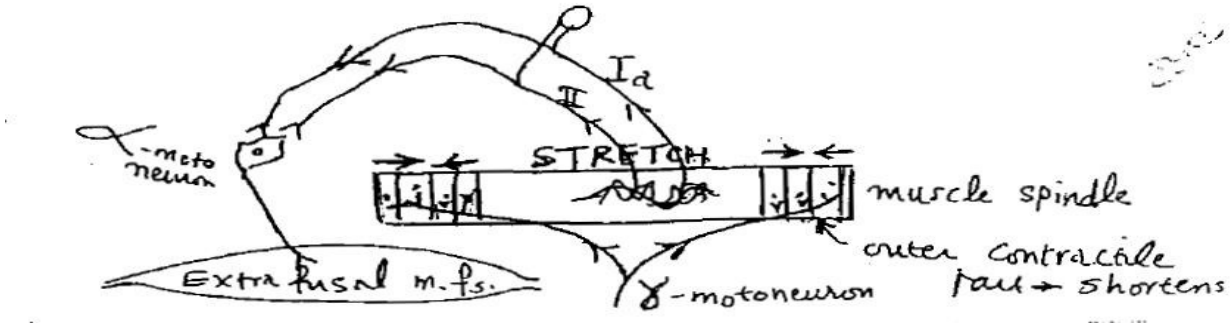
*What are the efferent nerve fibers reaching the outer contracting part of spindle?

~~γ fibers.~~ →

γ efferent fibers

- Dynamic Fibers → Gamma-d → Supply nuclear bag fibers.
- Static fibers → Gamma-s → Supply nuclear chain fibers.

Remember we said earlier that the muscle spindle can be activated by either stretching the muscle or by gamma fibers, now look at the picture below to understand the mechanism by which gamma works:



step #1: γ (whose axon, like the α-motor neuron, starts at the ventral root of the spinal nerve) innervates the outer contractile part of the muscle spindle causing its contraction and shortening, this leads to stretching the central part activating the spindle. (Notice that when the outer part contracts, the inner part stretches because the inner part is NOT contractile!)

step #2: Formation of a receptor potential (recall that muscle spindles are receptors), then action potential that runs with afferent fibers (primary Ia and secondary II) to α-motor neuron through the dorsal root.

step #3: α causes stretch reflex, which is partial contraction of extrafusal muscle fibers.

-Higher centers (cortex, brain stem, descending motor pathways) control gamma as well as alpha directly or indirectly (through interneurons), so all motor pathways (whether pyramidal or extrapyramidal) should reach alpha motor neurons either directly or through interneurons.

-Muscle spindle is inhibited by contraction, and again activated by gamma or stretch.

**For a fast contraction we stimulate alpha, which in turn stimulates the major extrafusal part of the muscle. BUT for a slow movement and muscle tone we stimulate gamma which works by the mechanism mentioned above.

- Alpha and Gamma co-activation (very important):

In order to produce a continuous contraction we must stimulate both alpha and gamma, because if we stimulate only alpha it causes fast contraction and as mentioned before, contraction inhibits the spindle, so gamma takes action to keep the spindle active and alpha would receive afferent fibers resulting in continuous contraction.

In a nutshell: For fast contraction: stimulate alpha. For muscle tone: stimulate gamma. For continuous contraction and a certain movement: stimulate both.

Stimulation of gamma is easier and simpler than alpha by higher centers .Since gamma motor neurons are **smaller than alpha motor neurons, they have **lower threshold** for excitability than the alpha motor neurons thus they are easily excited and have higher tonic discharge rates.

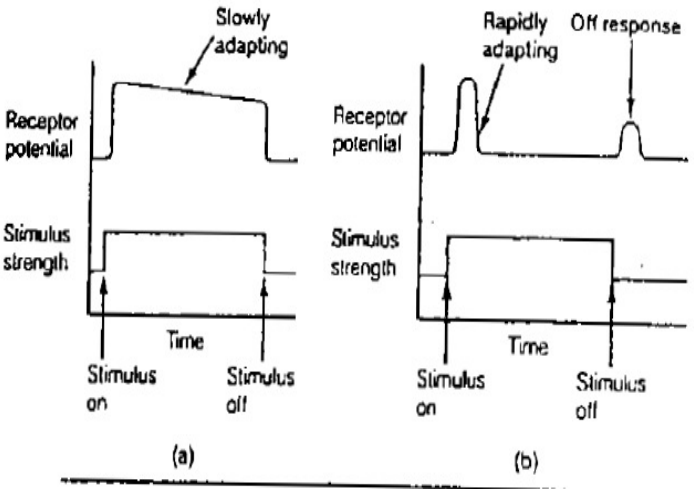
Muscle contraction could be produced either directly or indirectly. Directly through stimulation of alpha motoneurons by higher centers and indirectly through gamma motoneurons.

Always remember that:
*Muscle Spindle is a modified intrafusal skeletal muscles that work as stretch receptors.
*Stretch Reflex is the contraction of the passively stretched muscle by stimulating the spindle using gamma or passively stretching the muscle, bringing its length back to normal.
*Stimulated spindle sends impulses through afferent fibers (Ia and II) to alpha motor neuron. Then alpha sends impulses through efferent fibers to extrafusal muscle fibers causing its contraction.

- Does the stretch reflex maintain normal length of muscle? How?
Yes, whenever a muscle is stretched the stretch reflex is activated causing contraction of muscle and leading to the return of the muscle to its original length.

-Now look at the figure and notice the different responses from the afferent fibers:

Figure A- Static response of Secondary endings:slowly adapting receptors, as long as there is a stimulus they continue to discharge.
- stimulated by gamma-s.
Figure B- Dynamic Response of Primary Ending:rapidly adapting receptors, gives a fast response and then quickly goes back to baseline.
-stimulated by gamma-d.



Keep in mind that the primary endings are found in both nuclear bag and nuclear chain, whereas secondary is found only in nuclear chain.

Gamma-s supplies nuclear chain (hence secondary endings) whereas gamma-d supplies nuclear bag (hence primary endings).

Accordingly stretch reflex can be divided into:
(A) Dynamic stretch reflex:
Whenever we stimulate the tendon of a certain muscle, we can notice fast contraction of that muscle followed by rapid relaxation, because by this way we stimulated the primary endings (Ia).
Example: **Tendon Jerk**.

(B) Static stretch reflex:

This occurs upon stimulation of slowly adapting secondary never endings. This is the basis of **muscle tone.**

Dynamic stretch reflex	***Static stretch reflex***
Rapidly adapting	Slowly adapting
Phasic receptor	Tonic receptor
Baseline for jerk	Baseline for tone
Response of primary endings (Ia)	Response of secondary endings (II)
Stimulated by gamma-d	Stimulated by gamma-s

-NOTE: all the skeletal muscles in our body are shorter than the distance between origin and insertion, which means our muscles are always stretched, stimulating the stretch reflex and therefore creating muscle tone.

**Knee jerk: stimulating the patellar tendon, leads to contraction of quadriceps femoris by stimulating muscle spindle and sending impulses through primary afferent fibers to alpha in L2,3,4 (since the muscle is innervated by femoral nerve) Leading to rapid contraction followed by rapid relaxation.

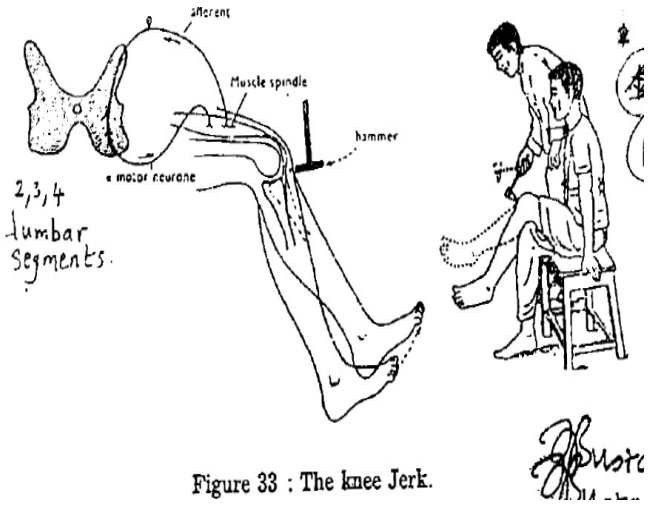


Figure 33 : The knee Jerk.

- What is the importance of a jerk?

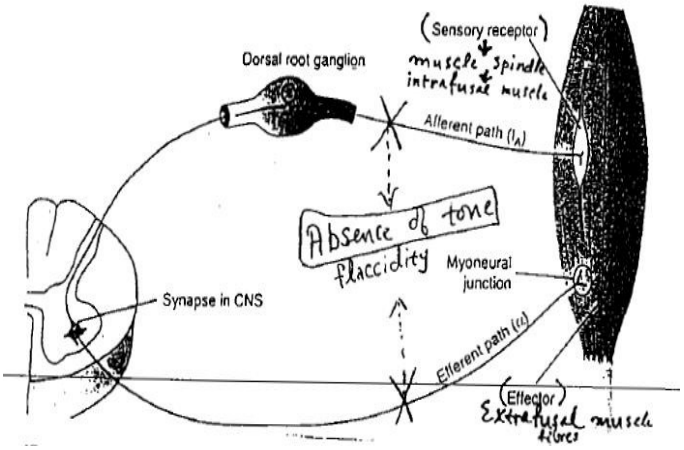
(1) If there was considerable contraction upon stimulating the tendon, then jerk is **normal**~~normal~~ afferent fibers, efferent fibers, and spinal cord segment that contains alpha.

(2)**Areflexia**: something is wrong either in the muscle spindle, or afferent or efferentfibres.
(3) **Hyper-reflexia**: this indicates that a certain part of this reflex is over-active; the major part responsible for this reflex is the muscle spindle.
-hypersensitivity of the muscle spindle indicates over-activity of gamma.
-gamma overactivity indicates upper motor neuron lesion (damage to area4, or 6, or downstream pathways)

Note that hyper-reflexia is not indicative of upper motor lesion and is usually of no medical significance unless associated with other signs, since it could be caused by stress.

**Skeletal Muscle Tone:

-Definition: partial contraction in a skeletal muscle.
- It is a neurogenic property (while plain muscle tone, like tone of GI tract muscles for example, is a myogenic property or property of the smooth muscle)
-Interruption of this reflex anywhere leads to atonia (no muscle tone) therefore no contraction.
Ex: Poliovirus damages alpha motor neuron leading to lower motor neuron lesion.
(Keep in mind that damage of the cell body leads to death of the axon)
- Any contraction is followed by relaxation unless gamma interferes (α γ co-activation or γ-loop)



*Methods of stimulating alpha:
1- Higher centers through descending motor pathways
2- By gamma that stimulates muscle spindle which sends impulses to alpha by afferent fibers.

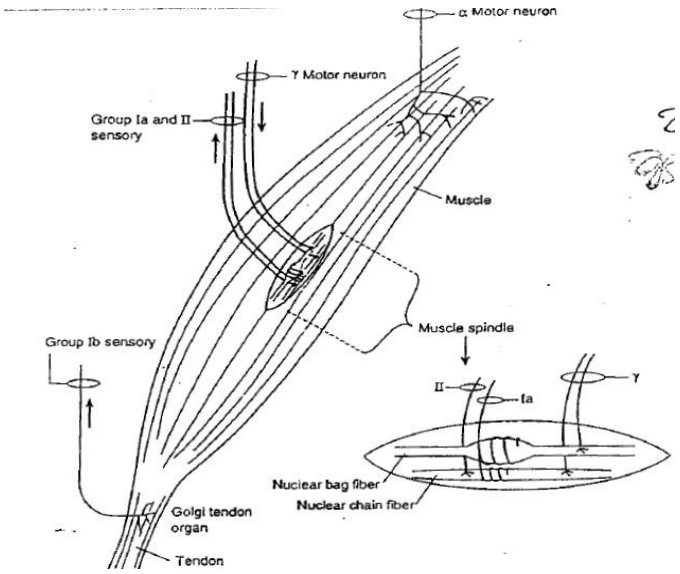
*Higher centers control stretch reflex through affecting gamma, if for some reason gamma is over-stimulated by excessive impulses from reticulospinal or corticospinal tract this would lead to convulsions/rigidity/spasticity (continuous contractions).
→ Gamma is the major controller of stretch reflex and muscle tone.

*Inversion Myotatic Reflex:

So far we have only talked about one type of receptors found in the skeletal muscle which is the muscle spindle (which is in the fleshy part of the muscle), now we will talk about a second type of receptors which is the Golgi tendon organ.

- Golgi tendon organ responds to tension in the muscle that results from contraction or stretch and sends afferent impulses through Ib (unlike muscle spindles which responded to stretch and send impulses through Ia and II)

- In the figure, notice the afferent fibers coming from muscle spindle: primary Ia coming from both nuclear bag and chain, and secondary II coming from nuclear chain only.
-Also, the afferent fibers coming from Golgi tendon organ: "Ib"
- If the muscle is contracting, then the response is faster from **Ib**, since the contraction inhibits the spindle. Whereas Stretch of the muscle activates muscle **spindle**more rapidly than Golgi tendon organ.

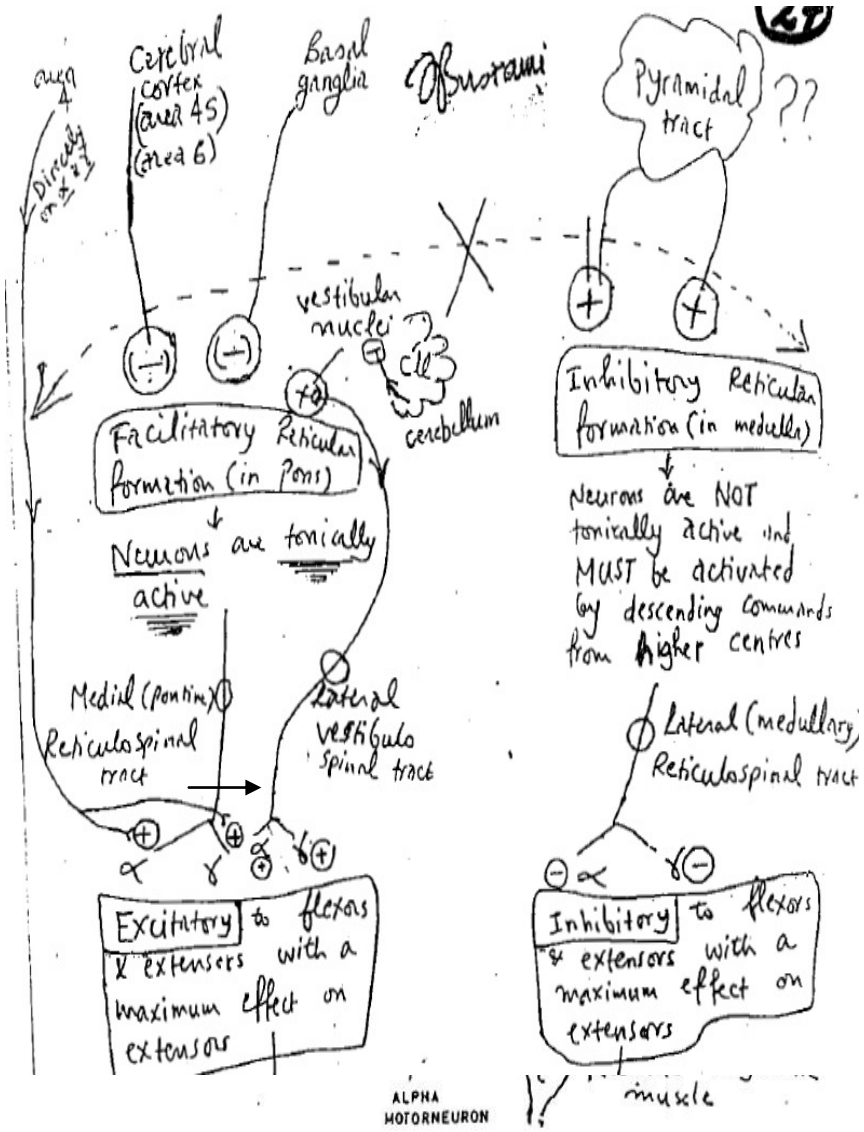


- What happens if we stimulate golgi organ?
Upon stimulation, mainly in response to contraction as mentioned earlier, it sends impulses through afferent Ib to (1) inhibitory interneurons which would inhibit alpha that stimulates the contracted muscle. This is a protective mechanism to prevent tearing of muscle upon high tension. (2) Excitatory interneurons that stimulate the antagonistic muscle.

- Upper motor neuron lesion such as stroke leads to an increase in tone **especially in antigravity muscles**(flexors of upper limb and extensors of lower limb and vertebral column), since they normally have higher tone than other muscles because they're stretched due to gravity.

- Inversion Myotatic reflex underlines the mechanism of the **"clasp-knife " phenomenon** (الموس). In such situations, passive stretching of the spastic muscle will be met with great resistance due to hyperactivation of muscle spindleleading to contraction up to a point after which the muscle gives way suddenly because it activated Golgi tendon organ leading to relaxation. The phenomenon has been termed "clasp knife "by Sherrington.

-Now pay attention to this final diagram:
we will start with the **pontinereticulospinaltract represented on the right:
- In the previous lecture, we mentioned that this tract causes activation of extensors and flexors, with a main effect on extensors.
- Neurons of this tract are tonically active, meaning that they are hyperactive continuously affecting α,γ
-It is inhibited by: 1- area (6) 2- area (4S) 3- Basal ganglia
If there was damage to brainstem between midbrain and pons, what will happen? We lose the inhibition on pontinereticul~~ospi~~nal excessive activati~~on o~~f α,γspasticity of antigravity muscles mainly.



**Now look at the left side of the figure, we have lateral medullary reticulospinal tract:
- It inhibits both α, γ of both flexors and extensors with a main effect on extensors.
- Neurons are inactive unless activated by cortex by pyramidal tract.
- Transection of brainstem between medulla and brain stem, leads to loss of excitation ⟶ the tract is inactive ⟶ loss of inhibition ⟶ excessive activation of extensors.

Conclusions:

- Normally there is a balance between facilitation (pontinereticulospinaland lateral vestibulospinal) and inhibition (medullary reticulospinal) of gamma neurons ensuring that gamma is always active and a continuous stretch reflex.
- Damage to brainstem leads to loss of both facilitation and inhibition to reticulospinal. Loss of facilitation (pyramidal tract to the medullary reticulospinal) is not important because the neurons are not tonically active, but loss of inhibition (cortex and basal ganglia) to the pontinereticulospinal leads to its over-activity and spasticity.
- Medullaryreticulospinal is not active unless activated by the pyramidal tract.
- Pontine reticulospinal is always active and facilitates excitation of extensors.
- Upper motor neuron lesion affects both pyramidal (excitatory) and extra-pyramidal (inhibitory). But spasticity is caused due to loss of extra-pyramidal inhibition.