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PHYSIOLOGY

وَلَقَدْ خَلَقْنَا ٱلْإِنسَٰنَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِۦ نَفْسُهُۥ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ ٱلْوَرِيدِ

MID | Lecture 11

Neural circuits

Reviewed by : Rand Alkhateeb

Color coding used in the modified:

- *Black: the original slides*
- *Blue: the doctor's explanation/words*
- *Gray: additional information and explanation*
- *Red: important information*

Introduction to Neurophysiology 5

Neural circuits

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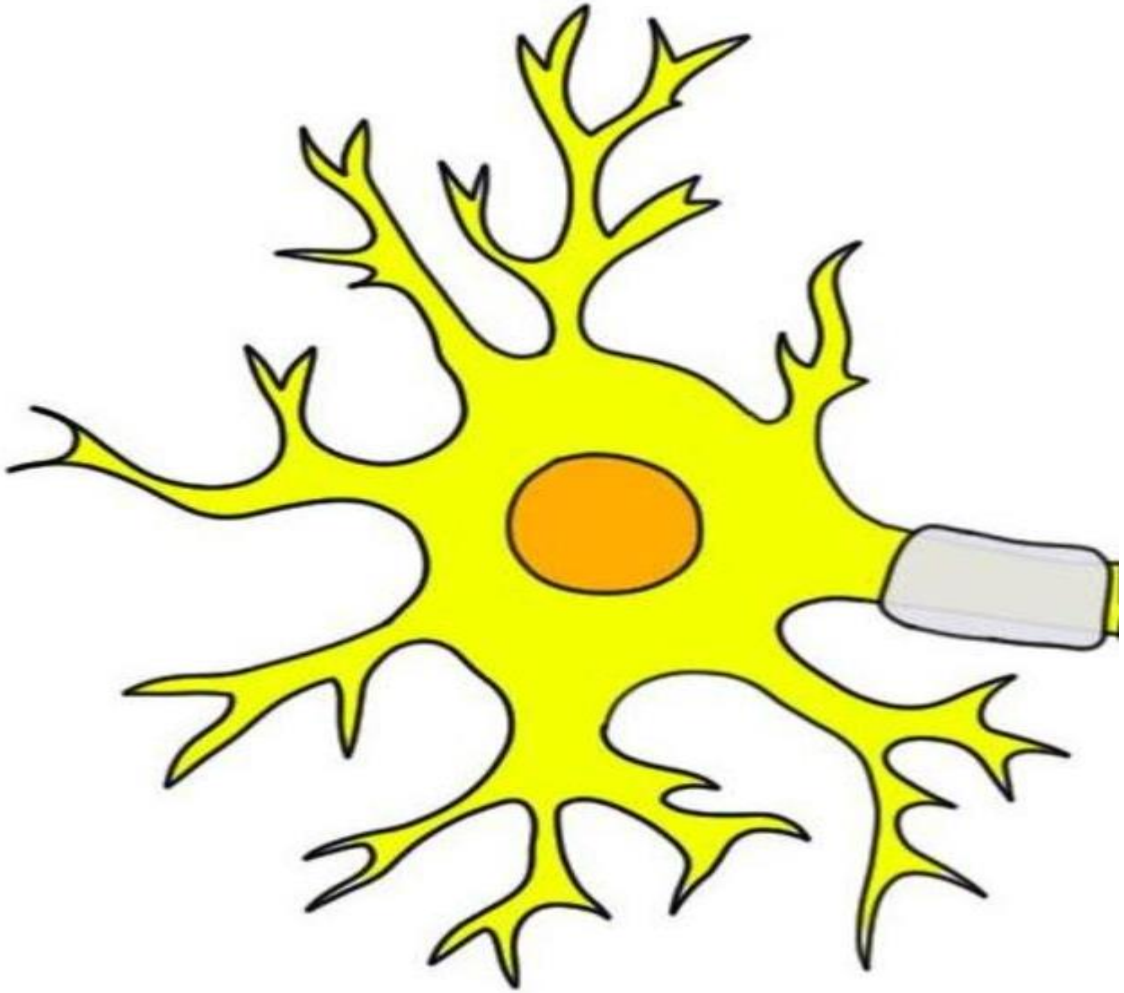
Dendrites:

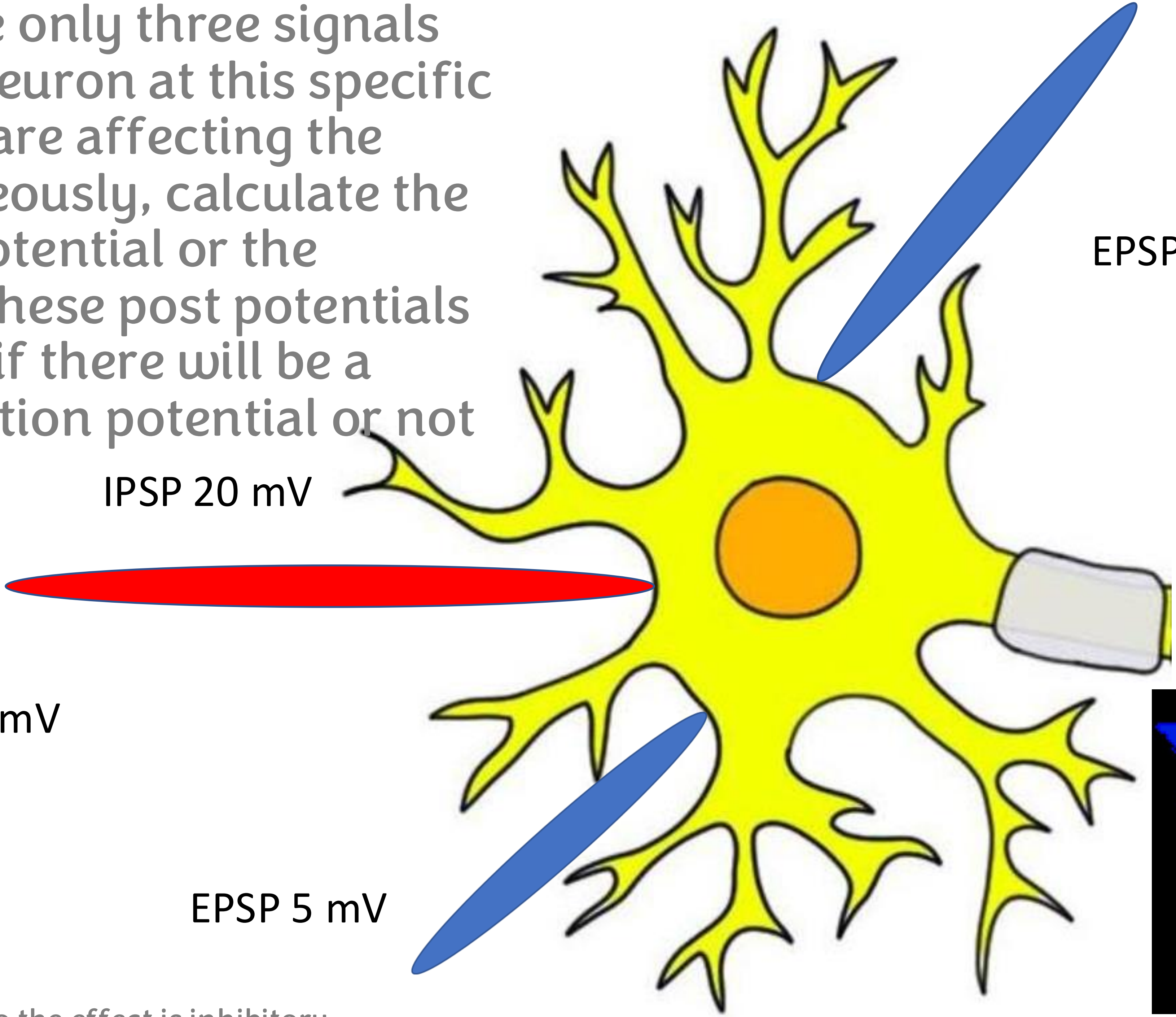
Large spatial field of excitation.

A great opportunity for summation of signals from many neurons.



Chances of summation in synapses are higher, but it doesn’t have a good amount of voltage gated channels and the conduction at the dendrites is decremental

*If these are the only three signals affecting this neuron at this specific time, and they are affecting the soma simultaneously, calculate the postsynaptic potential or the submission of these post potentials and determine if there will be a discharge of action potential or not in this case?

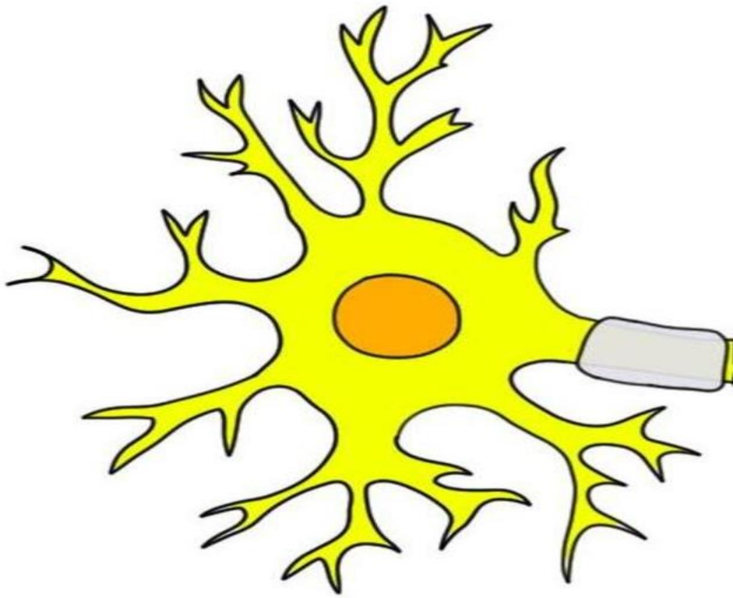


RMP -65 mV
Threshold -55 mV

Answer : psp will be -70 so the effect is inhibitory

Decremental conduction

The synapses that lie near the soma have far more effect in causing neuron excitation or inhibition than those that lie far away from the soma.



In other words, the same synapse will be **more effective** if it's nearer to the soma **than** if it's further away from the Soma (it depends on the distance)

Further explanation:

- The dendrites are long and their membranes are thin, what characterize these membranes that they are leaky to potassium ions, and we know that the potassium ions will act oppositely to the effect of the sodium ions (sodium ions will cause depolarization whereas potassium will cause repolarization), especially that the equilibrium potential is near the resting potential.

Resting membrane potential of neuronal soma

- Any change in potential in any part of the intra-somal fluid causes an almost exactly equal change in potential at all other points inside the soma.

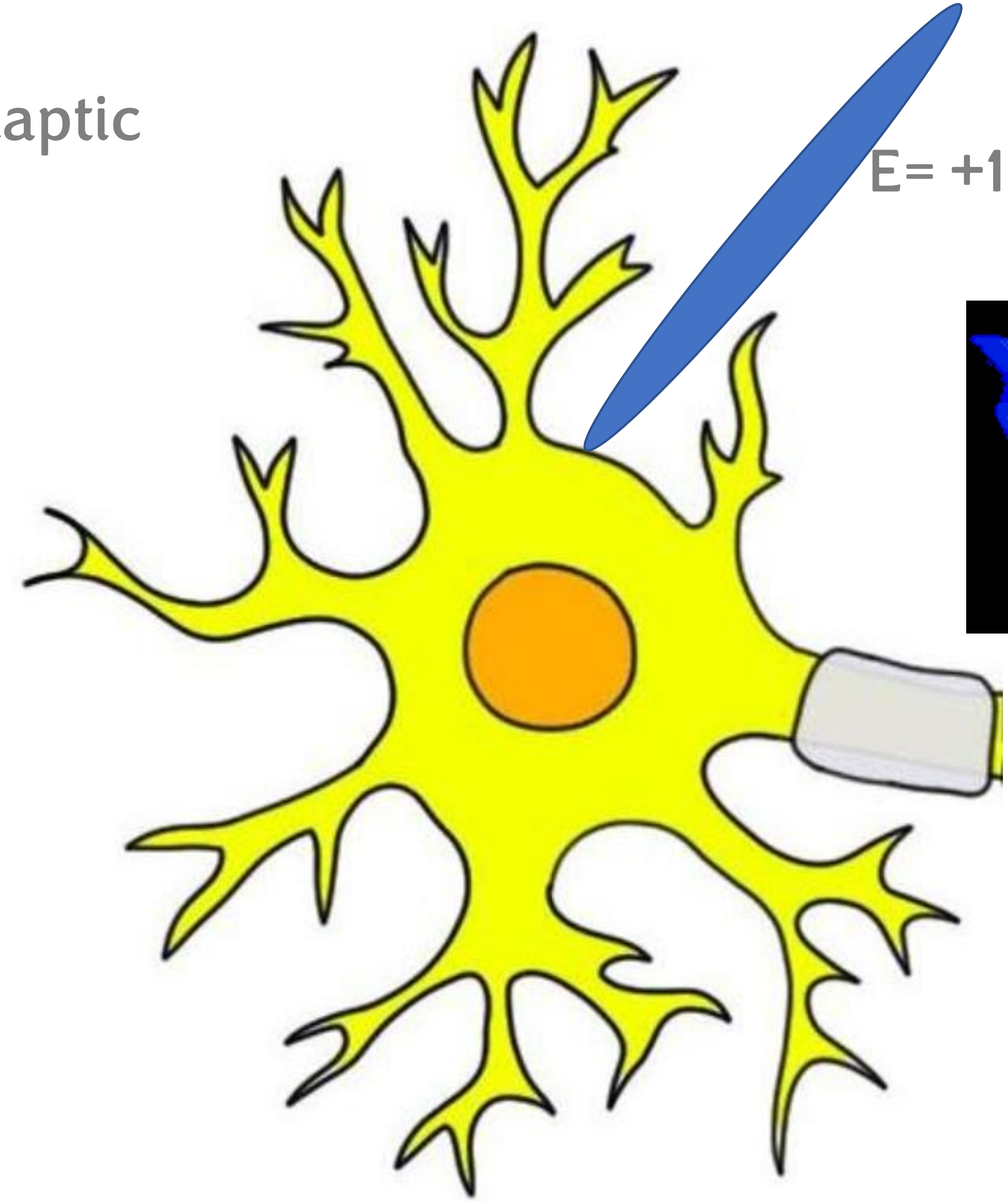
- This principle is important because it plays a major role in "summation" of signals entering the neuron from multiple sources.

Summation in neurons

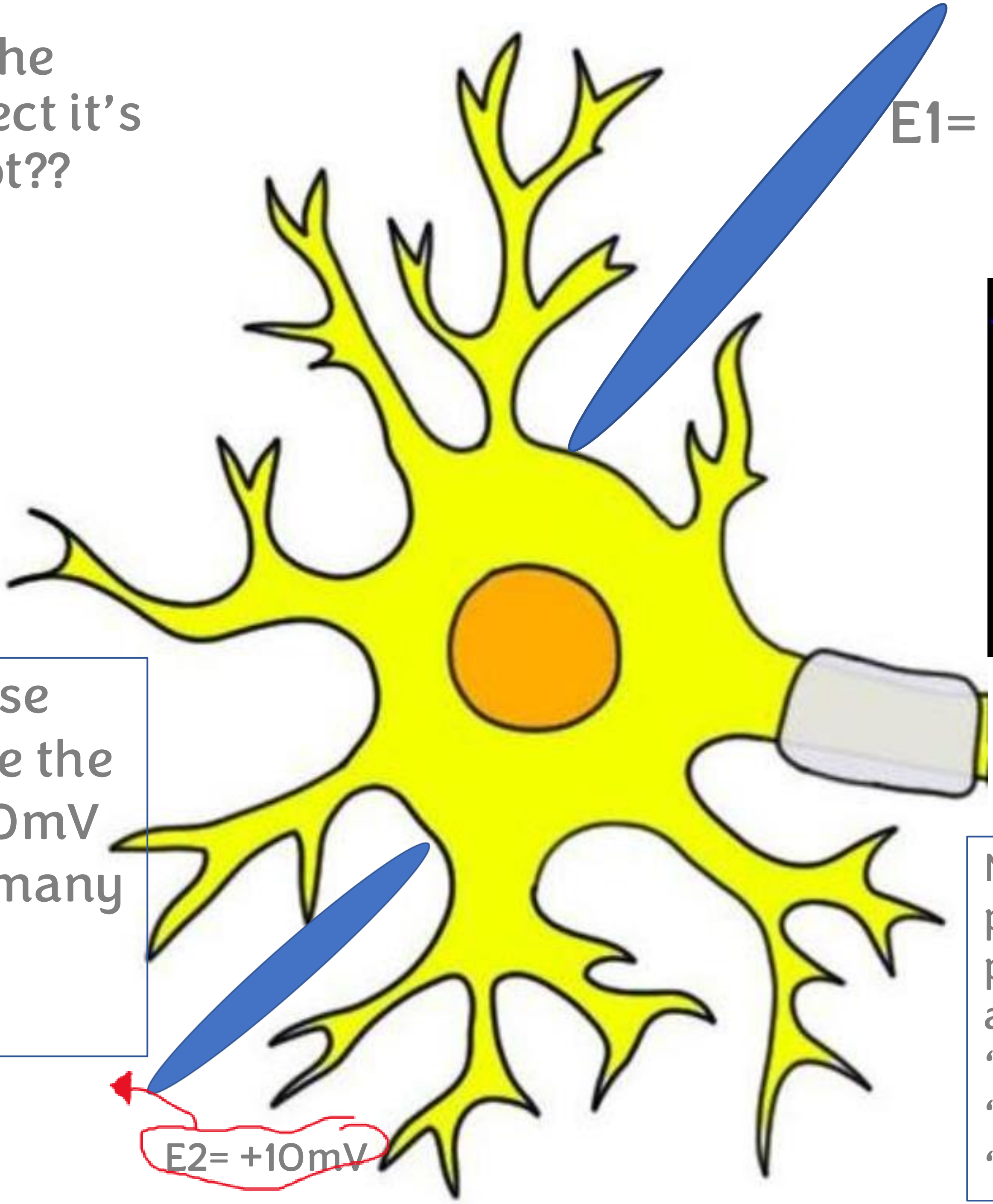
- Excitation of a single presynaptic terminal on the surface of a neuron almost never excites the neuron.

- The reason is that the amount of transmitter substance released by a single terminal to cause an EPSP is usually no greater than 0.5 to 1 millivolt, instead of the 10 to 20 millivolts normally required to reach threshold for excitation.

* Calculate the grand postsynaptic potential in this neuron?
If:
Resting potential = -70mV



*If we are assuming that these are the only excitatory signals, do you expect it's gonna fire an action potential or not??
If:
Resting membrane potential = -70mV
Threshold = -50mV



It's not very realistic actually because one synapse is most likely to change the membrane potential of 0.5mV to 1.0mV not that much. Usually we need so many synapses to reach this level.

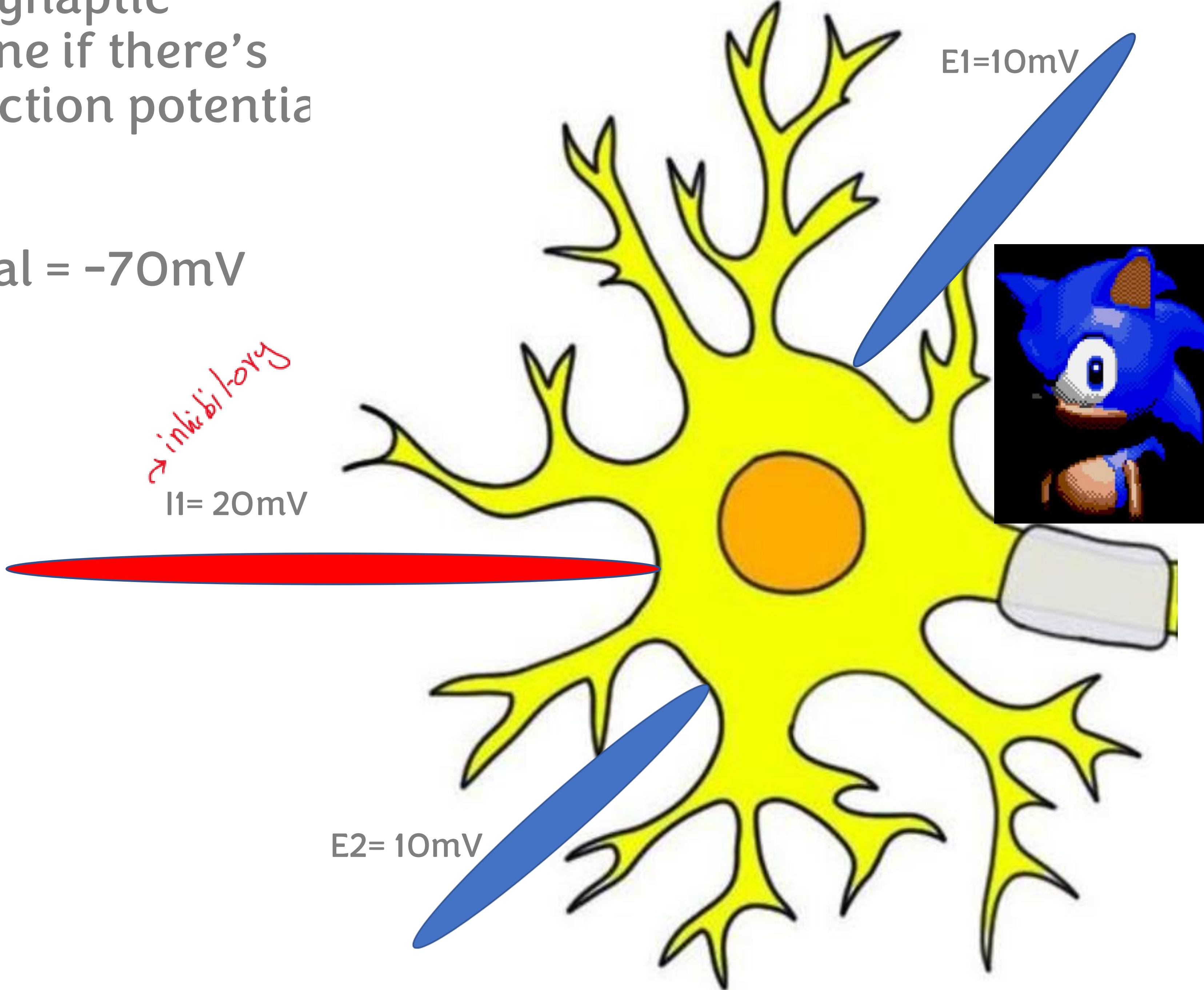
Note/ action potential propagation also means
"fire"
"Discharge"
"Generate"

* Calculate the grand postsynaptic potential and then determine if there's gonna be a generation of action potentia or not?

If:

Resting membrane potential = -70mV

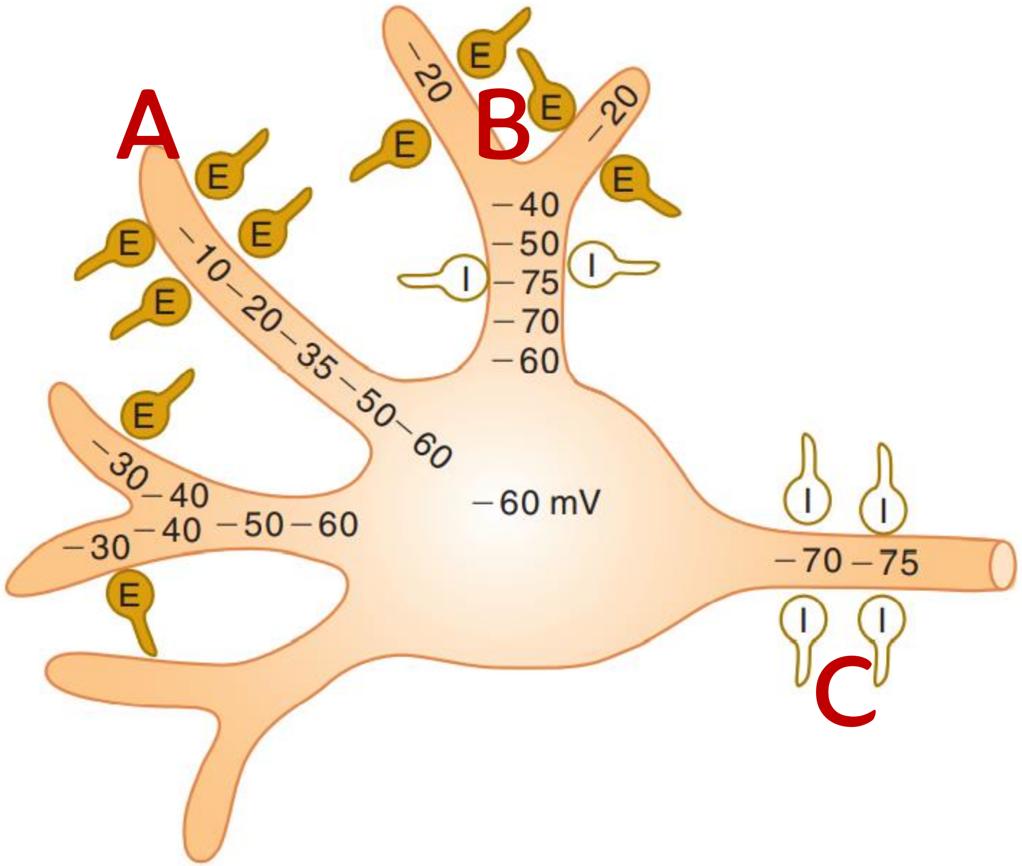
Threshold=-50mV



This picture shows how could I have many arrangements of synapses on the dendrites, Soma & axon.

- Check the **A** reason for example, it has almost 4 excitatory synapses, note that the resting membrane potential is -60 mV. Together, summation of the four excitatory synapsis will change the membrane potential from -60 to -10, the problem is as we go down towards the Soma the main potential will go back to its resting state with the help of Na/K pump and the leaky channels
- As for region **B**, you can see the excitatory synapses trying to excite the cell & as it go down, you can see the potential it's getting back to interesting state during that inhibitory synapses will affect the membrane potential and inhibit it even more hyperpolarization, it could face and excitatory synapses afterwards, but in this case as we reach the soma, it will go back to it resting state finally

At **C**, these are inhibitory synapses at the initial segment of the axon, so the strongest effect would be absolutely on this site because this is where the action potential is generated, in this case, the neuron will not fire an action potential because it's inhibited



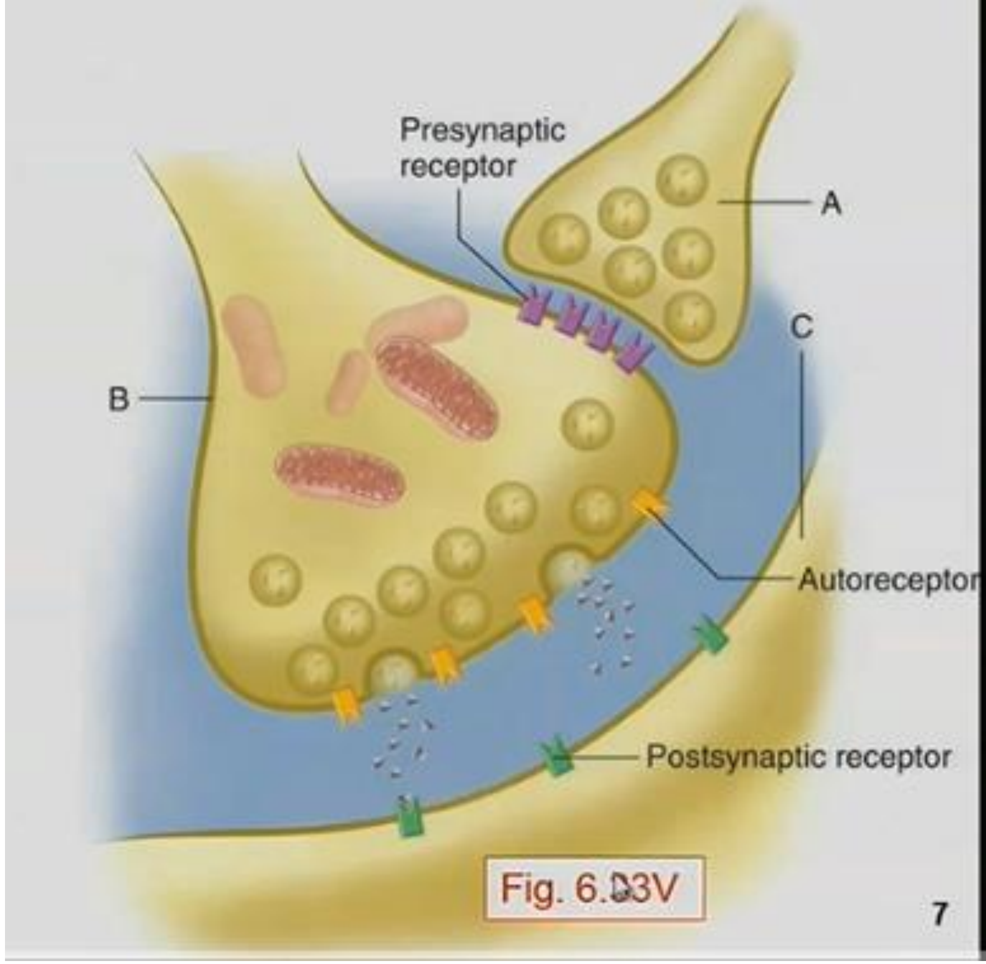
Presynaptic inhibition

- In addition to inhibition caused by inhibitory synapses operating at the neuronal membrane, which is called **postsynaptic inhibition**.

- **Presynaptic inhibition** is caused by release of an inhibitory substance onto the outsides of the presynaptic nerve fibrils before their own endings terminate on the postsynaptic neuron.
- In most instances, the inhibitory transmitter substance is GABA.
- This release has a specific effect of opening anion channels, allowing large numbers of Cl- ions to diffuse into the terminal fibril.

As we discussed, the changes that will happen at the PSP could be inhibitory or excitatory, depending on the type of the neurotransmitter & the PSP
Another thing that could happen is presynaptic inhibition (A) just before (pre) the main synapse as you can see in the picture, (the pre-synapse is usually inhibitory , we could have GABA as an inhibitory neurotransmitter to a chloride channel, for example, to decrease the rate of depolarization which drifts the potential away from the threshold so it won't stimulate the voltage gated calcium channels)

This is an important mechanism for sensory function. When a pin integrates your skin, for example, sensory neurons will fire because of the pain, but to identify the injury place, a lateral presynaptic inhibition must happen to allow just the action potential at the injury place to be transported, therefore, I can pull the pin and treat the injury



Presynaptic inhibition

- Presynaptic inhibition occurs in many of the sensory pathways in the nervous system, such as, adjacent sensory nerve fibers often mutually inhibit one another, which minimizes sideways spread and mixing of signals in sensory tracts.

One of the important applications of the presynaptic inhibition is in the sensory system, we have what we call it "lateral inhibition" or presynaptic inhibition is in (vision) to make the border of the images much more clearer.

Facilitation of neurons

- Often the summated postsynaptic potential is excitatory but has not risen high enough to reach the threshold for firing by the postsynaptic neuron.
- When this situation occurs, the neuron is said to be facilitated.
- That is, its membrane potential is nearer the threshold for firing than normal but is not yet at the firing level.
- Consequently, another excitatory signal entering the neuron from some other source can then excite the neuron very easily.

On the other hand, in this kind of presynapse it's the opposite, the neurotransmitter receptor complex is excitatory so the potency of this synapse is even more increased, we call it presynaptic facilitation.
The activity of this synapse is going to be altered depending on the other factors that are controlling it.

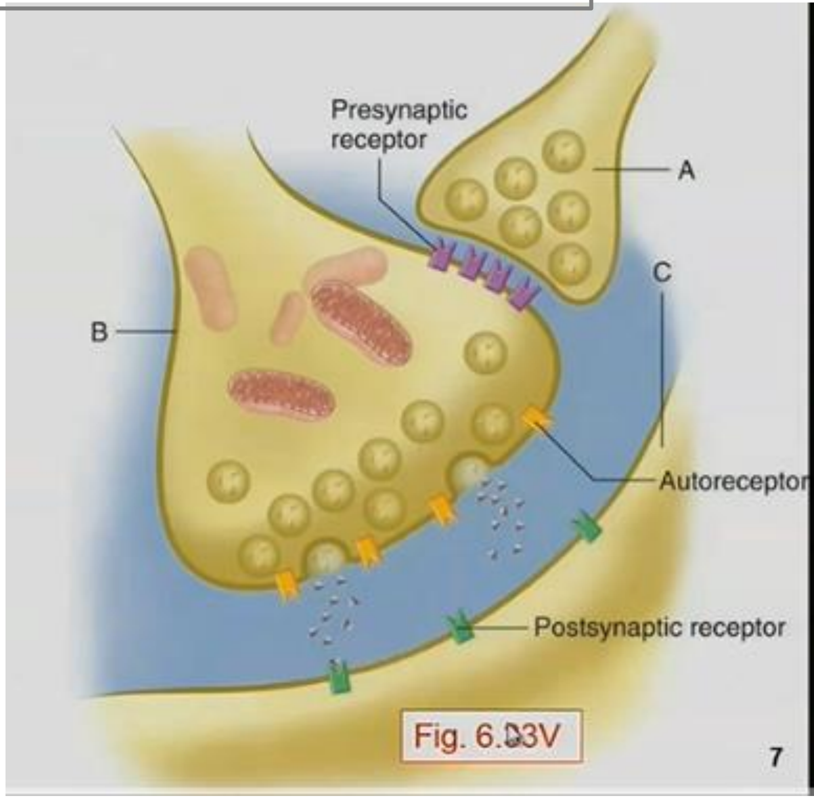
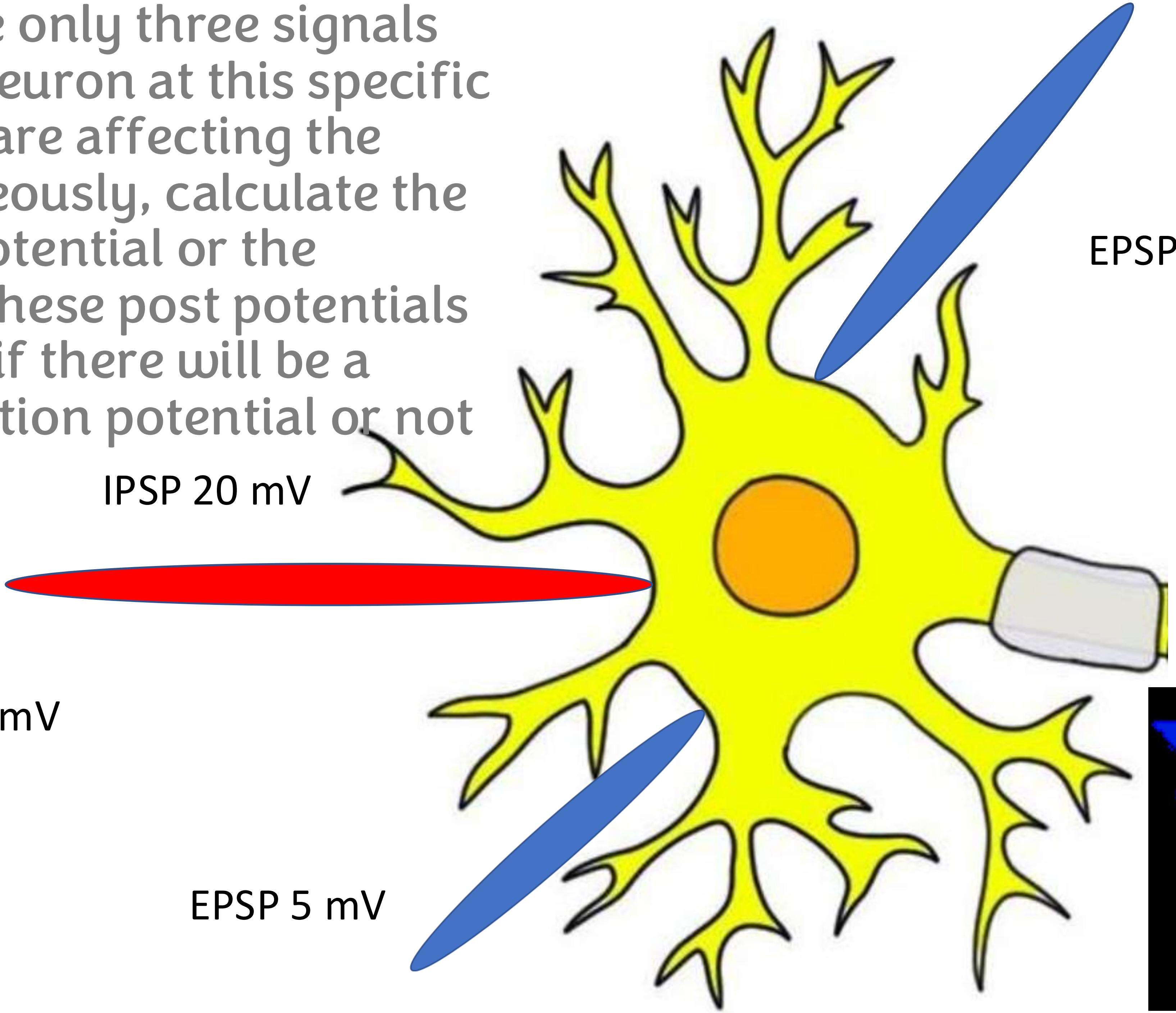


Fig. 6.33V

*If these are the only three signals affecting this neuron at this specific time, and they are affecting the soma simultaneously, calculate the postsynaptic potential or the submission of these post potentials and determine if there will be a discharge of action potential or not in this case?

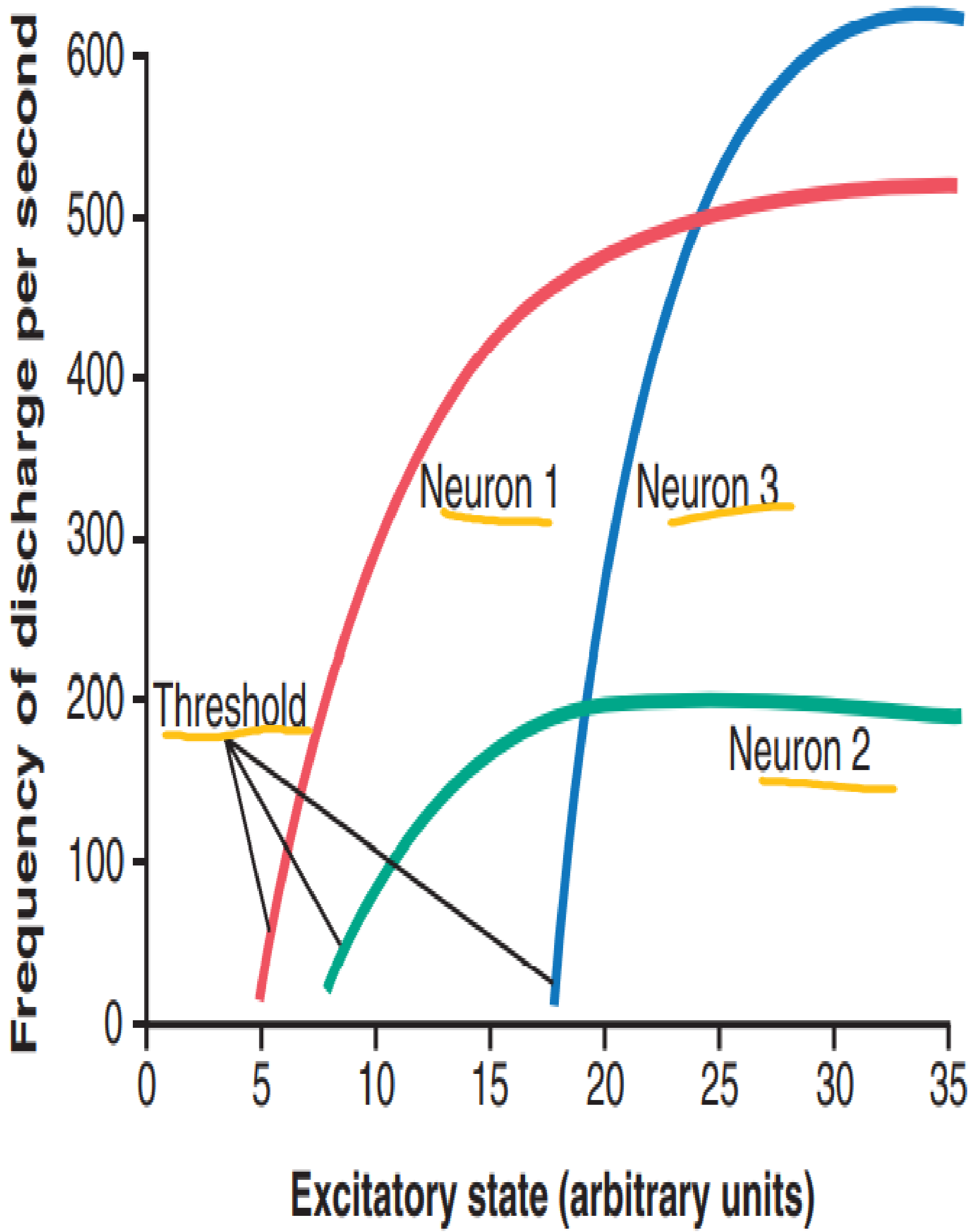


RMP -65 mV
Threshold -55 mV

Neural circuit

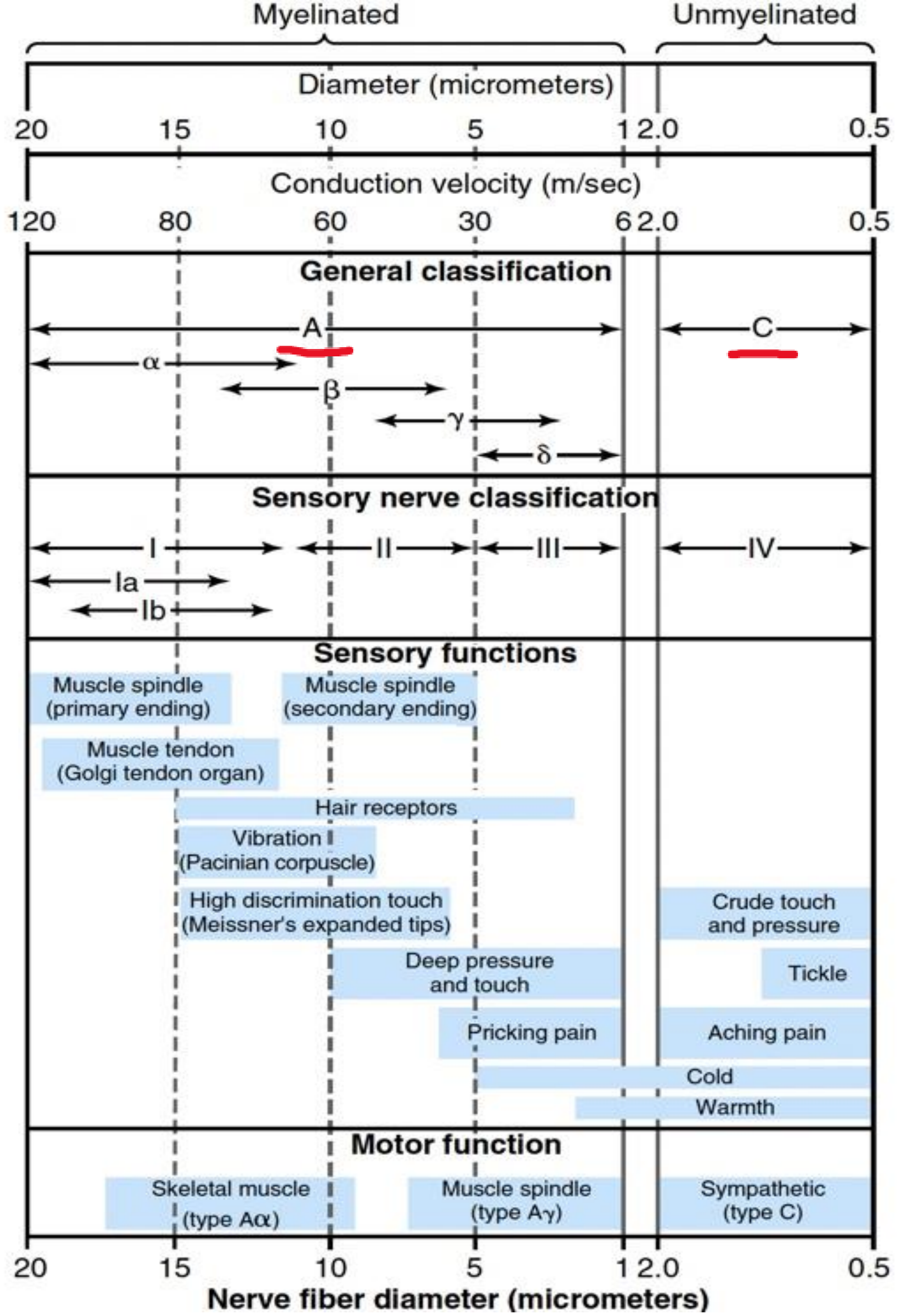
All neurons share the same main principles, such as:(action potential, postsynaptic potential, chemical synapses, neurotransmitters, neurotransmitter-receptor complex), however, neurons serve different characteristics with different thresholds, and that's because they serve different functions. Sometimes we need a neuron to transmit the signal super fast so they fire almost continuously & should have very low threshold for generation action potential (neuron 1)
As for (neuron 3) which has a huge range of frequency of discharge, meaning that it gives more ranges for frequency of action potential so I can get different degrees of simulation to send more frequent action potential .

~ look at this graph, there is (neuron 1),(neuron 3) & (neuron 2), you can see that neuron1 here has the lowest threshold so it fires very fast, but you can see that the maximum frequency of discharge is for neuron3 which has almost the highest threshold so it takes long until it generates action potential but once it's reached , it can reach a maximum frequency of action potentials per second that is much higher than neuron1 or neuron2.
So again, these kind of differences in their exact value of the threshold for the action potential and in their frequency of discharge or the rate of discharge in each neuron, they are very important in serving different functions because nervous system is composed of different pools and these pools will serve different functions



The conduction velocity varies, depending on the diameter of the neurons (less resistance). We have a wide range of changes in diameters between neurons (from 0.5 µm to 20 µm) myelinated neurons usually have large diameters
Also, myelinated nerve fibers are very much quicker or faster in transmitting signals than the unmyelinated ones.
There are several classifications for these nerve fibers of neurons depending on the diameter and also the myelination.

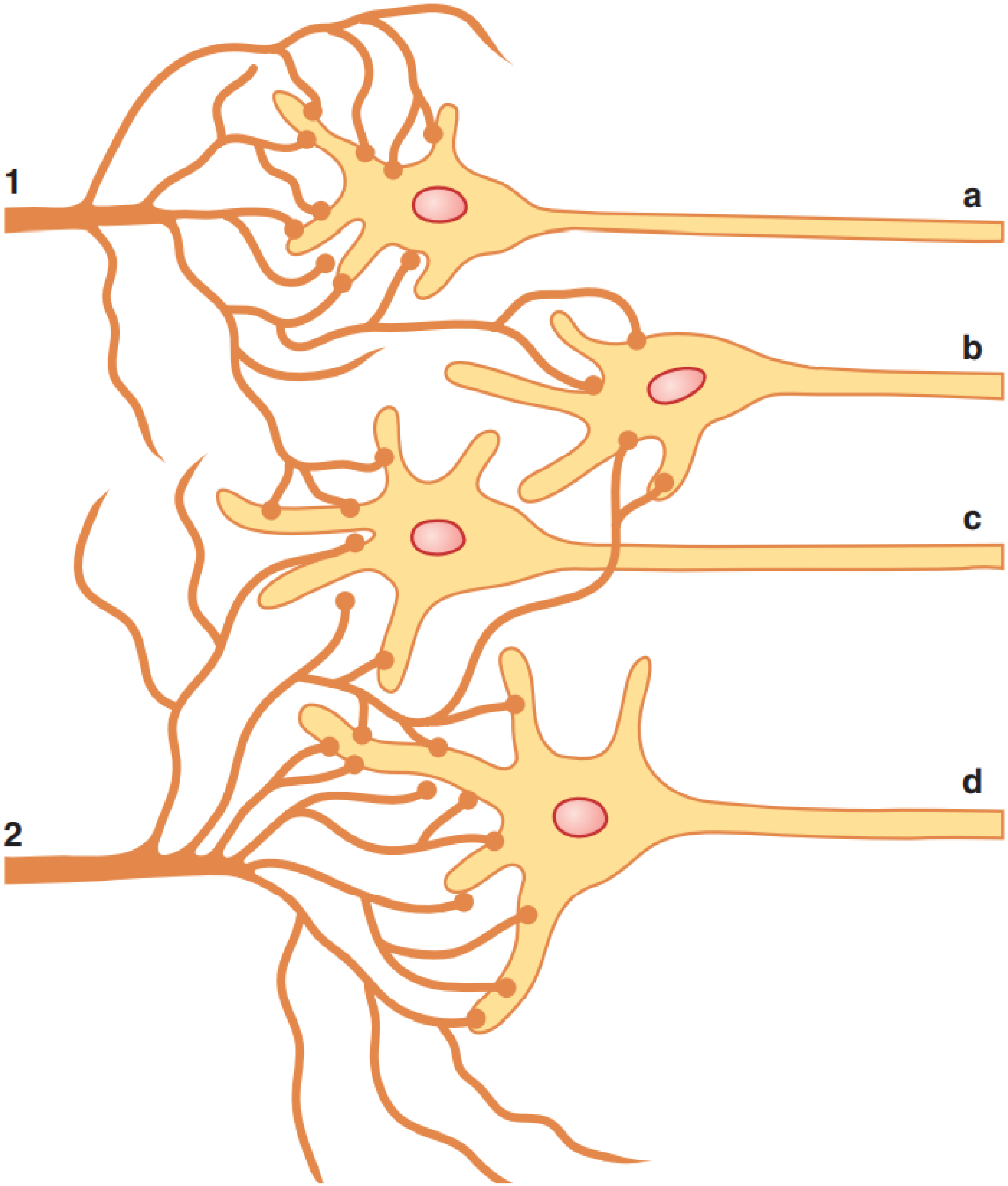
For example, neuron fibers type A are myelinated and have large diameter (better conductance), while type C, which are small unmyelinated fibers.
If you have a signal that you need to be transmitted super fast would you choose fiber A or fiber C? it's definitely fiber A
for example, in our reflexes like a stretch reflexes in the spinal cord, you want this signal to reach very fast and finish this reflex super fast in order to protect the muscle from injury to maintain the balance of the body so these type of signals, they have type A fiber. (Acute pain)
whereas if you're talking about, for example, the inflammatory changes that have been going for a long time especially in an internal organ, they are transmitted through type C fibers because they are not crucial to the function of our body. (chronic pain.)



Stimulatory field:

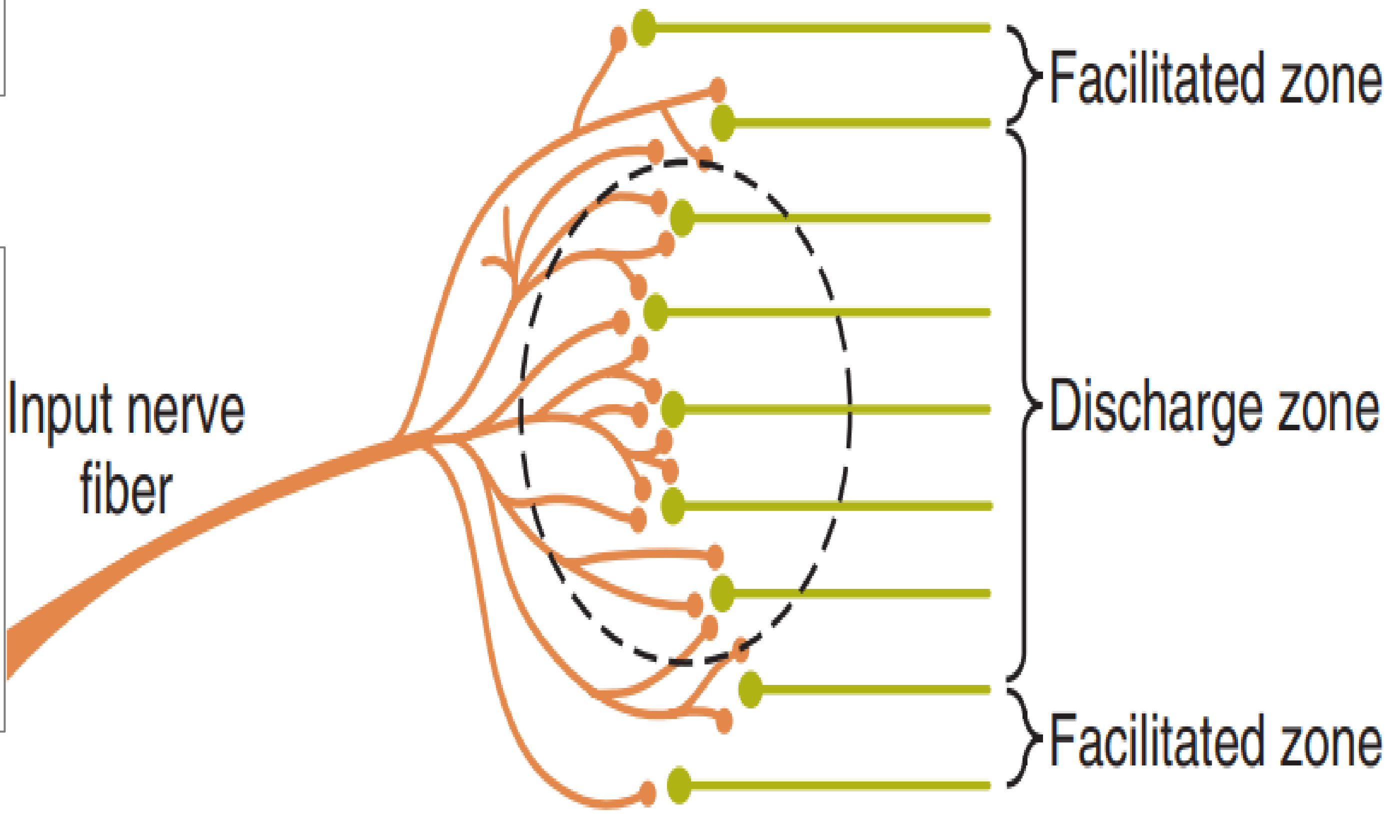
The neuronal area stimulated by each incoming nerve fiber.

For neuron 2 the stimulator field will be on neuron d, c, & d
If neuron two was an excitatory (discharged) neuron, it will mostly affect neuron d cause it have more summation synapses with it, in this case, it can fire an action potential so we can call it discharged zone, then goes c then b & a will not be affected



You can see here in this stimulatory field that the concentration of the synapses is the highest in the center so in this area if there is an excitatory signal, then there is a higher chance of reaching a threshold (a higher chance of discharge) that's why this central area is called the "discharge zone".

Whereas on the periphery this kind of excitatory signal is not strong enough and the number is not large enough to reach a threshold so it cause what we call it "facilitation" so it's making it nearer to the threshold that in the next time or by the next synapses this neuron may fire an action potential.

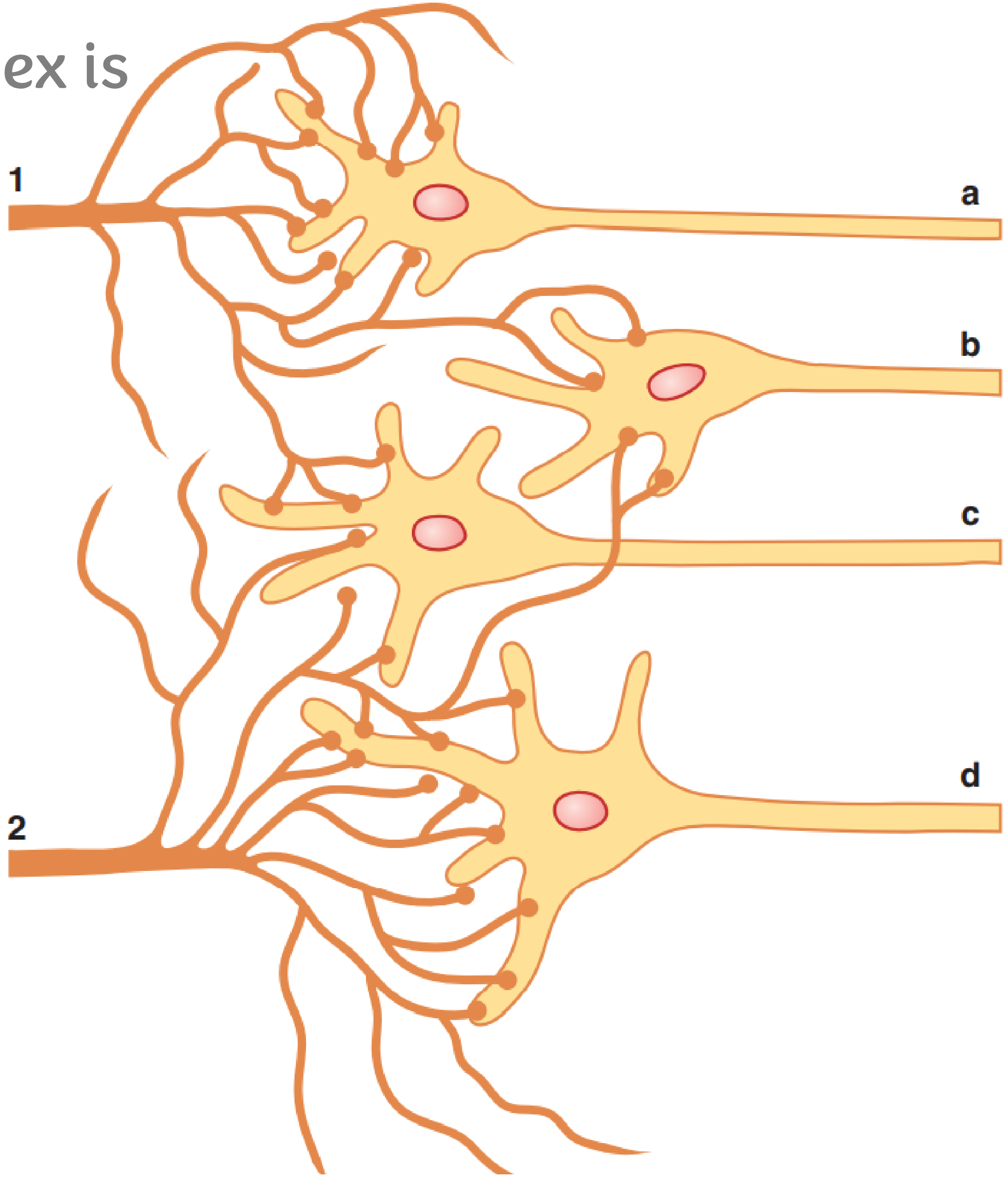


Assuming that the effect of the neurotransmitter-receptor complex is inhibitory

Inhibitory zone:

Greatest inhibition in the center of the zone.

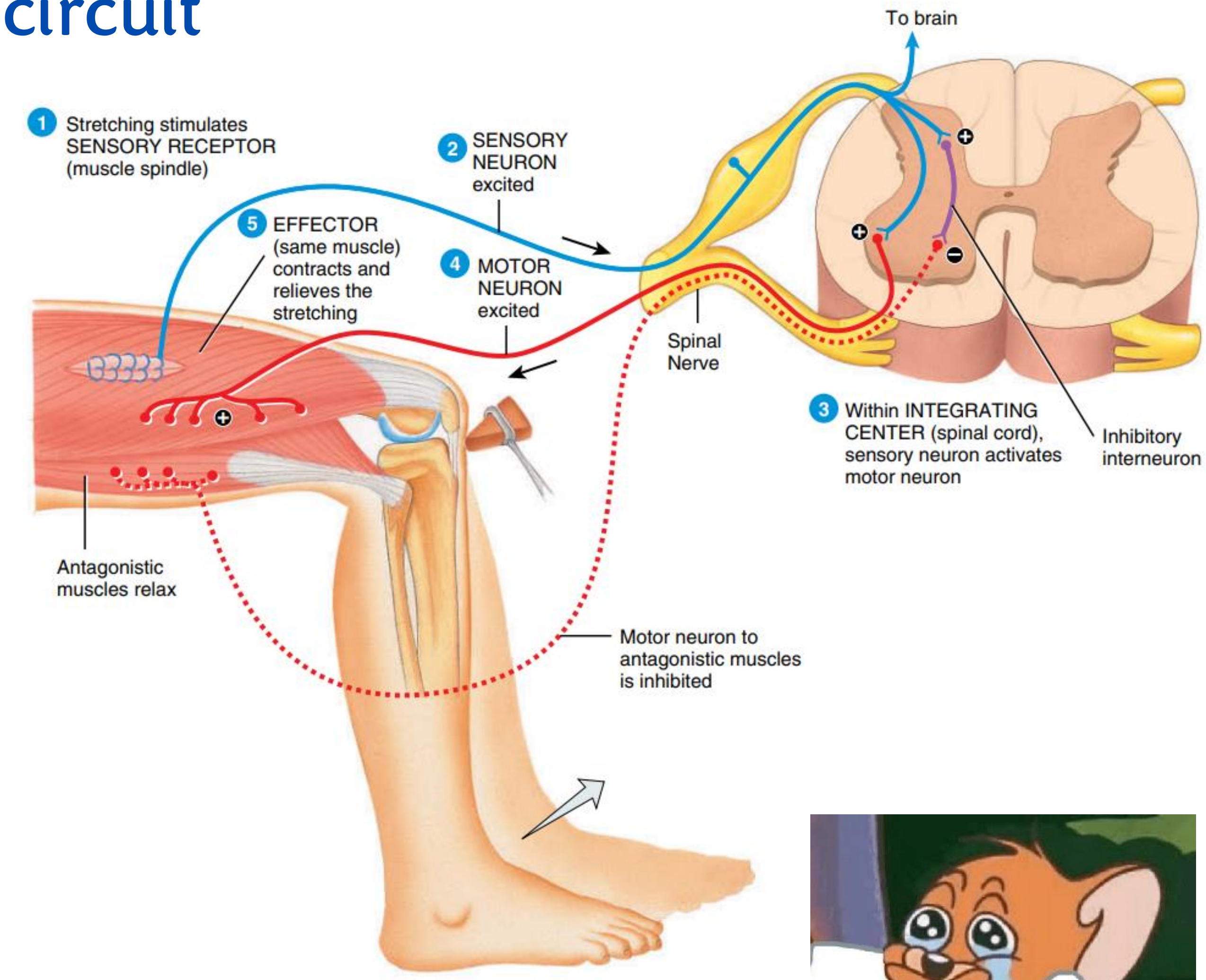
For neuron 1 the stimulator field will be on neuron a, b, & c
If neuron two was an inhibitory neuron, it will mostly affect neuron a cause it have more summation synapses with it, in this case, it can affected very strongly, then goes b then c & d will not be affected



Neuronal circuits

- A group of interconnected neurons that perform certain function.

*Example of common neuron circuit



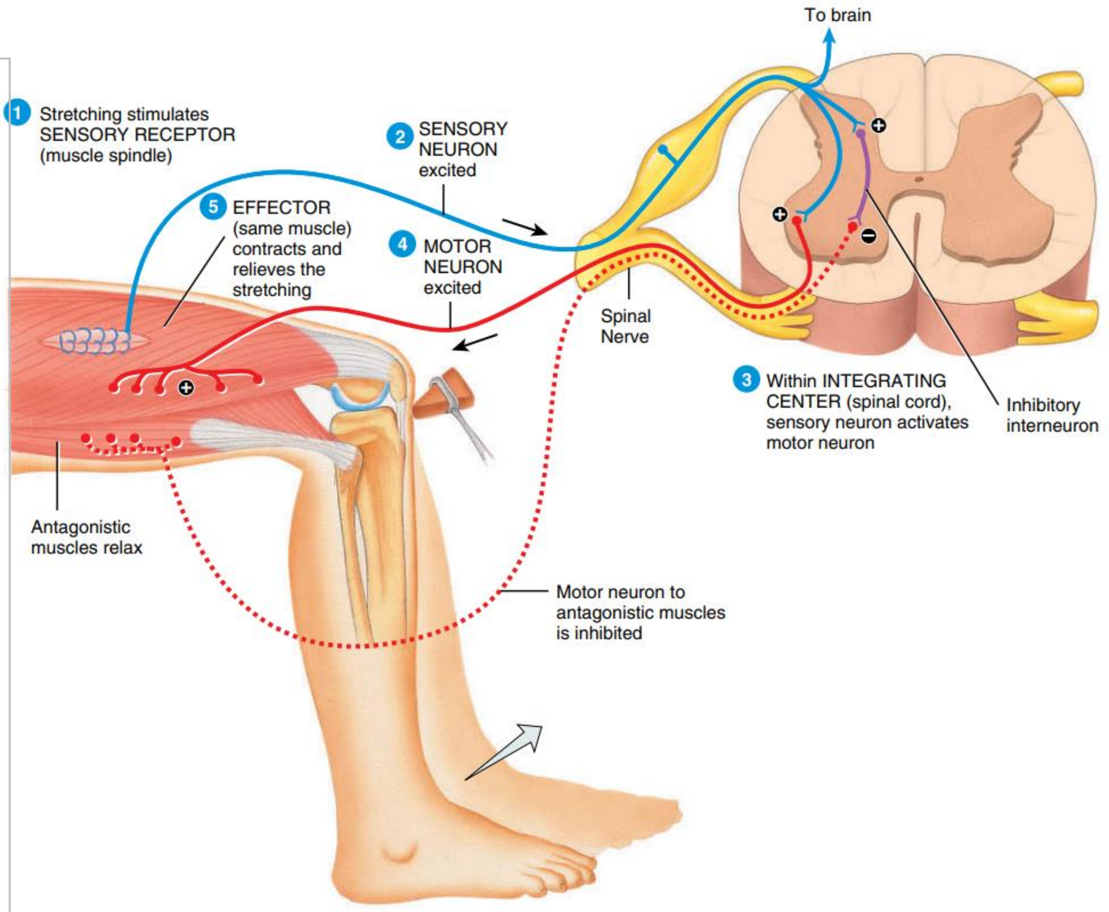
This kind of neuronal circuit is called a reflex (in this case a spinal reflex because the integration is at the gray matter of the spinal cord)
It is a " stretch reflex" because the stimulus here was a " stretch stimulus ". So you're Gonna ask the patient to sit according to the picture without his foot, touching the ground and relax, then you will hit the patella tendon with a hammer. Here you are stimulating this muscle,(stretching the muscle) which will activate a specific receptors here in the bulk of the muscle called **stretch receptors** or the muscle spindle, that signal will be sent through the sensory neuron (the one in blue in the photo) and it will enter the CNS to the grey matter of the spinal cord where it will synapse immediately with a **motor neuron** by glutamate, usually which will act to stimulate the extensor of the muscle to **contract** by acetylcholine that will be at the neuromuscular junction (the proper response) to protect this muscle fibers from injury.the response is contraction of the same muscle to protect this muscle.

Neuronal pools

- Functional groups of neurons occurring in the grey matter of the brain and spinal cord, which process and integrate incoming information received from other sources, such as the sense organs, and transmit the processed information to other destinations. (oxfordreference.com)

- Each neuronal pool has its own special organization that causes it to process signals in its own unique way and perform certain function.

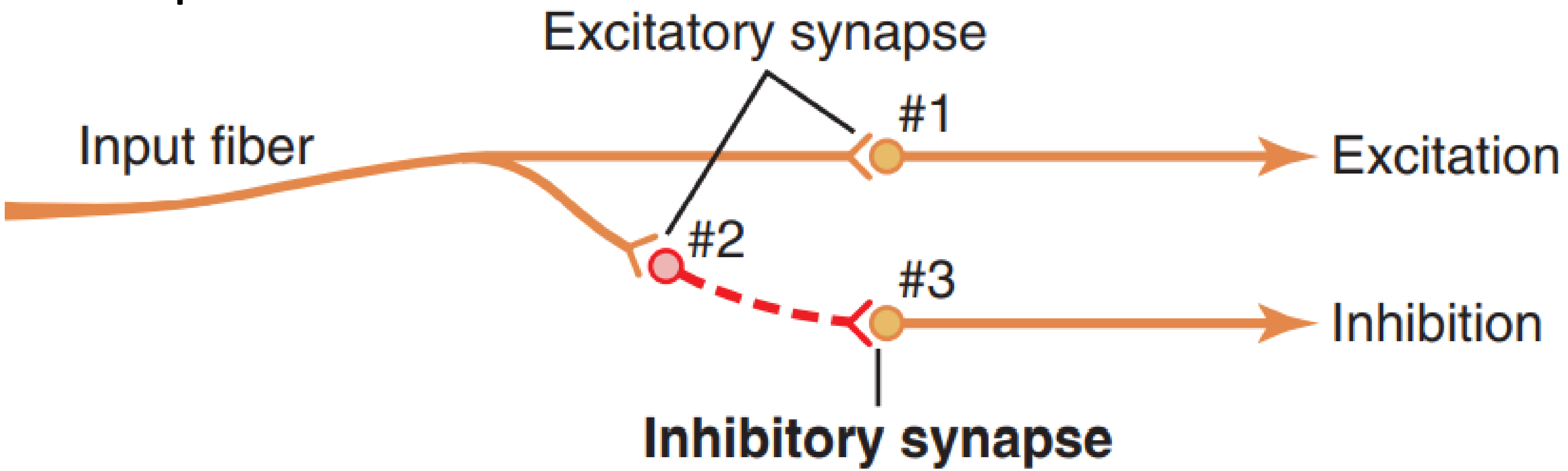
We can see that this sensory neuron is sending other branches, remember when you learned about movements of the limbs that we have “antagonistic muscles” if there is a muscle that will do an extension on the knee, there is another muscle that will do flexion on the knee, so if you are contracting a muscle, you should relax the antagonistic muscle, and this is the same in this case so if I’m contracting this extensor muscle, I have to relax the flexor.
But how do we do this if we have the same stimulus and sensory neuron which has almost the same neurotransmitter (in this case it’s an excitatory neurotransmitter)?
The thing here if u want to do an excitatory effect on a side, you’re gonna do an inhibitory effect on the other side.. So in this case, we added here an **enter neuron** and it’s an **inhibitory neuron** so you can see from the photo the sensory neuron is sending an excitatory signal to the entery neuron and this entery neuron is synapsing with the motor neuron of the antagonistic muscle.. This inhibitory entry neuron release an **inhibitory neurotransmitter, for example: “glycine”** and that will cause inhibition of this motor neuron to the antagonistic skeletal muscle causing inhibition in it.
Because this is an opposite effect, we call it “circuit reciprocal inhibition”, (reciprocal innervation).

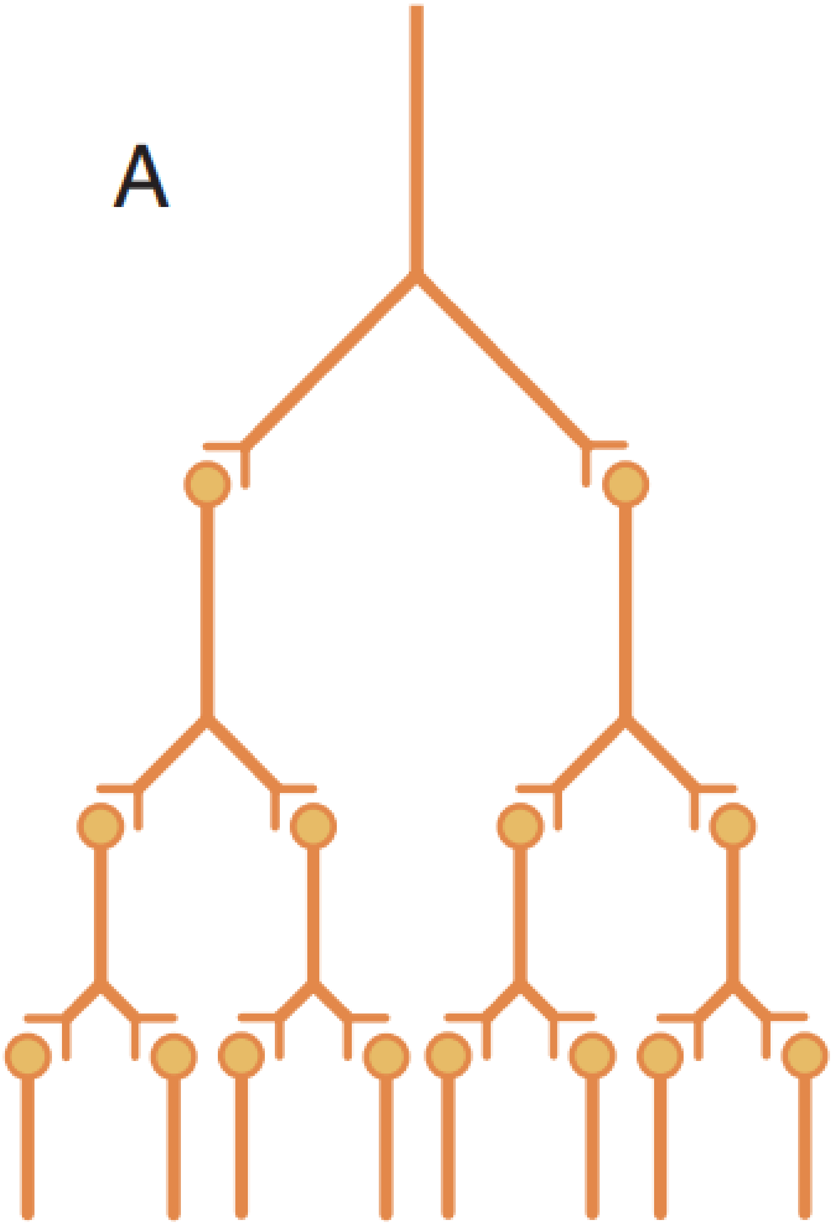


Reciprocal inhibition

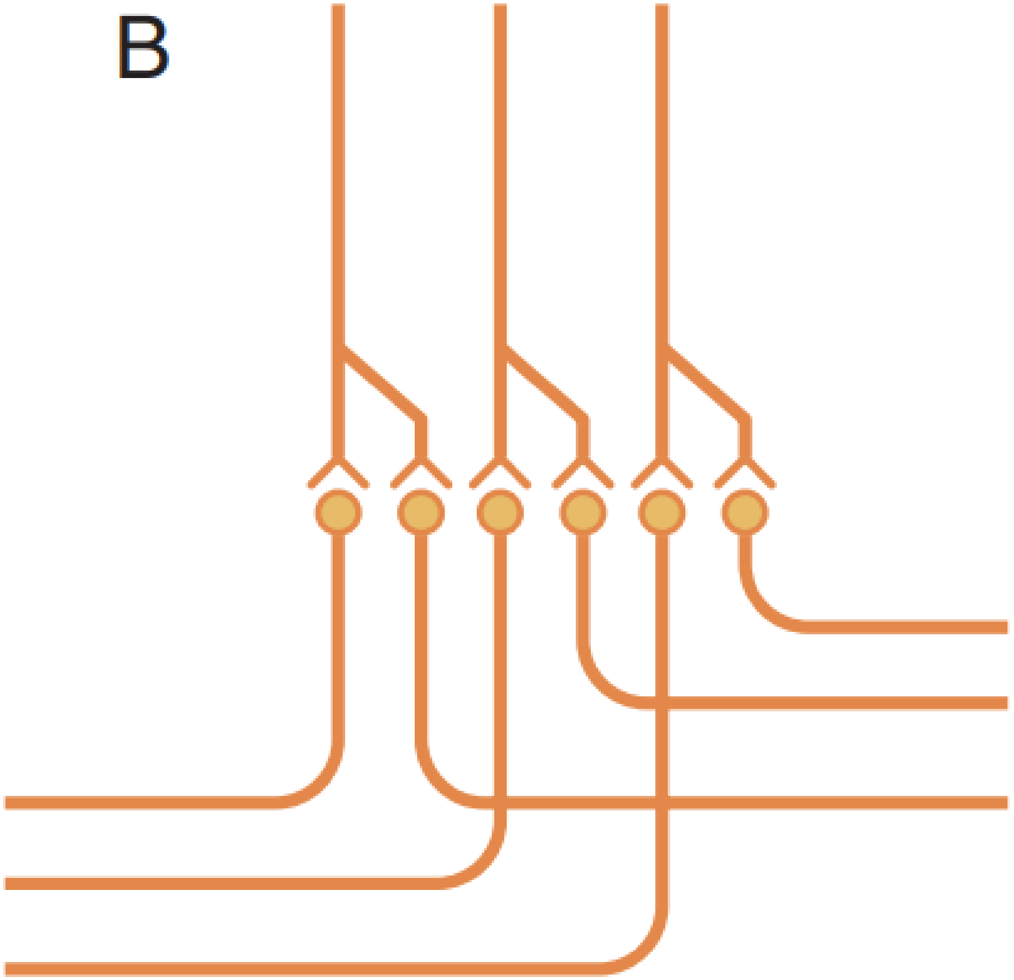
- Sometimes an incoming signal to a neuronal pool causes an output excitatory signal going in one direction and at the same time an inhibitory signal going elsewhere.

- This type of circuit is characteristic for controlling all antagonistic pairs of muscles.

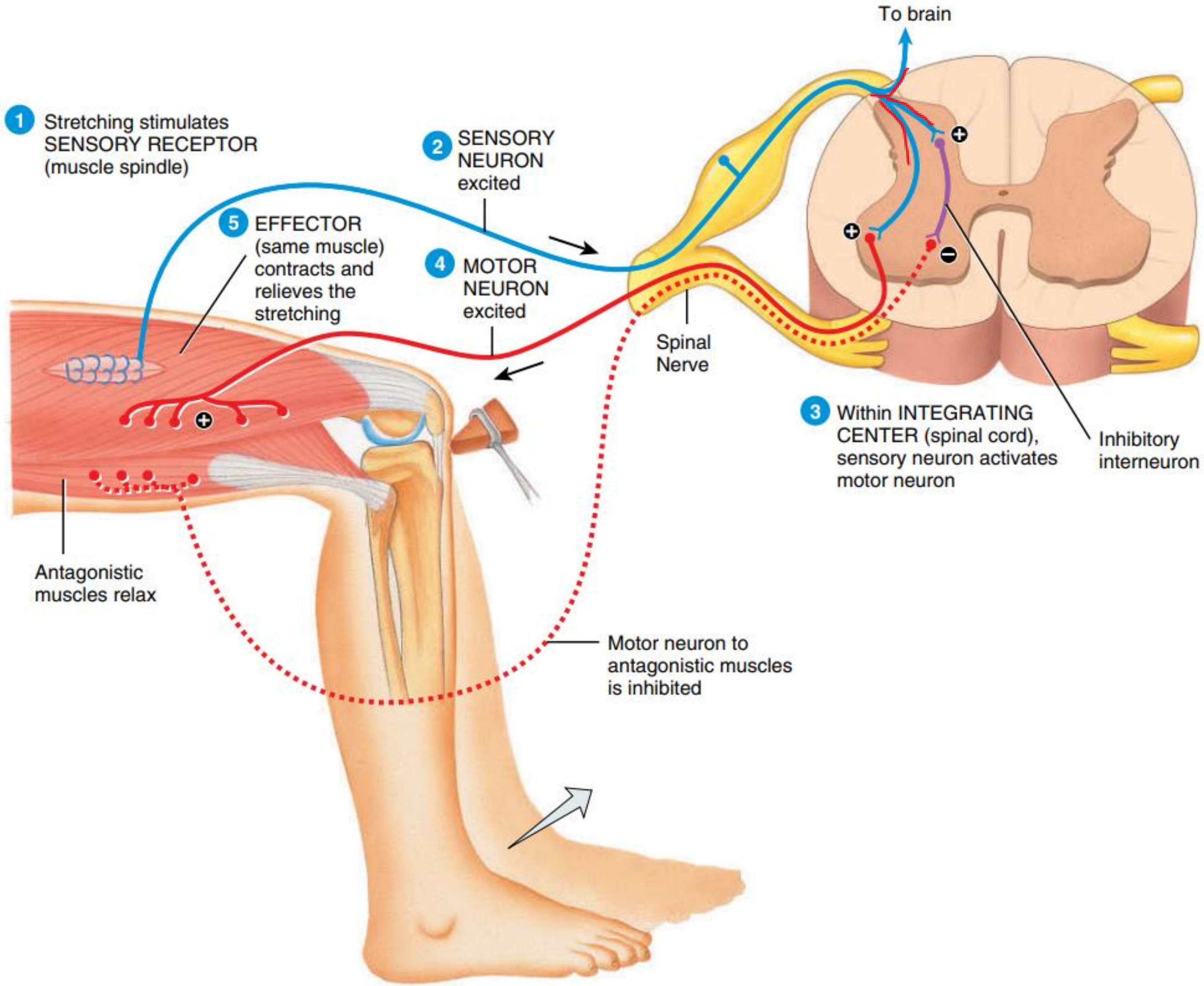




Divergence in same tract



Divergence into multiple tracts

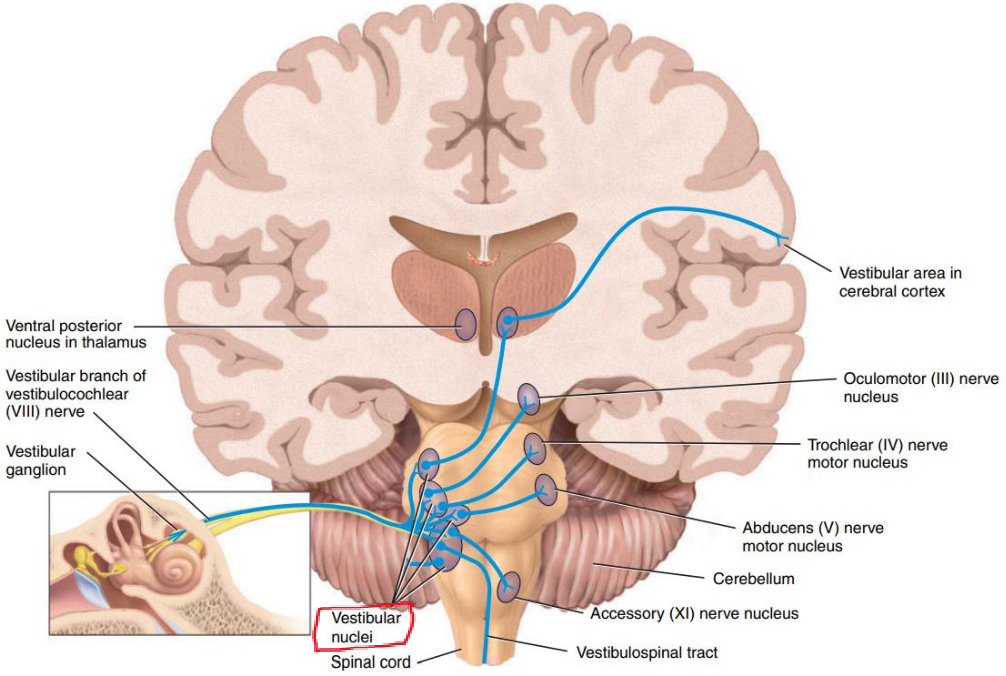


~ Note here that this sensory neuron branches to give different stimuli to different outputs..for example, the first branch stimulates the muscle, the other one is to inhibit the antagonistic muscle and the third one is to give an **information** to the higher areas in the brain about this change during the reflex, you need to be consciously aware of that,You need to know this kind of information in order to coordinate your balance coordination..
so this divergence of signal is important to serve different functions in the nervous system.

It's very important to have this holistic kind of integration of information to this Intergrative part of processing information

For example: when you move your head there are specialized sensory receptors present in the inner ear(the sensory receptors there aren't just for hearing, but also for balance of the head), this kind of information will be transmitted through one of the cranial nerves called the "vestibular cochlear "(vestibular stands for balance, and cochlear stands for hearing), this nerve will enter to the nuclei in the brainstem, the vestibular nuclei has to diverge this signal from the vestibular nerve has to diverge into multiple tracks.. For example, when you move your head, you need to move your neck. You need also to maintain the balance in your body by making proper contraction relaxation of your axial body muscles.

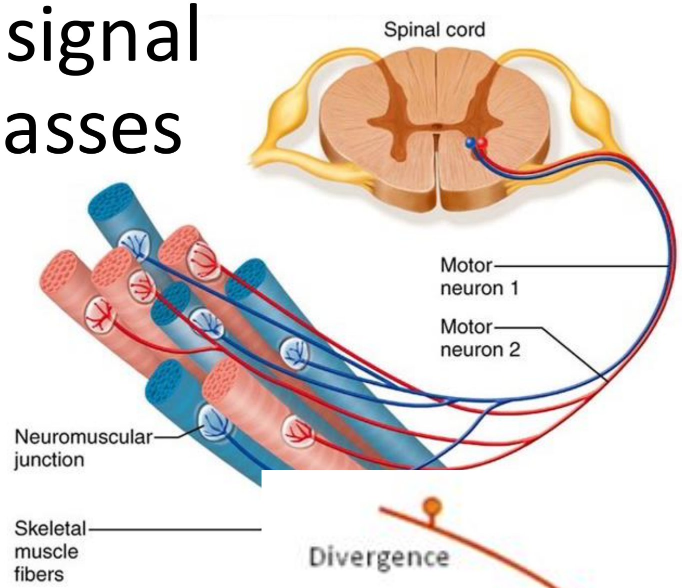
Divergence into multiple tracts



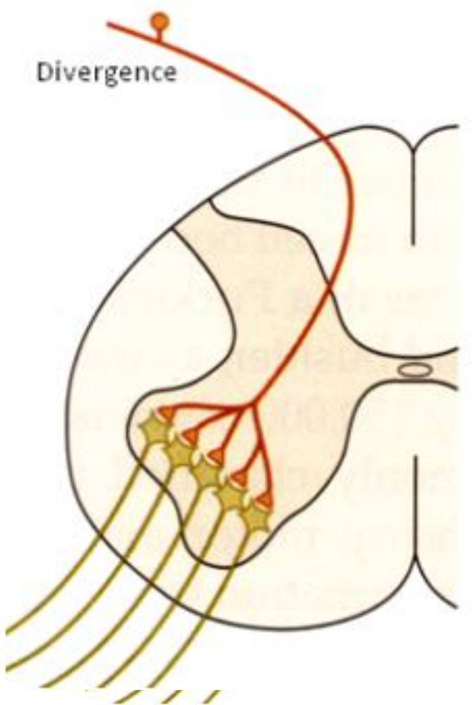
You also need to coordinate that with movements of your eyes so you can fixate that on special visual field so you don't fall down
also you need to send these signals to the cerebral cortex so you can be consciously aware of the movement of your head.

Amplifying divergence

- Amplifying divergence means simply that an input signal spreads to an increasing number of neurons as it passes through successive orders of neurons in its path.



Divergence in the same track is important to serve a function of amplification of the signal.
For example, in the motor neurons which are the neurons that are gonna supply this skeletal muscle fibers, you can see if there is one motor neuron for each muscle fiber, we will have to have like thousands to millions of motor neurons which is not efficient, so the other way is that the same motor neuron will branch several time to diverge into multiple muscle fiber cells that act as a one unit called the "motor unit" to contract at the same time and provides a more pronounced type of movement or more effective movement.

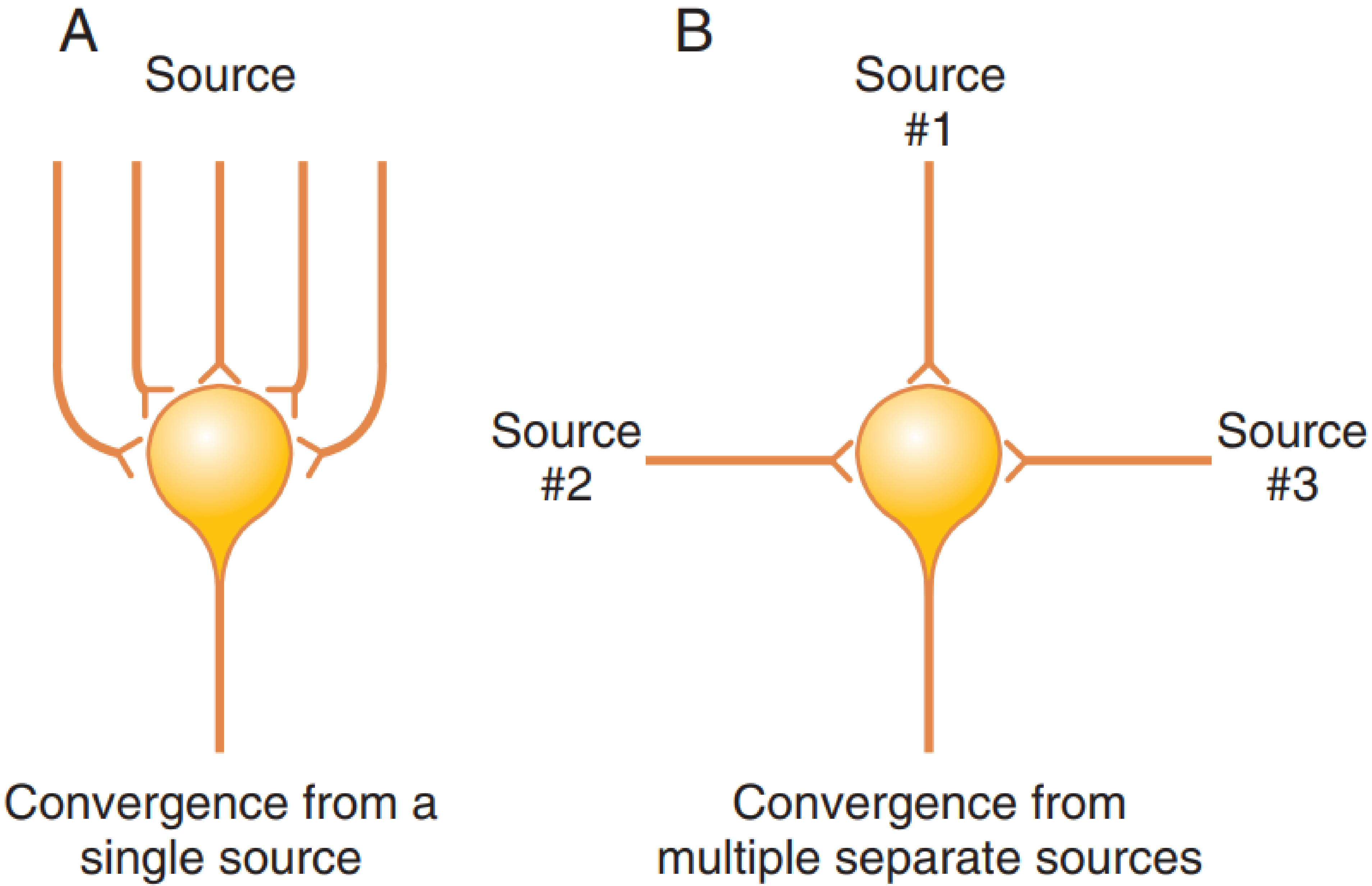


Convergence

- Convergence means signals from multiple inputs uniting to excite a single neuron (one output).

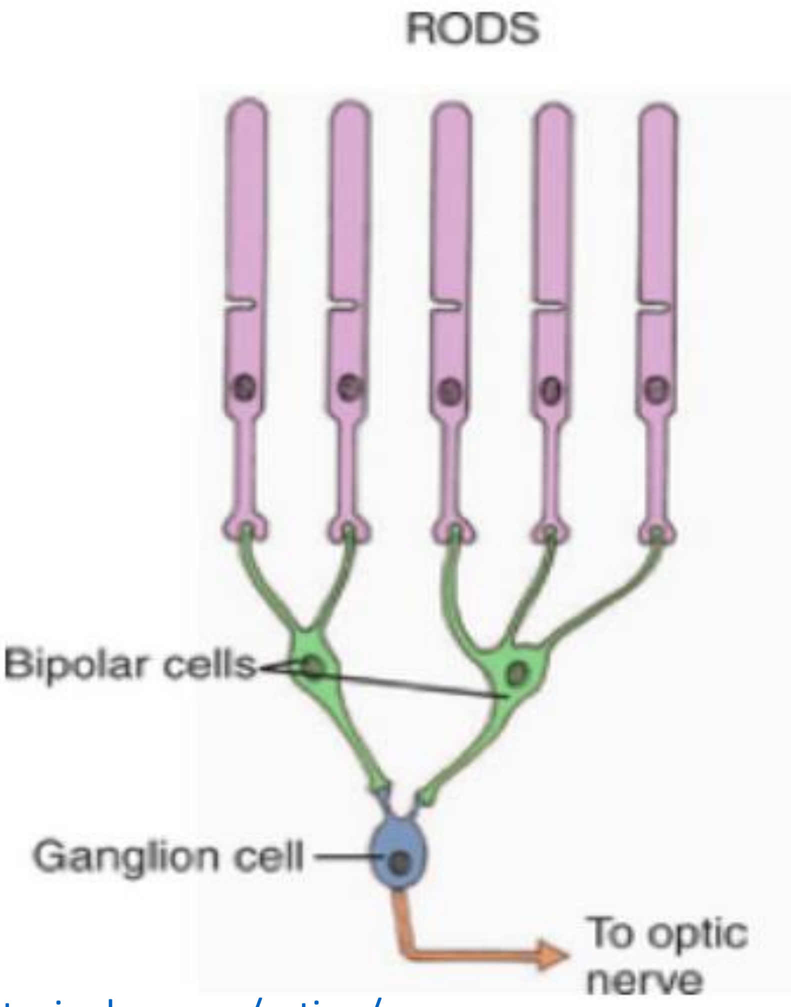
- The importance of this type is summation.

- Convergence is one of the important means by which the central nervous system correlates, summates, and sorts different types of information


A
Source
B
Source
#1
Source
#2
Source
#3
Convergence from a
single source
Convergence from
multiple separate sources

Convergence in photoreceptors (Rods)

- An important example of convergence is what happenes at the level of the retina in our eyes, we have photoreceptors called **rods** (for wihte-black contrast, while the cones are for colors and bright vision) **which** help us see in the dark, so it's very important for us in the dark to detect even the tiny changes of the light or the tiny stimuli from the light so these rods act like this so we can see better in the dark.. And what helps with that is the convergence on a single ganglionic cell, because this excitatory or this postsynaptic potential that's occurring at the level of the receptor is most likely not enough to cause a firing of the action potential to reach a threshold, however, if multiple (summation) potentials converge on the same postsynaptic neuron it might be enough to reach a threshold and reach the cerebral cortex therefore we will be consciously aware of the changes of the light during the dark.



http://veteriankey.com/retina/

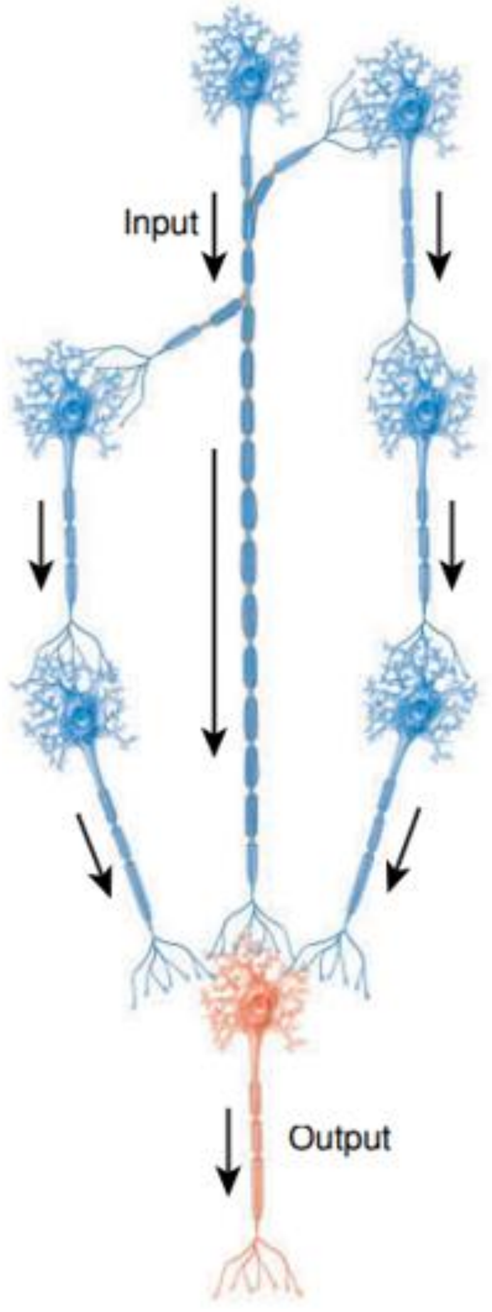
After-discharge

- A signal entering a pool causes a prolonged output discharge.

- **Synaptic afterdischarge**: as in some neuropeptides.

Parallel after-discharge

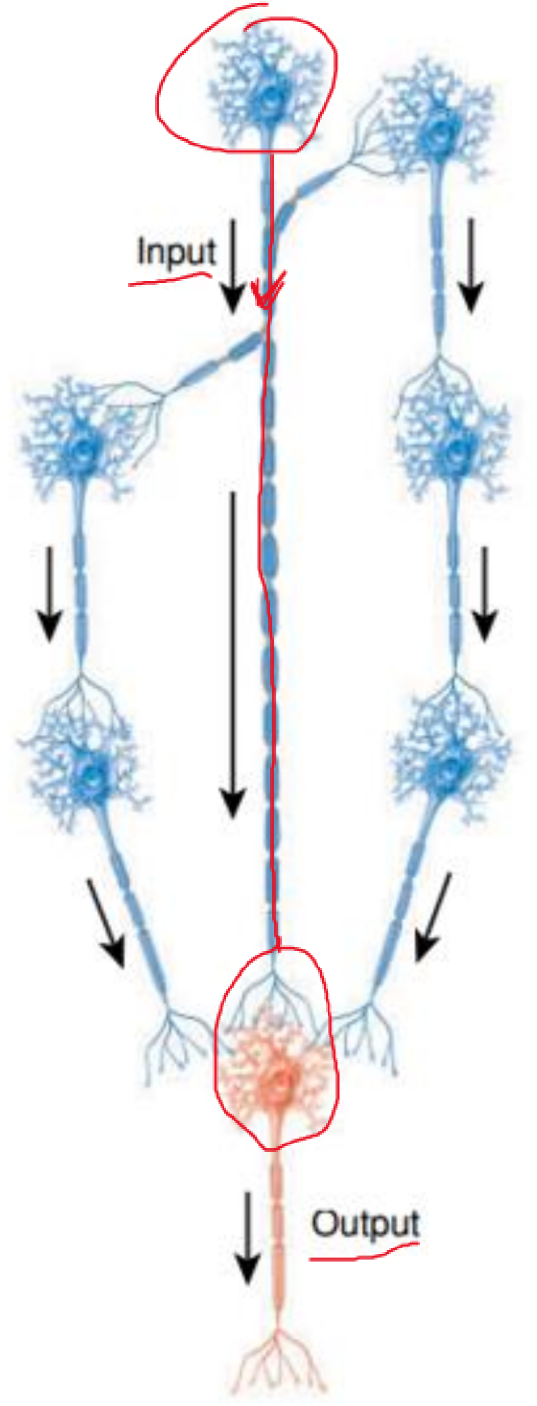
- Continued firing after the stimulus has stopped, so prolonged output discharge.
- a neuron inputs to several chains of neurons.
- Each chain is made up of a different number of neurons, but their signals converge onto one output neuron.
- Reach output at varying times.
- No feedback loop as in the reverberating circuit.



Saladin, K. *Human anatomy* (3rd ed.). McGraw-Hill. p. 364. ISBN 9780071222075.

We know that we can do prolongation of the effect if the neurotransmitter act on the metabotropic receptors rather than an ionotropic receptor, so that will cause a longer action on the postynaptict neuron, and another example of elongation signal is something we call it **parallel after discharge**.

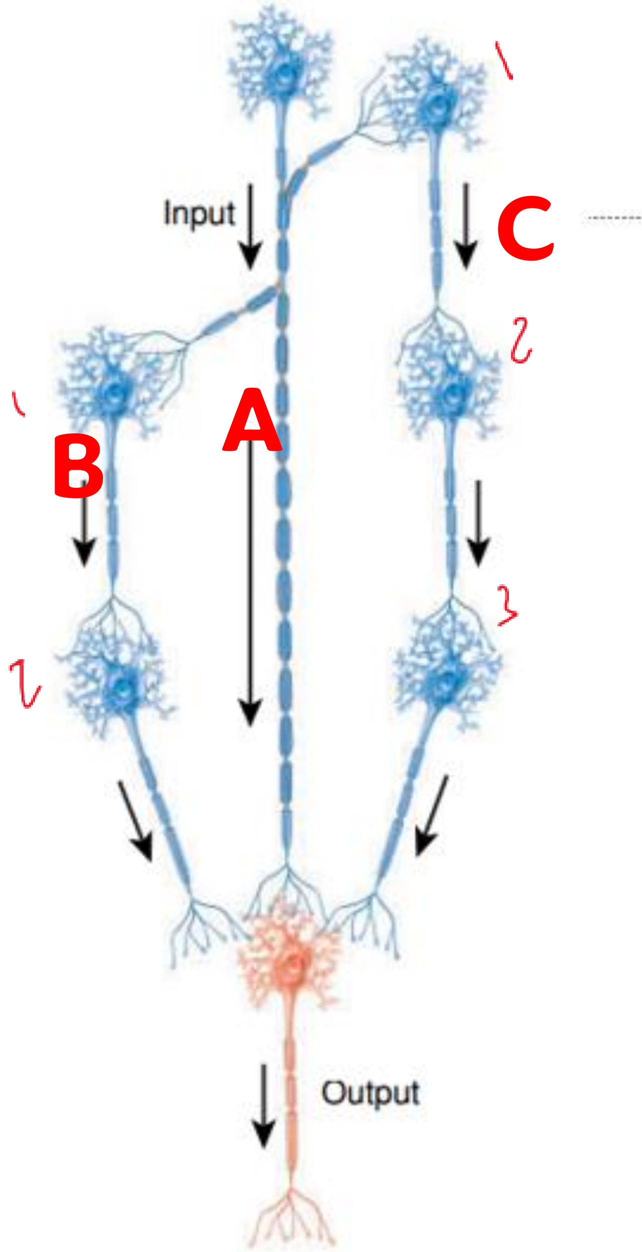
Simply, if we're talking about this neuron as a first neuron, it's a firing neuron so it's sending the signal through this synapse with the second neuron, so signals from the input neuron (look at the photo) to the output neuron, but if we need this signal reaching (in the output neuron) to be prolonged, I need this neuron to fire for a longer time But the signal that is coming from the input neuron on only lasts for a few milliseconds then it stops..



One of the mechanisms that the nervous system does is that we do branches collateral of the same neuron that's firing and what characterizes these branches that they're gonna snap with **different numbers of neurons** so basically this is a direct synapse between the blue neuron with the orange neuron (A) but there is also two entering neurons in between (B) and there are three entering neurons in between (C).

What does that mean, as we know that the principle of delayed transmission at the level of the synapse, so if you are increasing the number of synapsing, you are delaying the effect or the signal more and more.

This type of circuits is common in the learning, progress and memory, or when you're doing something and you want to remember the last step you've done just a few seconds ago



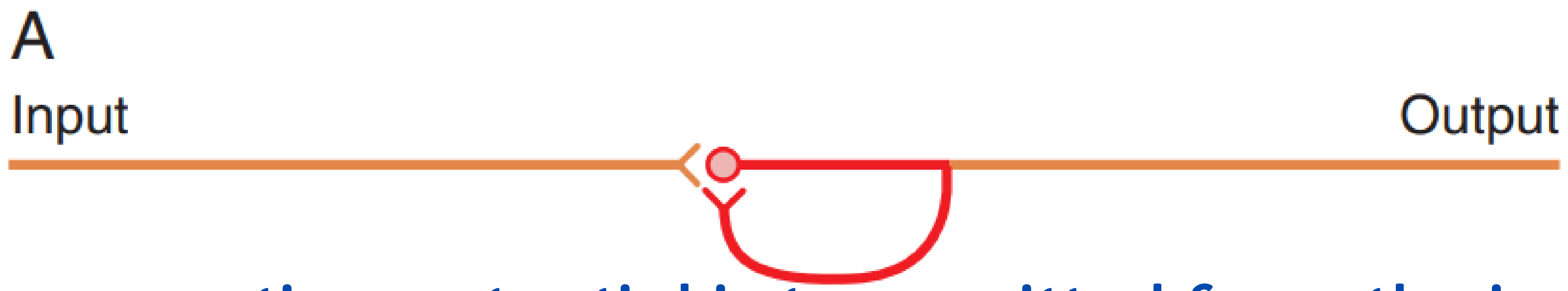
Reverberatory (Oscillatory) circuits

- One of the most important circuits in the nervous system.

- Caused by **positive feedback** **(which is different from the parallel afterdischarge)**within the neuronal circuit that feeds back to re-excite the input of the same circuit.

- Consequently, once stimulated, the circuit may discharge repetitively for a long time.

The output neuron sends a collateral nerve fiber back to its own dendrites or soma to restimulate itself.

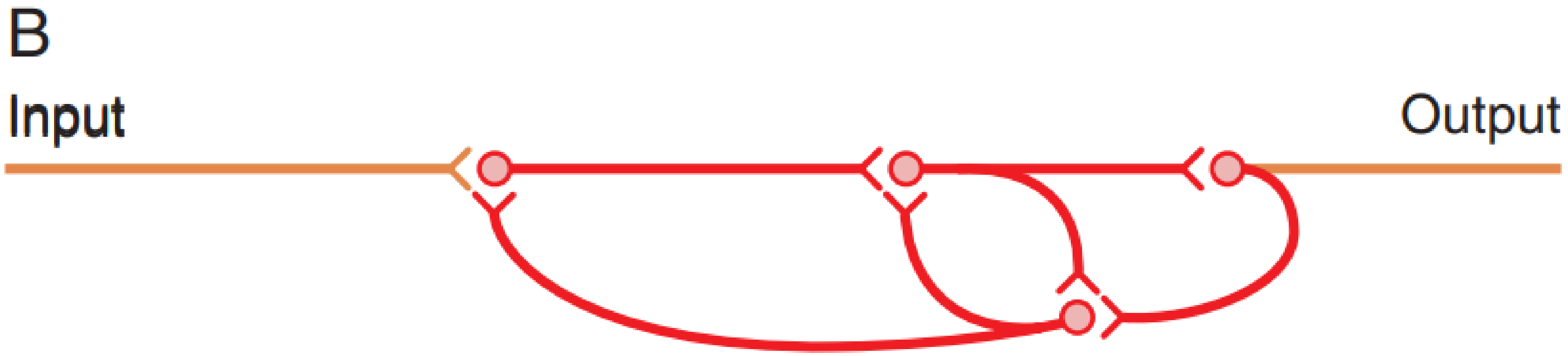


In this case, action potential is transmitted from the input neuron to the output neuron which will fire an action potential, but let's say that nervous system need this neuron almost continuously or to prolong this output from the neuron, so in this case there is a collateral (a branch) coming out from the same neuron (the output neuron) and again it's synapsing with the same neuron (as you can see from the example above) There's a collateral coming out and its axon terminals synapses with either the dendrites or the soma of the same neuron, so again another action potential will continue, and that will continue until something happens in the nervous system which we'll talk about later.

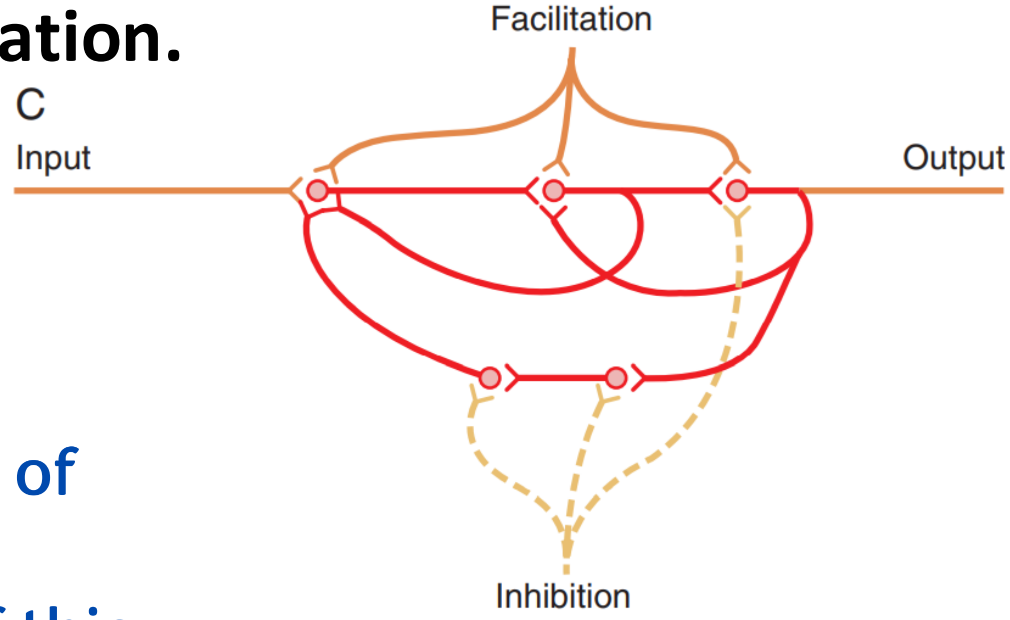
Reverberatory circuits

~They can get much more complex

A few additional neurons in the feedback circuit, which causes a longer delay between initial discharge and the feedback signal. Could be used in locomotion (walking)



A facilitatory signal enhances the intensity and frequency of reverberation, whereas an inhibitory signal depresses or stops the reverberation.



This example shows you that we need these reverberatory signals for continuous type of signaling that we need all the time, but circumstances can happen and changes in the environment, that we need to modulate the rate of the signaling in this pathway or changes in this pathway either we by facilitation or inhibition of this pathway

For example let's say this is a kind of reverberatory circuit controls the respiratory rate in our body, as you need to breathe all the time you need these signals all the time coming but sometimes let's say you are exercising so you want to increase your respiratory rate so you're gonna activate the facilitation kind of circuit to increase the respiratory rate to breathe more oxygen for your muscles to exercise.
Sometimes you need the inhibition to decrease this respiratory rate.

Continuous signal output

- Some neuronal circuits emit output signals continuously, even without excitatory input signals.

- At least two mechanisms can cause this effect:
- (1) continuous intrinsic neuronal discharge
- (2) continuous reverberatory signals

It's resting potential is very close to the threshold point. So it can generate an action potential by itself even without a stimulus like the cardiac muscles

Stability of neuronal circuits

- Almost every part of the brain connects either directly or indirectly with every other part, which creates a serious challenge.

- Two basic mechanisms that stabilize the central nervous system:

- (1) inhibitory circuits
- (2) fatigue of synapses.

*very important for patients with the generalized type of epilepsy when they have seizure attacks they might have these tonic-clonic attacks for seconds or maybe minutes but then it stop due to the fatigue that happens in the nerves system

Stability of neuronal circuits

INHIBITORY CIRCUITS

Two types of inhibitory circuits in widespread areas of the brain help prevent excessive spread of signals:

(1) inhibitory feedback circuits that return from the termini of pathways back to the initial excitatory neurons of the same pathways (like in sensory nervous pathways).

Stability of neuronal circuits

INHIBITORY CIRCUITS

(2) some neuronal pools that exert gross inhibitory control over widespread areas of the brain (for instance, many of the basal nuclei exert inhibitory influences throughout the muscle control system).

-We have a specialized structures in our brain. They are called basil nuclei, and they send inhibitor signals to the cerebral cortex (the motor cortex that control our skeletal muscle) so in case of impairment in the function of these basal nuclei one of the manifestation that will cause rigidity in the muscle (which is excess stimulation) this muscle will interfere with your daily activity because it is abnormal to be excited all the time.

Fatigue of synaptic transmission

- Depletion of transmitter stores.
- Progressive inactivation of postsynaptic membrane receptors.
- Slow development of abnormal concentrations of ions inside the postsynaptic neuronal cell.

-It can occur at different levels specially at the synapses.. One reason of the mechanism is the depletion of of neurotransmitters, which means we have no more neurotransmitters available to be released. Another reason could be changes in the membrane potential of the postsynaptic neuron, which makes it not responsive to the signal. Also saturation of the receptors in the postsynaptic can happen.

Effect of alkalosis on synaptic transmission

- **Most neurons are highly responsive to changes in pH** of the surrounding interstitial fluids.

- **Alkalosis increases neuronal excitability** and may cause cerebral **epileptic seizures**.

- In a person who is predisposed to epileptic seizures, even a short period of hyperventilation, which lowers CO_2 and elevates the pH, may precipitate an epileptic attack.

If someone had a panic attack, he will breathe very fast which washes out CO_2 from his body. This increases the Ph level in the blood aka “alkalosis” that’s why if someone had a panic attack we give him a paper bag to let him inhale the CO_2 that He have just exhaled to maintain the CO_2 levels in the body. (we shall have a full topic about alkalosis and acidosis in the biochemistry course the next semester 🙂👍)

Effect of acidosis on synaptic transmission

- Conversely, **acidosis greatly depresses neuronal activity**; a fall in pH may cause a comatose state (for diabetic ketoacidosis patients)

- For instance, in very severe **diabetic or uremic acidosis, coma** almost always develops.

Effect of hypoxia on synaptic transmission

- Neuronal excitability is also highly dependent on an adequate supply of oxygen.

- **Cessation of oxygen for only a few seconds can cause complete inexcitability of some neurons**.

- This effect is observed when the brain's blood flow is temporarily interrupted because within 3 to 7 seconds, the person becomes unconscious.

Stroke, patients who had their brain's blood flow interrupted for a long time would have an irreversible damage to their neurons in the brain
There is a case called transient ischemic attack TIA which is a mini attack caused by a temporarily blood flow blockage, when a patient suddenly lose the ability to talk, fall, lose his consciousness

Effect of drugs on synaptic transmission

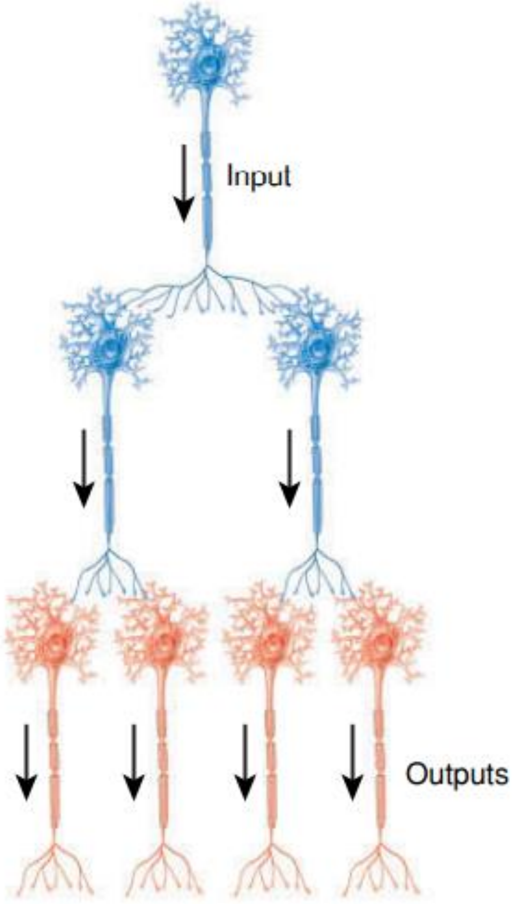
- Many drugs are known to increase the excitability of neurons, and others are known to decrease excitability.

- For instance, **caffeine, theophylline, and theobromine**, which are found in coffee, tea, and cocoa, respectively, all **increase neuronal excitability**, presumably by reducing the threshold for excitation of neurons.

Effect of drugs on synaptic transmission

- Most **anesthetics** increase the neuronal membrane threshold for excitation and thereby **decrease synaptic transmission** at many points in the nervous system.

*Have a look at these examples and determine what type of neuron circuits are they? Check the answers from the slides before.



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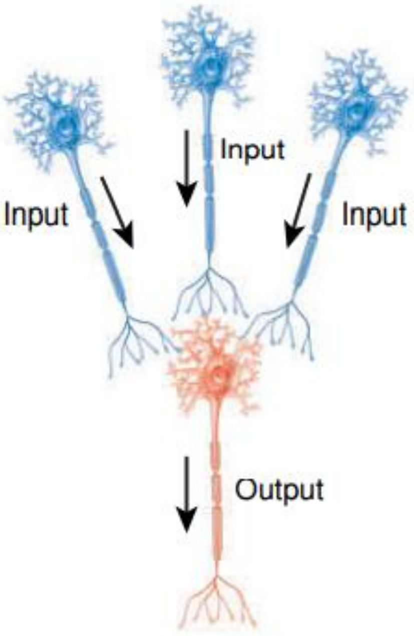

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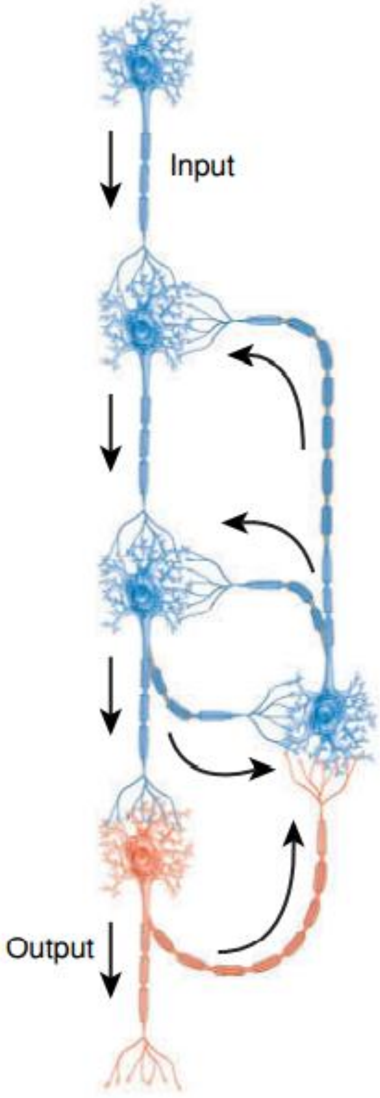

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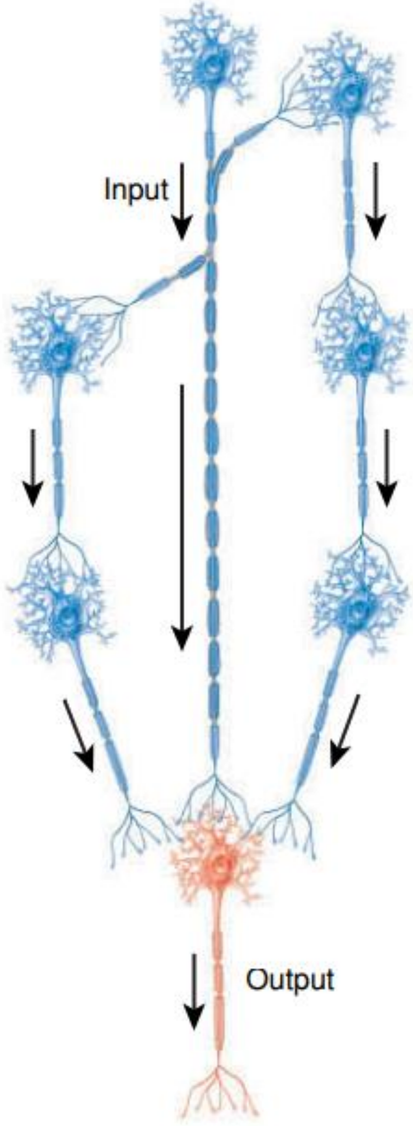

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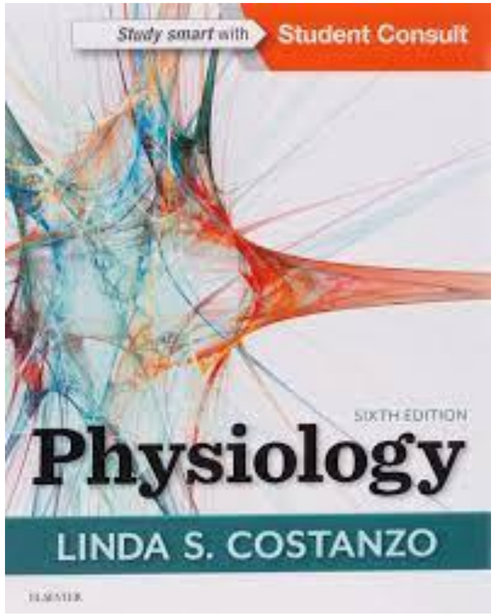
References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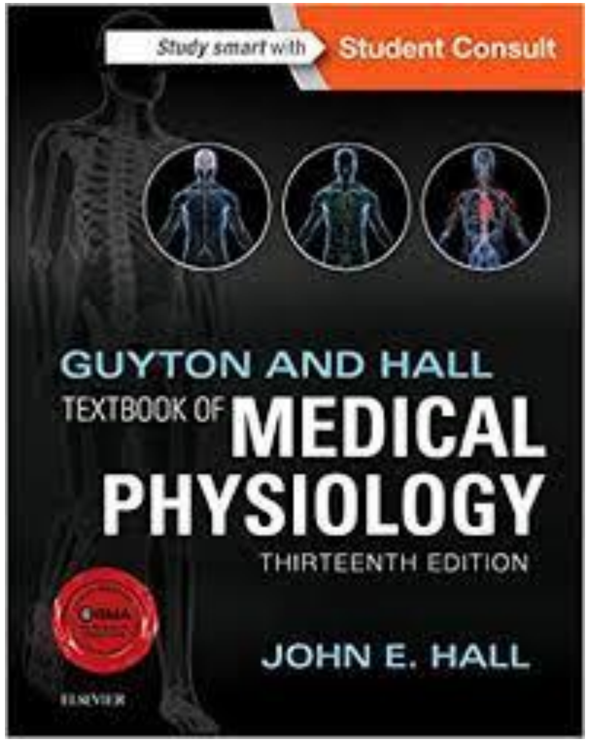
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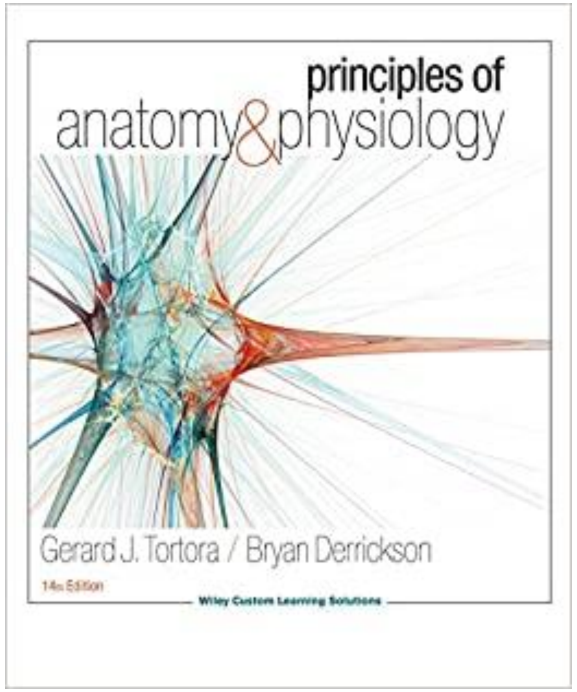
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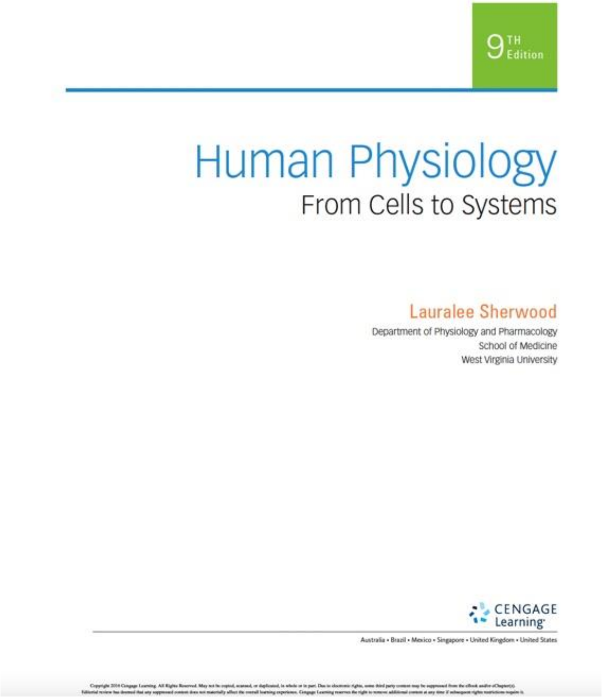
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Additional Resources:

رسالة من الفريق العلمي:

Reference Used:
(numbered in order as cited in the text)

1. Dr. Fatima’s slides

Extra References for the Reader to Use:

1. Check the interactive videos on the e-learning

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