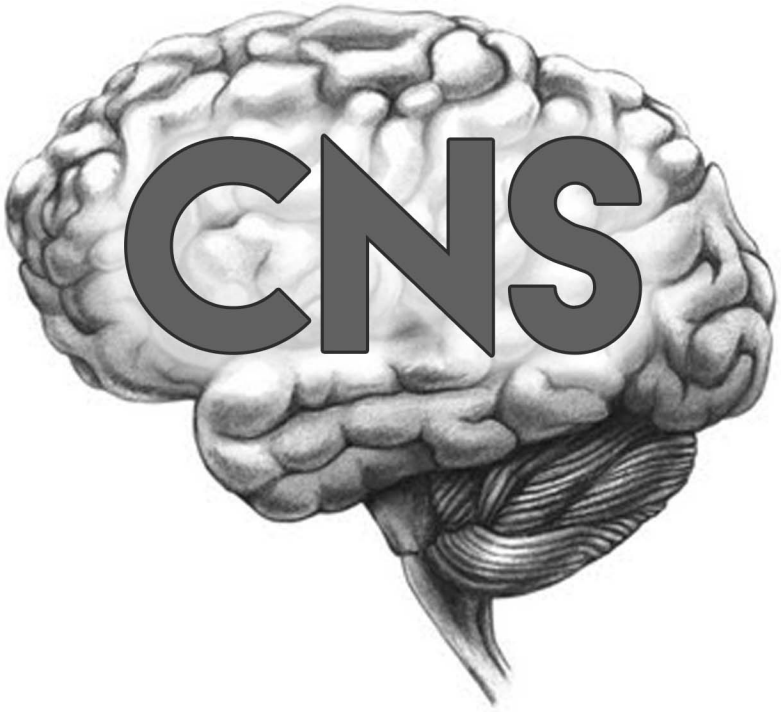

ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

6

Subject

Motor Pathways

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Date: **00/00/2016**

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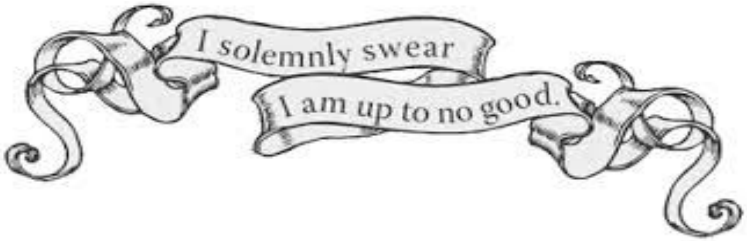


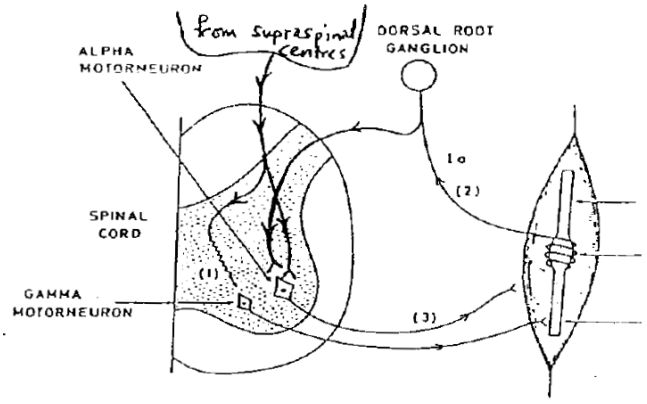
- This sheet has been written according to the record of section 1.
- The Doctor started with a review of the stretch reflex from the previous lecture.

❖ There are two methods by which we induce a stretch reflex in a muscle:

1. Stretching of the entire skeletal muscle, and as a result stretching the muscle spindle (also called the intrafusal muscle fibers). This occurs in all of our muscles as the length of the muscle is less than the distance between the origin and the insertion.
2. Stimulation of the **Gamma** motor neurons, which supply the peripheral contractile part of the muscle spindle causing it to contract and shorten on both sides of the muscle spindle, and stretching the central part (the receptor area). This is important during muscle contraction, because as the muscle becomes shorter the muscle spindle will no longer be stretched, so the action of these Gamma neurons keeps it stretched.

When does the muscle spindle get inhibited? Whenever the muscle contracts that's why we need alpha-gamma co-stimulation(aka :gamma loop) mentioned in the previous lecture.

After the central part of the spindle is stretched in both methods, it will send an impulse through its afferent nerve fibers through the dorsal root of the spinal nerve, and in the spinal cord these afferent fibers will synapse directly with **Alpha** motor neurons that will move through the ventral root and cause the muscle (the extrafusal fibers) to contract, thus maintain the muscle length. This is called a **Monosynaptic reflex**.



And remember there are two ways to stimulate the Alpha motor neurons:

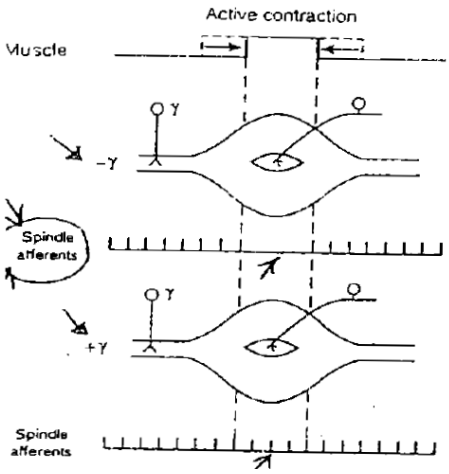
1. From the cortex through the pathways we took previously (corticospinal, corticobulbar …etc.) (the higher centers).

2. The monosynaptic reflex by stretching the muscle or through the gamma motor neurons stimulating the muscle spindle.

The Gamma motor neurons are easier to stimulate than the alpha motor neurons (they are smaller so they reach the threshold faster), making the gamma the first to start the contraction and also the one responsible for maintaining minimal contraction in the muscle.

Always bare in mind that stretch reflex maintains muscle tone & muscle length.

The picture on the right shows the effect of the gamma neurons during an active contraction. If you inhibit the gamma you will notice a loss in the action potential of the afferent nerve fibers leaving the spindle. However, when the gamma is stimulated you find that the action potential is maintained.

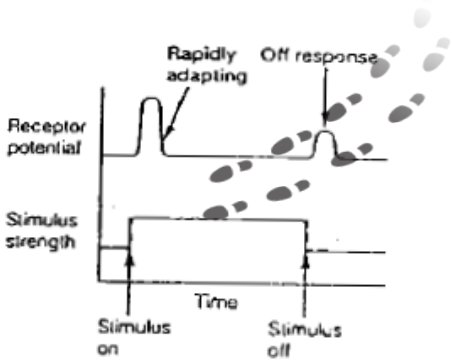


- Function of the muscle spindle:

The muscle spindles and their reflex connections constitute a feedback mechanism which maintains the length of muscles constant, as well as maintaining the muscle tone.

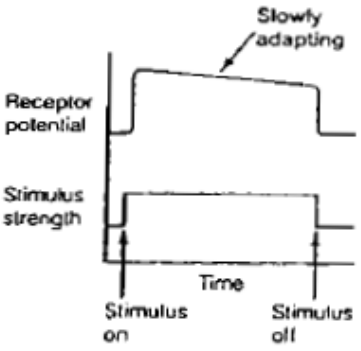
There are two types of afferents leaving the muscle spindle:

1. Primary endings (type Ia).
2. Secondary endings (type II).



Each type has a different response:

- The Primary endings are **rapidly adapting** (Phasic receptors), which means that once you stimulate the spindle you will have a sudden increase in action potential followed by a sudden decrease. This is called a **Dynamic response** because it depends on the increase in the length of muscle fibers, for e.g. when



you hit the tendon with a hammer during a **tendon jerk** (deep tendon reflex test) you are increasing the length of the muscle fibers and you get a fast contraction then a fast relaxation, a dynamic response.

- On the other hand, the Secondary endings are **slowly adapting** (Tonic receptors), meaning that as long as the spindle is stimulated there will be an increase in action potential. This is called a **Static response** because as long as the spindle is stimulated (by the normal muscle stretch) there will be a persistent contraction. This static response is the one responsible for **Muscle tone**.

Primary endings	⟶ Rapidly adapting	⟶ Dynamic response	e.g. Tendon jerk
Secondary endings	⟶ Slowly adapting	⟶ Static response	e.g. Muscle tone

When we talk about skeletal muscle tone we have to differentiate between muscle tone and tetanic contractions.

Tetanic contractions are continuous contractions that occur only in skeletal muscles (never in cardiac muscles) for e.g. when you carry something heavy. On the other hand, muscle tone is a continuous partial contraction involving only a few motor units, so it is not considered a tetanic contraction but rather **sub-tetanic**.

Muscle tone is also **neurogenic**; it depends on the nerve supply to the motor units.

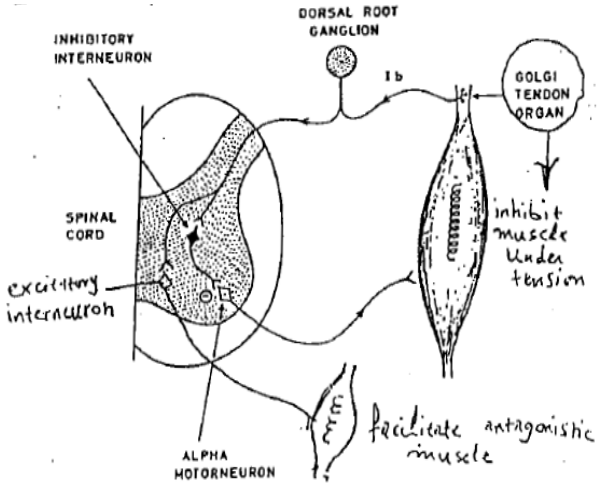
No nerve supply ⟶ No contraction ⟶ No muscle tone

Muscle tone is highest in **anti-gravity muscles**; extensors of the vertebral column, flexors of the upper limbs and extensors of the lower limbs.

❖ Inverse stretch reflex: (maintenance of muscle tension)

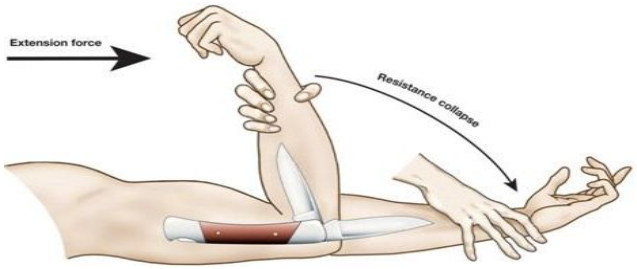
It is a protective reflex that occurs when the muscle is under **severe tension** whether due to contraction or stress.(example: carrying something heavy). This severe tension is produced by either **stretching** OR **contracting** the muscle, and will stimulate a receptor present in the muscle tendon called the **Golgi tendon organ**. The afferents leaving this receptor are of the **Ib type**, they enter the spinal cord and synapse with an **inhibitory interneuron**, which inhibits the **alpha** motor neuron supplying the tensed muscle and causing its relaxation (the complete opposite of stretch reflex).

It is considered a protective reflex because if this relaxation doesn't occur, the severe tension might cause the muscle to tear or rupture.



Because we have interneurons here, it is a **Disynaptic reflex**.

Recall in the stretch reflex we inhibited the antagonist muscle in order to contract the first muscle. In the inverse stretch reflex we stimulate the antagonist muscle and cause it to contract in order to reduce the tension of the first muscle. So now we can conclude that the inverse stretch reflex **maintains muscle tension**, while the stretch reflex **maintains muscle length**. The figure from the handout shows the inverse stretch reflex.



❖ One phenomenon that can be explained by the inverse stretch reflex is the "Clasp knife" phenomenon that occurs in some upper motor neuron lesions (strokes). One of the symptoms of these upper motor neuron lesions is an increase in muscle tone "Hypertonia" of the flexors of the upper limbs and the extensors of the lower limbs (the anti-gravity muscles), so when you approach these patients you find that their elbows and hands are flexed and their knees are extended. If you try, for example, to extend the elbow, you will notice that there is an initial resistance to the extension and then suddenly this resistance is gone and the muscle gives way for the elbow to be extended. This is called **"Clasp knife"**,

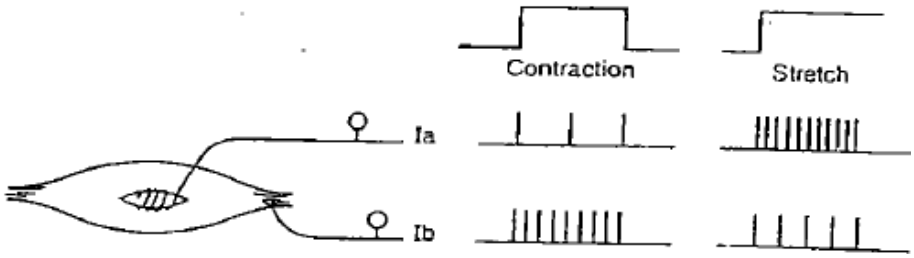
Wikipedia
Clasp-knife response refers to a stretch reflex with a rapid decrease in resistance when attempting to flex a joint, usually during a neurological examination. It is one of the characteristic responses of an upper motor neuron lesion.

named by a scientist called Sherington because of its similarity to the action of a jack knife.

- But why does this phenomenon occur?

When you first apply pressure on the arm, you are applying pressure on the tendon of the contracted muscle causing it to stretch, and therefore activating the stretch reflex that will cause the initial resistance. After a while of applying pressure the tension in the muscle will increase and will be enough to activate the Golgi tendon organs in the muscle tendon, activating the inverse stretch reflex which will cause the relaxation and the muscle gives way for the desired movement to occur.

- The figure below compares the effects of both contraction and stretch on the Ia fibers (from the muscle spindle) and on the Ib fibers (from the Golgi tendon organ). Ia responds largely to stretch and minimally to contraction, while Ib mainly responds to contraction and to a lesser extent to stretch.



Muscle spindle: responds better to stretch, gets inactivated by contraction. (Maintenance of muscle length).

Golgi tendon organ: responds better to contraction. (Maintenance of muscle tension).

- ❖ Both the pyramidal and extrapyramidal tracts start in area 4 and area 6 in the cortex; the pyramidal mainly in area 4 and the extrapyramidal mainly in area 6. And we already mentioned that both the pontine and medullary reticulospinal tracts start apparently in the reticular formation in the pons and the medulla respectively, but they receive orders from area 6 making them corticoreticulospinal, and the same with the rest of the extrapyramidal tracts.
- ❖ Remember that one of the most important pathways in the extrapyramidal tract is the reticulospinal tract, which is divided into medial pontine, which stimulates mainly the extensors and to a lesser extent the flexors, and lateral medullary, which inhibits mainly the extensors and to a lesser extent the flexors. And keep in mind

that the extrapyramidal tract is in the medial motor system which affects alpha and gamma motor neurons of the axial and proximal muscles needed for posture and walking .

IMPORTNANT: The pontine reticulospinal tract arises from the **facilitatory reticular formation** in the pons, here the neurons are **tonically active**, meaning they are continuously firing and activated just like the alpha motor neurons (we reduce the activity of alpha neurons through **Renshaw cells**). Now how do we reduce the activity of the tonically active neurons that the pontine reticulospinal tract arises from?

Wikipedia
Renshaw cells are inhibitory interneurons found in the gray matter of the spinal cord, and are associated in two ways with an alpha motor **neuron**. They receive an excitatory collateral from the alpha **neuron's** axon as they emerge from the motor root, and are thus "kept informed" of how vigorously that **neuron** is firing.

By three areas in the higher centers and they are:

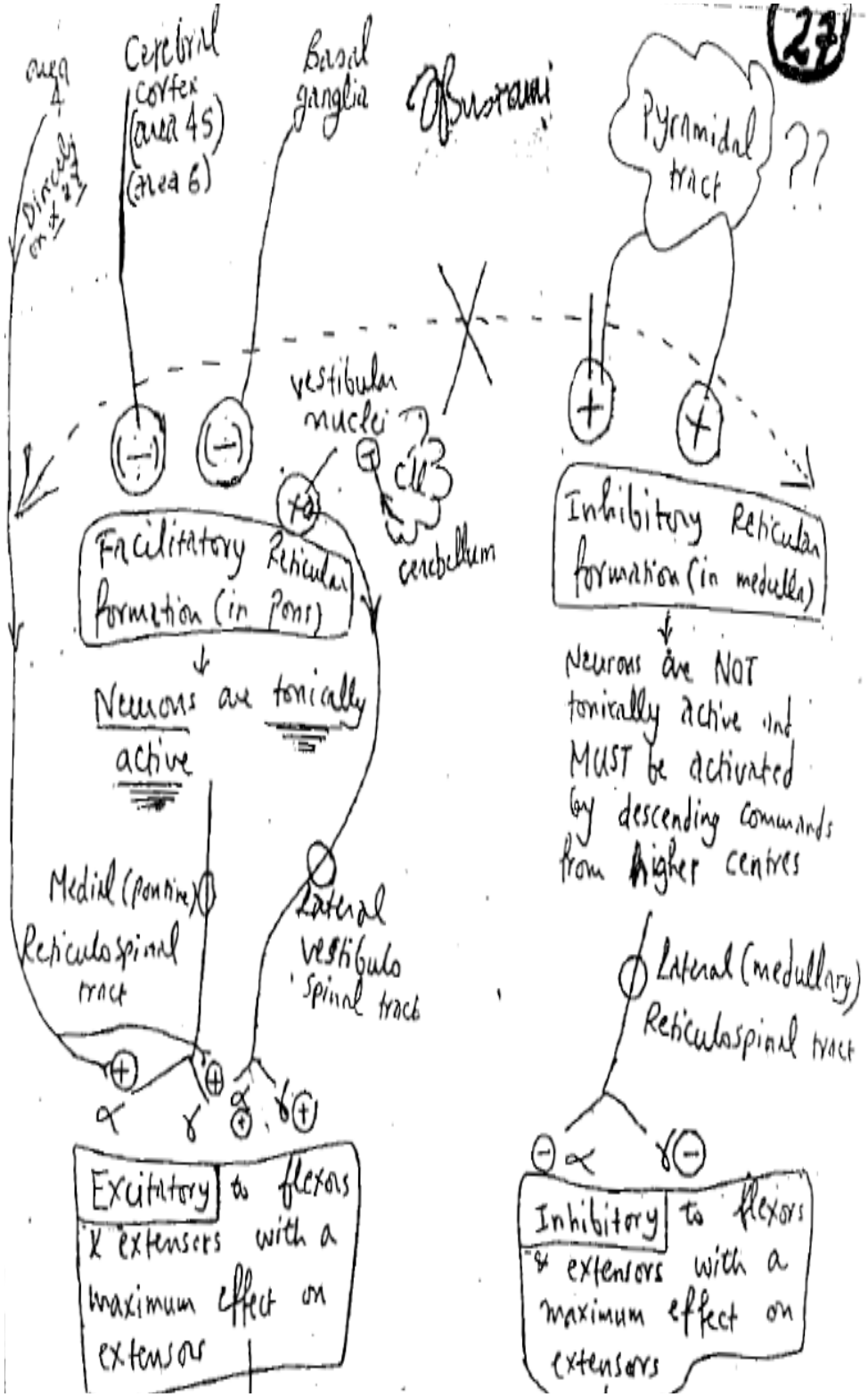
1. Area 6 (mainly).
2. Basal ganglia.
3. Area 4s ("s" for "suppression", and it is the anterior part of area 4).

In the medullary reticulospinal tract however, the neurons are NOT tonically active, they need stimulation and they receive this stimulation from the pyramidal tract. So the neurons of the medial pontine tract are normally under **inhibition**, whereas the neurons of the lateral medullary tract are normally under **stimulation**.

In normal conditions, both of these tracts are functioning and keeping a balance; pontine increasing the tone and the medullary decreasing the tone. The result of this balance is seen through keeping the action of the **gamma** motor neurons within normal ranges, why gamma? Obviously, because they are the ones acting on the stretch reflex, thus ensuring proper activation of the stretch reflex and normal muscle tone (normal postural tone due to proper activation of anti-gravity muscles).

Now if we cut the connection between the pontine tract and the higher centers, which are inhibitory to the tract, we will have an increase in the activity of the pontine tract and we will have **hypertonia** of the muscle (due to increased stimulation of gamma neurons), this is called disinhibition or release of inhibition. On the other hand, if we cut the connection between the medullary tract and the pyramidal tract, the medullary tract will stop functioning and it will **no longer affect the muscle tone**.

Remember that along with the pontine reticulospinal tract, there is another tract with same function (increasing the tone of the extensors) and that is **the lateral vestibulospinal tract** (recall it is inhibited by the cerebellum). But for the time being forget about this tract, we will come to its importance later on.

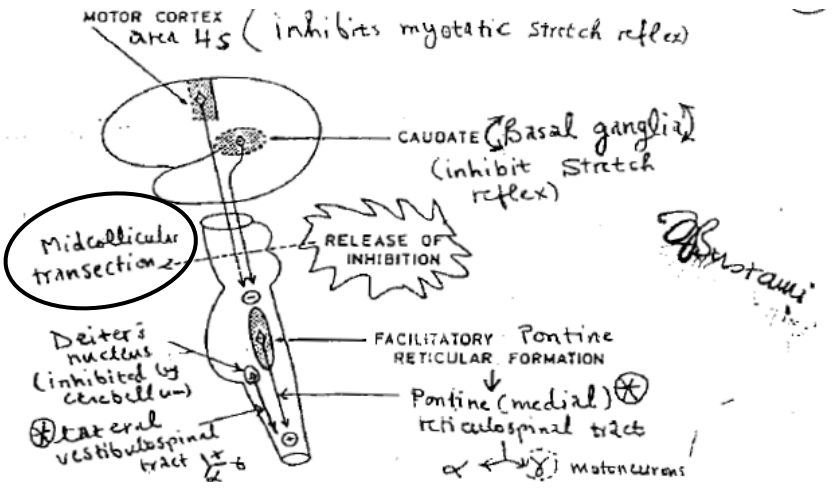


- Oh this is just a picture of my dog chilling in the driver's seat, and congrats you're about to skip an entire page in less than a minute!

Anyway, back to our point!

In an experimental animal, we can cut the midbrain in the middle (between the superior colliculus and the inferior colliculus of the tectum) this is called **Midcollicular transection** or **Midbrain transection** (or we can block the blood supply to this area instead by blocking **the basilar artery**). This means we have separated the higher centers from both the pons and the medulla. So again, you get disinhibition of the medial pontine tract, stimulation of alpha and gamma, gamma causing hypertonia of the muscles (spasticity or rigidity) and this resulting condition is called **decerebrate rigidity**.

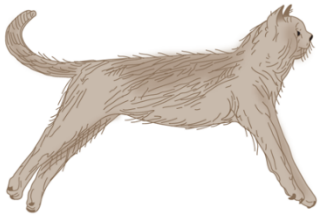
- In this figure, the medullary tract is not mentioned because it had lost its effect on the muscles (as if it doesn't exist) as we have said earlier.

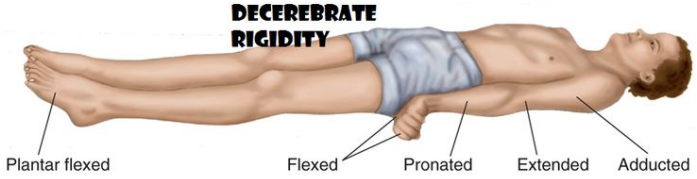


- How do we know that the gamma motor neurons are the ones causing this rigidity in the muscles?

We make a cut in the dorsal root of any affected spastic muscle in the experimental animal, and by that we are cutting the Ia fibers coming from the muscle spindle, then we will notice that the muscle had lost its rigidity, so the cause of the rigidity came from the muscle spindle, and since the muscle spindle is stimulated by gamma motor neurons then it's now clear to us that the gamma is the one causing the decerebrate rigidity.

Animals and humans differ in their anti-gravity muscles, in cats, for example, they are in the forelimbs and the hindlimbs, in humans as you know they are in the flexors of the upper limb and mainly the extensors of the lower limb, and the effect of decerebrate rigidity mainly affects the extensors, which gives a different appearance between animals and humans.



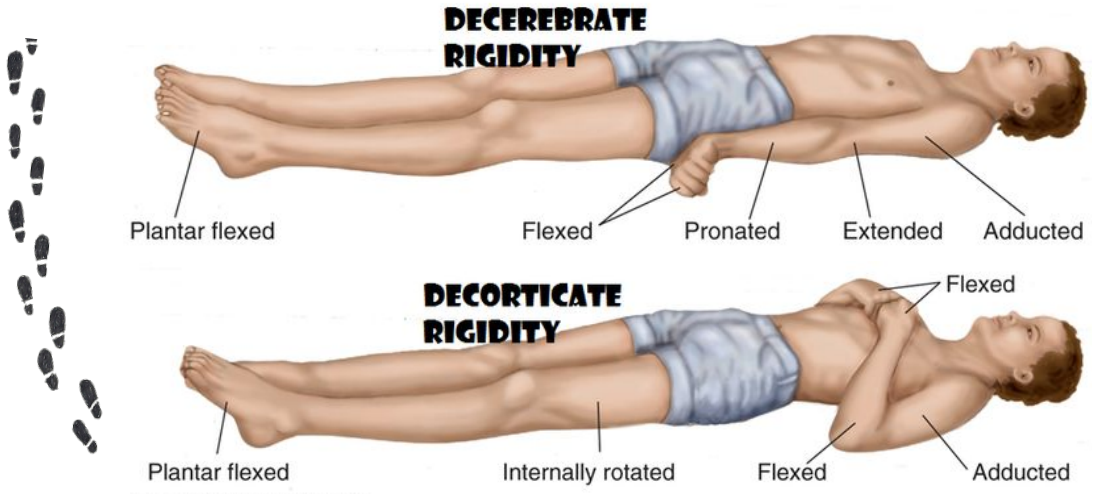


- Can we create an alpha rigidity?

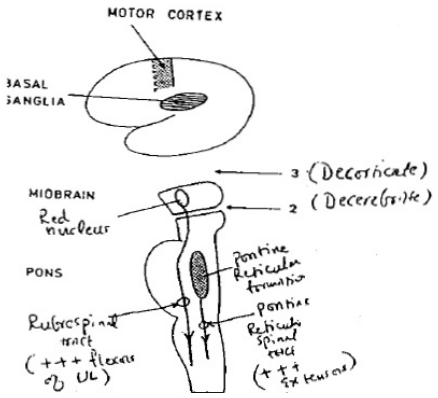
Yes, after cutting the midbrain (or blocking the basilar artery)and causing the decerebrate rigidity, we cut the dorsal arteries of the muscles and remove the gamma rigidity thus reducing the muscle tone back to normal, so all we need to do now is to excite the alpha neurons, this is established by the lateral vestibulospinal tract (yes now is the time to remember it). We release the lateral vestibulospinal tract from its inhibition by removing the cerebellum (recall that the lateral vestibular nucleus or Deiter's nucleus is inhibited by the cerebellum). So now this tract will stimulate both alpha and gamma motor neurons, but since the dorsal roots are cut this means that stimulating the gamma is useless, however stimulating the alpha will increase the muscle tone and cause **alpha rigidity**.

Keep in mind that "cutting" means we are referring to experimental animals, however when we talk about humans we're not going to cut the midbrain in order to observe the decerebrate rigidity. It rather occurs when a problem causes blockage of the blood supply(basilar artery), a tumor, or even multiple sclerosis affecting the midbrain, causing medial pontine disinhibition, stimulation of alpha and gamma, gamma causing hypertonia of mainly the extensors resulting in decerebrate rigidity.

In **stroke** patients, the **internal capsule** (above the level of the midbrain) is usually affected by blockage of its blood supply, and this results in a condition called **decorticate rigidity**, where there is hypertonia of flexors of the upper limbs and extensors of the lower limbs, as opposed to decerebrate rigidity, where we find extension of both the upper limbs and the lower limbs.



- Why do these two cases differ in the appearance of the upper and lower limbs?



In the decerebrate rigidity, the damage occurs below the red nucleus, which means that **rubrospinal tract** is affected by this damage and thus inhibited (recall this tract arises from the red nucleus and causes stimulation of the flexors, mainly in the distal muscles but also the proximal muscles), while in decorticate rigidity, the damage is above the level of the red nucleus, so the rubrospinal tract remains active causing the flexion seen in the upper limbs.

- Which of these two cases is more dangerous (has a worse prognosis)?

The decerebrate rigidity is more dangerous, and that is because it means there is a problem in the blood supply to the midbrain, which is closer to the medulla than the internal capsule (in the case of decorticate rigidity). So the problem that caused the decerebrate rigidity is more likely to affect the blood supply of the medulla (recall it contains the respiratory centers), which will cause respiratory failure and death.

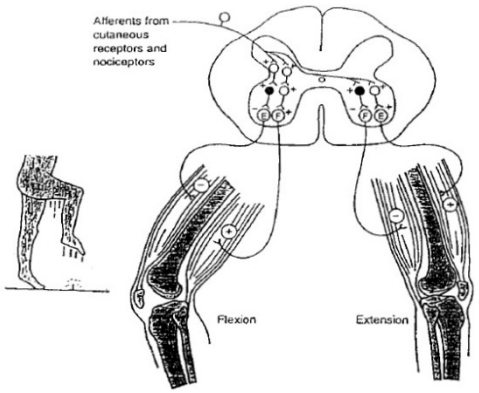
- Patients in both cases are in **comatose state**.
- A comatose patient can alter from decerebrate to decorticate rigidity if the damage spreads to a higher level.

❖ Another important reflex is the **Flexion reflex** (also called **Withdrawal reflex** or **Flexion-crossed extension reflex**):

This reflex occurs when you **step on a sharp object**, or **high temperature** by accident (pain stimulation) which causes flexion of the knee of the affected limb (protective; to keep the foot away from the pain stimulus) and extension of the knee of the contralateral limb (so that this limb can hold your body weight off the ground and prevent you from falling), and that's why it's also called withdrawal reflex (for protection) and flexion-crossed extension reflex (flexion in one limb and extension in the other). It also occurs during the normal **walking mechanism**, as it involves flexion of one knee and extension of the other.

- If you look at the figure below, you will notice that this reflex is **Polysynaptic**; receptors in the skin, joints, or muscles send impulses through afferent fibers, these fibers are of types **II**, **III**, and **IV**, these fibers are collectively called **flexor reflex afferents (FRA)** and they enter the spinal cord through the dorsal root. The

FRA will first synapse with inhibitory interneurons that will inhibit the alpha motor neurons of the antagonist muscle of the same limb through other interneurons and cause the muscle to relax (Quadriceps femoris)*, then after the antagonist muscle is relaxed the FRA stimulates excitatory interneurons that activate the alpha motor neurons of the flexor muscles of the limb through other interneurons and cause them to contract (the Hamstrings)*. At the same time, the FRA are doing the opposite to the flexors and extensors of the other limb.



*Remember from the musculoskeletal system that quadriceps femoris is the main extensor of the knee joint, and the hamstring muscles are the flexors.

- These excitatory and inhibitory interneurons in this polysynaptic reflex, are thought to be under the control of the pyramidal tract (remember it's affected by area 4). If there is damage to the pyramidal tract, the function of these interneurons will be affected; the excitatory interneurons become inhibitory and the inhibitory become excitatory.

The doctor didn't mention many of the details written in the handout about this topic, so I added the description in the handout here just in case.

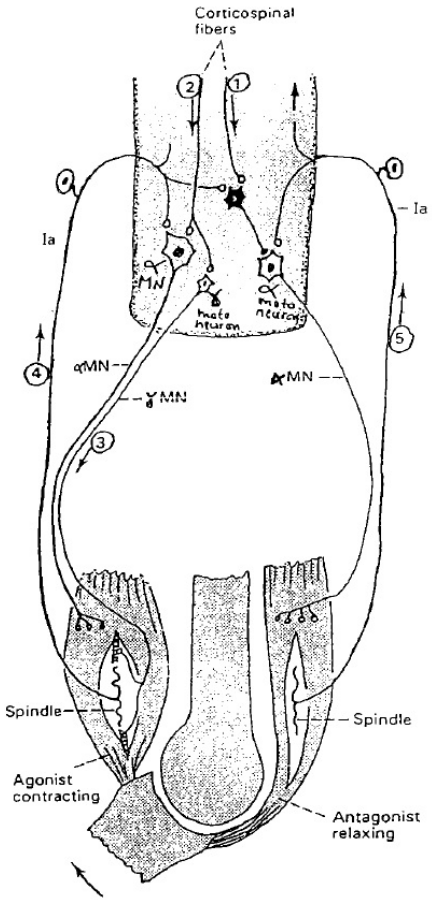
Flexion reflexes are important in a number of behavioral patterns; e.g., flexion of limbs is part of the activity involved in walking. One of the most obvious functions of the flexion reflex is withdrawal of a limb from painful, noxious stimuli. Hence the flexion reflex is frequently called the withdrawal reflex. Also, since flexion of the limb ipsilateral to the stimulus is usually accompanied by an extension of the contralateral limb(s), this reflex is also referred to as the flexion-crossed extension reflex.

The flexion reflex can be initiated by activity in afferent fibers from a variety of sensory receptor organs. These sensory receptors may be in the skin, in muscle, and in joints and involve afferent fibers II, III, and IV; collectively, these are called flexor reflex afferents (FRA). The degree of flexion response can vary from a flexor twitch in response to relatively innocuous stimulation to a complete withdrawal of the limb from a noxious stimulus. A very strong stimulus to the FRA fibers results in activity of all four limbs. This response is mediated via intersegmental connections and is sometimes referred to as irradiation of the stimulus; the stronger the stimulus, the more extensive is the reflex reaction.

The flexion reflex is polysynaptic (Fig. 11-7). The afferent fibers enter the spinal cord and excite interneurons of the dorsal horn. The interneurons then act on alpha motor neurons through relay pathways involving other interneurons. The response is an excitation of alpha motor neurons to the flexor muscles and inhibition of alpha motor neurons to the extensor muscle of the stimulated limb (ipsilateral). In addition, this is frequently accompanied by excitation of alpha motor neurons to extensor muscles and inhibition of flexors to the contralateral muscle. This behavior is the appropriate response to painful stimuli; for example, if a person steps on a sharp object, the injured foot is withdrawn (flexion), while the other limb of the pair is extended, thereby providing support for the body and preventing the person from toppling.

❖ Sequence of events in voluntary movement

If we want to start a voluntary movement, for example, flex the knee joint, we have to first relax the antagonist muscle through an inhibitory interneuron, which brings us to the first step:



1. The corticospinal tract will activate **Ia inhibitory interneurons** which inhibit the alpha motor neurons supplying the antagonist muscle causing the muscle to relax (Quadriceps femoris).
2. The corticospinal tract directly activates both alpha and gamma motor neurons supplying the agonist muscle (the Hamstrings).
3. This causes contraction of both the extrafusal (the muscle bulk, through alpha stimulation) and intrafusal muscle fibers (muscle spindle, through gamma stimulation).
4. As a result of stimulation of the muscle spindle, there will be feedback from the muscle spindle that increases the alpha motor neurons excitation of the agonist muscle, and increases Ia interneuron inhibitory function on the antagonist muscle.
5. Due to the contraction of the agonist and flexion of the knee, there will be an increase in passive stretch of the antagonist muscle (Quadriceps) which stimulates its muscle spindle. When the spindle sends feedback through Ia fibers to the alpha motor neurons of quadriceps, these alpha neurons will still be in their **refractory period** (the phase of the action potential when the neuron doesn't respond to any stimulus), so the alpha neurons won't respond, and instead the impulse is transmitted to the higher centers.

- **Alpha-gamma coactivation** or **linkage**: the alpha and gamma motor neurons are stimulated together in voluntary movement.
- The **Gamma loop** constitutes of gamma motor neurons, Ia afferent fibers, and alpha motor neurons. Through this loop, the gamma motor neurons reinforce the alpha excitation by activating the muscle spindle.

❖ The sheet is finally over. Please note that this was my first attempt in writing a sheet and I give you my deepest apologies for any mistake I may have made that went unnoticed. Now that I got that off my chest, allow me to send a shout out to these wonderful friends in my life: Deena Younis, Ahmad AlHadidi, Sarah Jamos, Abdullah AlSaa'd, Besher Darweesh, Hanin Saleh and the rest of the wonderful people in the squad <3