

# PHYSAOLOGY

SHEET NO. 12 WRITER : Doctor 018 CORRECTOR : Rawan Fratekh DOCTOR : Faisal Mohammad Each type is supplied as we said by sensory neurons.

- 4 The nuclear bag fiber is supplied with group 1a fibers (large myelinated) which are considered the primary afferent fibers (called annulospiral) and dynamic.
- **the nuclear chain fiber** is supplied with group 2 fibers which are considered secondary afferent fibers (called flowerspray) and static. [it is supplied also by primary afferent fibers]

They're also supplied by Gamma motor neurons: *dynamic* fibers only supply the nuclear bag, while static fibers supply both.

There are two ways to activate a muscle spindle (stretch receptors):

- 1- Any stretch in the muscle that lengthens it will stimulate the center of the muscle spindle.
- 2- Activation of gamma motor fibers will contract the contractile parts of the muscle spindle stimulating and keeping it stretched.

#### Static Response of the Muscle Spindle

When the center of spindle is stretched slowly - the number of impulses generated by the primary and secondary endings increases in proportion to the degree of stretch. This is the 'static response'.

Function of the static nuclear bag and nuclear chain fibers.

#### Dynamic Response of the Muscle Spindle

When the center of the spindle is stretched

Motor Sensory Motor 11 la ά γ Y 14 µm 5 µm 17 µm 8 µm/5 µm Alpha motor Sheath Primary Extrafusal fibers ending ending Intrafusal Gamma motor Fluid Secondary cavity ending ending fibers -1 cm Static y fiber Dynamic y fiber Group la fiber (efferent) (efferent) (primary afferent) Group II fiber Plate ending (secondary afferent) Nuclear bag fiber (intrafusal muscle)

Nuclear chain fiber (intrafusal muscle)

Figure 54-3

Trail ending

Details of nerve connections from the nuclear bag and nuclear rapidly - the number of impulses generated by the chain muscle spindle fibers. (Modified from Stein RB: Peripheral control of movement. Physiol Rev 54:225, 1974.)

primary endings increases inproportion to the rate of change of the length. This is the 'dynamic response'.

Function of the <u>Index nuclear</u> bag fiber ONLY.

#### Why is it important to sense the rate (speed) of the change of the length?

When our nervous system knows the rate (speed) of change of the length and the distance we want to go through, it will calculate the time predicted to cut this distance and it will stop when we reach the intended place.

Imagine someone having a problem or damage in the muscle spindles, making his nervous system unable to know the rate of change of the length and therefore can't predict when he will reach the intended place, so if he wanted to reach a wall far from him 10m, he will not stop when he finishes those 10m instead he will stop when he hits the wall.

#### Let's study the difference between group 1a fibers (dynamic) and group 2 fibers (static)



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- Normally both types of receptors (nuclear bag and nuclear chain) have <u>basal rate offiring</u> and that's important because it makes positive and negative (inhibition) control possible. When the basal rate is 100 impulse/sec we can increase it to 200 impulse/sec or decrease it to 50 impulse/sec. If there is no basal rate (equals zero), we can't decrease the rate of firing.
  - $\checkmark$  In the first case we have a dynamic change in it then it goes back to static :
    - During the dynamic change the rate of firing in group 1a fibers (dynamic) increases a lot [when rate of stretch increases, rate of firing increases], while in group 2 fibers (static) the rate of firing increases but to a lesser extent than in group 1a → positive control
    - Now back to static stretch, group 1a fibers decrease their rate of firing returning to the basal rate, while with group 2 fibers firing rate stays almost the same.
- ✓ When there is a release of stretch (stretch decreases), the rate of firing in group 1a fibers will decrease a lot, while in group 2 fibers will decrease to lesser extent stayingalmost basal → negative control
- ✓ How much increase or decrease in firing rate depends on the rate of change.

Something important to know before we proceed is that when the muscle is stretched, the muscle spindle will sense that stretch whether it was static or dynamic and those sensory neurons will go back to the spinal cord and to the brain telling it about this change (as we explained earlier), the same sensory neurons will synapse in the spinal cord with interneurons and those interneurons synapse with alpha motor neurons that supply the extrafusal muscle fibers contracting them that's because we don't want the muscle to stretch (increase in length) more than normal, contraction will shorten the muscle preserving it. Another explanation we took in anatomy, is that the muscle length in rest is shorter than its origin and insertion, which means that when the muscle is between its origin and insertion it is stretched – that stretch will stimulate muscle spindles which in turn will stimulate alpha motor neurons keeping the muscle contracting almost all the time maintaining its tone which helps us against the gravity.

#### Physiologic Function of the Muscle Spindle

- 1- Comparator of length between the intrafusal and extrafusal muscle fiber.
- 2- Opposes a change in length of the muscle.
- 3- When the muscle is stretched the spindle returns it to its original length.
- 4- Leads to the stretch reflex.

If the muscle spindle was de-innervated, we'd still be able to contract and relax the muscle [extrafusal fibers, but we wouldn't be able to *sustain* the contraction.

#### Smoothening effect of the Muscle Spindle

# Now, let's get things together by talking about a phenomenon known as **alpha- gamma** coactivation

When the muscle is going to contract, alpha fibers must be stimulated. Sustained contraction will cause muscle spindle to become loose not sensing any change in stretch. Here comes the role of gamma fibers (which are **co-stimulated** with alpha fibers) causing contraction in the contractile parts of the muscle spindle stretching it (the same cycle is repeated | muscle spindle senses that stretch -> sends it back to the spinal cord -> activating alpha motor fibers allowing it to sustain the contraction)

Let's think about having those muscle spindles de-innervated (no gamma motor neurons are there) we will lose the stretch in the intrafusal fibers, no feedback stimulation for alpha motor fibers (inhibition), no sustained contraction, the contraction will lead to relaxation after it [the green graph].



#### Figure 54–5

Muscle contraction caused by a spinal cord signal under two conditions: curve A, in a normal muscle, and curve B, in a muscle whose muscle spindles were denervated by section of the posterior roots of the cord 82 days previously. Note the smoothing effect of the muscle spindle reflex in curve A. (Modified from Creed RS, et al: Reflex Activity of the Spinal Cord. New York: Oxford University Press, 1932.) In this experiment, they attached a load to the muscle which will stretch the muscle and the muscle spindle in it, and they recorded the rate of firing in 1a sensory fibers.

### What happens?

1a fibers [sensory from the nuclear bag to the spinal cord] will sense the stretch and activate alpha motor fibers causing the muscle to contract and shorten. This will also shorten the muscle spindle and deactivate the stretch receptors. However, gamma motor fibers will be activated and they will contract the muscle spindle. Because of the unique arrangement of sarcomeres and nuclei in the muscle spindle, the peripheral parts will contract while the middle [which contains only nuclei] will stretch, reactivating the stretch receptors and sustaining the contraction of the muscle. <u>Alpha-gamma coactivation helps to maintain muscle contraction. Without this muscle spindle spindle innervation through the coactivation, contraction can't be maintained.</u>



In A we are recording a (static) stretch, there is basal firing in muscle spindle sensory fibers, without any change in the length of the muscle (isotonic).

- In B we are recording change in stretch (dynamic) but only alpha motor neurons are stimulated, that means we have contraction in the muscle, but notice there is no firing in the muscle spindle sensory fibers (why?) because muscle contraction will shorten muscle spindles loosening and inhibiting them, <u>without the presence of gamma</u> fibers there will be no recording of any tension sensed by muscle spindle.
- In C we are recording change in stretch (dynamic) with alpha and gamma neurons stimulated together (coactivated), muscle spindle will be contracted and activated all the time, when the muscle contracts there will be a decrease in the firing rate of muscle spindles. (<u>There is always a</u> <u>feedback to the CNS about the degree and change</u> in stretch even in the case of contraction)





increase in the firing rate then it goes back to normal [adapt]. When the load is released (the stretch is decreased), there's no firing. This happens in phasic receptors [on/off].

In the static fibers [group II], the rate of firing changes [increase and decrease] but it never reaches zero.

#### Function of the Gamma System:

- 1- Spindle is normally tonically active as a result of input from higher brain centers. Alpha-gamma co-activation helps maintain muscle contraction.
- 2- Controls the intensity of the stretch reflex.
- 3- Performs a damping function by adjusting sensitivity.

#### Effect of gamma motor fibers (Dynamic and static)

When there is stretch change (dynamic), this will stimulate static gamma fibers increasing their impulse rate and stimulate dynamic gamma fibers increasing their impulse rate more. And when this change finishes, they will go back to their basal rate. Just like what happens with group 1a and 2 sensory fibers. But the increase and decrease of the firing rate in dynamic gamma fibers is more pronounced than in static gamma fibers.



#### Control of the Gamma Motor System (Fusimotor System)

- Gamma signal excited by the bulboreticular facilitatory area of the brain stem.
- Secondarily by areas that send impulses to this area. [cerebellum, basal ganglia, cortex]
- Little is known about the precise control of this system.
- **11 |** Page

Reflexes in general are very important in testing the functions of the spinal cord because they happen mainly within the spinal cord. so, when we test the reflexes, we're actually testing the integrity of the spinal cord.

Reflex Arc

**3** INTEGRATING CENTER

motor neurons)

(one or more regions within the CNS that relay impulses from sensory to

Interneuron

It is true that those reflexes work through spinal cord, but they can be affected by the cortex, to avoid the effect of the cortex we ask the patient to hold his hands together and keeps holding them until we finish to distract him.

#### A reflex is a rapid automatic (involuntary) movement upon a specific stimulus.

#### Reflex Arc:

for any reflex to occur it needs the following parts:

1-receptor

2-sensory (afferent) neuron

3-interneuron (integrating center)

4-motor (efferent) neuron

5-effector

Let's start with our first reflex:

#### Stretch reflex

Notice that the start of any reflex which is the stimulus isn't part of the reflex arc and it can be any type of sensation (pain, touch...)

(axon conducts impulses from

tegrating center to effector)

4 MOTOR NEURON

2 SENSORY NEURON

(axon conducts impulses from receptor to integrating center) **1** SENSORY RECEPTOR

(responds to a stimulu

by producing a genera

or receptor potential)

5 EFFECTOR

(muscle or gland that

responds to motor nerve impulses)

This reflex can be done with almost any muscle, as an example we will focus on the **knee jerk reflex**. Jerk means something abnormal or can't be expected. / other examples: ankle reflex [gastrocnemiusmuscle], biceps, triceps....

In this reflex we will test the movement of the quadriceps muscle around the knee.

We start by hitting the patellar tendon with a hammer -> this will create an artificial **stretch in the quadriceps muscle** -> the stretch is sensed by the muscle spindles found inside the quadriceps muscle -> the afferent sensory fibers connected with these receptors are going toward the spinal cord to:

1- send information about the stretch to higher centers (brain) and, as we know from the previous sheet, these spinocerebellar fibers are responsible of telling the cerebellum [feedback] what is exactly happing down on the level of the muscle regarding the tension (length). 2- Synapse with -> alpha motor fibers that are going to the quadriceps muscle -> the quadriceps muscle will contract -> the knee is extended (moves forward)

➔ Index of the facilitation of the gamma efferents. [recall that whenever there is muscle contraction this will shorten the muscle spindles inhibiting them. And gamma motor efferents will stretch the spindles again activating them]

→ Cortical lesions usually increase muscle stretch reflexes [specifically UMNLs]

 In the pictures to the right, you can find that the antagonistic muscle (hamstring muscle = flexor) is inhibited by the same sensory afferent neurons synapsing with inhibitory interneurons. And that's phenomenon is called reciprocal inhibition.

<u>When the agnostic muscle</u> <u>contracts, the antagonistic muscle</u> <u>relaxes and vice versa.</u>

# Special features of stretch reflex:

 Causes contraction of a skeletal muscle in response to stretching of the muscle.

Patellar or knee-jerk reflex: Stretching of a muscle  $\rightarrow$  activation of muscle spindles  $\rightarrow$  sensory neuron  $\rightarrow$  spinal cord  $\rightarrow$  motor neuron  $\rightarrow$  muscle contraction. (**Excitatory** reflex)

- 2- Monosynaptic reflex.
- 3- **Ipsilateral**. [when you hit the right tendon, the right knee will extend]
- 4- Receptors are in the same muscle stimulated by lengthening of muscle (stretch).



#### Tendon reflex

Keep in your mind that this reflex is **protective**. Because when the muscle is contracted a lot, this creates a very high stretch on the tendon, so we must inhibit this contraction to keep the tendon safe without torn.

Contraction of a muscle causes too much tension in the tendon which might separate the tendon from the tibia -> Golgi tendon organs (receptors) sense this stretch -> the sensory afferent neurons (group

1b fibers) connected with these receptors will go back to the spinal cord to:

- send information to the brain about the tension and the rate of change in tension [static/dynamic]
- 2- synapse with 1. inhibitory interneurons that are synapsing with alpha motor fibers -> relaxation in the muscle (tension in the tendon decreases), 2. excitatory interneuron that activates alpha fibers that innervate the antagonistic muscle causing its contraction

Inhibitory MOTOR NEURON EFFECTOR inhibited (muscle attached G SENSORY 0 to same tendon) relaxes and excited .... relieves excess Increased tension tension stimulates SENSORY **RECEPTOR** (tend Spinal nerve Excitatory Within INTEGRATING interneu CENTER (spinal cord), sensory neuron activates inhibitory interneuron Antagonisti muscles Motor neuron to antagonistic muscles is excited **The Tendon Reflex** 

# special features of tendon reflex:

#### 1- Polysynaptic reflex. (Di-synaptic)

- Control muscle tension by causing muscle relaxation when muscle tension is great.
  ↑ Tension applied to the tendon → tendon organ stimulation → nerve impulse → spinal cord → motor neuron causes muscle relaxation and relieves tension (inhibitory reflex)
- 3- Sensory receptors- Golgi tendon organs (same muscle stimulated by tension applied on the muscle in series with muscle fibers).



Just read ⓒ Golgi Tendon Reflex: Mediated by the Golgi tendon organ receptor located in the tendon. This receptor responds to tension. When the tension becomes too great the reflex inhibits the motor fibers



3 | Page

## Notes on stretch reflex and Golgi tendon reflex:

- Stretch reflex is faster than tendon reflex that's because stretch reflex is monosynaptic while tendon reflex is polysynaptic
- Both work in the same segment where the interneurons enter the spinal cord [unisegment]
- Reciprocal inhibition is present in both
- Transmission of Stretch Information to Higher Centers: Muscle spindle and Golgi tendon signals are transmitted to higher centers. This informs the brain of the tension and stretch of the muscle. Information is transmitted at 120 m/sec. Important for feedback control of motor activity.
- <u>Golgi tendon organs are in series</u> with muscle fibers [tension]
  While <u>muscle spindles are in parallel</u> with muscle fibers [length/stretch]
  Flexor (withdrawal) reflex

A **painful** stimulus causes the limb to automatically withdraw from the stimulus.

Here we start with pain receptor (nociceptor) activation, this receptor is present in the skin -> through C and A $\delta$  fibers (slow fibers) this sensation will be transmitted to the spinal cord -> they go up or down (one or two segments) then they synapse with interneurons -> interneurons synapse with alpha motor fibers activating the flexor muscles [group of muscles]-> flexion of the leg. [there is also inhibition of the extensors in the same leg / reciprocal innervation]

nociceptor activation transmitted to the spinal cord -> synapses with pool of interneurons that diverge the to the muscles for withdrawal, inhibit antagonist muscles, and activate reverberating circuits to prolong muscle contraction -> duration of the after discharge depends on strength of the stimulus

what is after discharge? EPSP stays for 20ms while AP occurs within <1ms // if one stimulus gives us an ESPS that is above the threshold generating AP, it will continue producing this AP for 20ms, it is one way to prolong the impulse.

# special features of flexor (withdrawal) reflex:

- 1- Polysynaptic reflex [slower than stretch reflex and tendon reflex]
- 2- Ipsilateral.
- 3- Multi segmental
- 4- The receptor isn't present in the same muscle! It is in the skin. Stepping on a tack (stimulus) → nerve impulse → activation of the interneuron → activation of the motor neuron →muscle contraction →withdrawal of the leg (excitatory reflex) // There is reciprocal inhibition (i.e. inhibition of antagonist group of muscles on the same side)

Any synapse has a synaptic delay that is about 0.5ms.

#### Crossed extensor reflex

<u>Painful stimulus elicits a flexor reflex in affected limb and an extensor reflex in the</u> <u>opposite limb</u>. Extensor reflex begins 0.2 - 0.5 seconds after the painful stimulus. Serves to push body away from the stimulus, also to shift weight to the opposite limb [so you won't fall down].

we start here also from a pain receptor (nociceptor) activation, this receptor is present in the skin -> through C and Aδ fibers this sensation will be transmitted to the spinal cord -> they go up or down one or two segments then they synapse with interneurons-> these interneurons will cross the midline and synapse with alpha motor fibers that activate the extensors and inhibit the flexor in the other side -> to support the body while it's doing the flexor (withdrawal) reflex

#### Special features of crossed extensor reflex:

- 1- **Polysynaptic** reflex. [slower than stretch reflex and tendon reflex]
- 2- Contralateral reflex.
  Contraction of muscles that extend joints in the opposite limb in response to a painful stimulus.

Stepping on a tack (stimulus)  $\rightarrow$  nerve impulse  $\rightarrow$  activation of several interneuron  $\rightarrow$  activation of the motor neurons  $\rightarrow$  muscle contraction causing **flexion** of the leg stepping on a tack & **extension** on the opposite side. There is reciprocal inhibition (i.e. inhibition of antagonist group of muscles on the same side)

3- Multi segmental

 4- The receptor isn't present in the same muscle! It is in the skin







#### Myograms of flexor and crossed extensor reflexes

The onset of flexor (withdrawal) reflex is faster than crossed extensor reflex. That's <u>crossed extensor reflex needs more time to develop</u> because of having too many synapses.

(in terms of speed: stretch reflex > tendon reflex > flexor (withdrawal) reflex > crossed extensor reflex)

Regarding after discharge, crossed extensor reflex have longer after discharge [more synapses] than flexor (withdrawal) reflex, that is, <u>crossed extensor reflex</u> <u>needs more time to stop</u>.



#### Other Reflexes for Posture and Locomotion [important for babies]

- Pressure on the bottom of the feet cause extensor reflex more complex than flexorcrossed extensor reflex
- Basic walking reflexes reside in the spinal cord.

Reflexes that Cause Muscle Spasm

- Pain signals can cause reflex activation and spasm of local muscles.
- Inflammation of peritoneum can cause abdominal muscle spasm.
- Muscle cramps caused by painful stimulus in muscle: can be due to cold, ischemia, of overactivity [distension]. reflex contraction increases painful stimulus and causes more muscle contraction.

Examples: when there is inflammation in the appendix, in the beginning the pain will be referred around the umbilicus then when the inflammation increases and reach the peritoneum, the pain will be transmitted to the spinal cord by spinal nerves/sensory neurons, those neurons will cause excitation and spasms in the muscles around the area of the appendix [LRQ]. the same happens when there is inflammation in the gallbladder it starts as referred pain in the right shoulder then it will cause muscle spasm around the area of gallbladder.

6 | Page