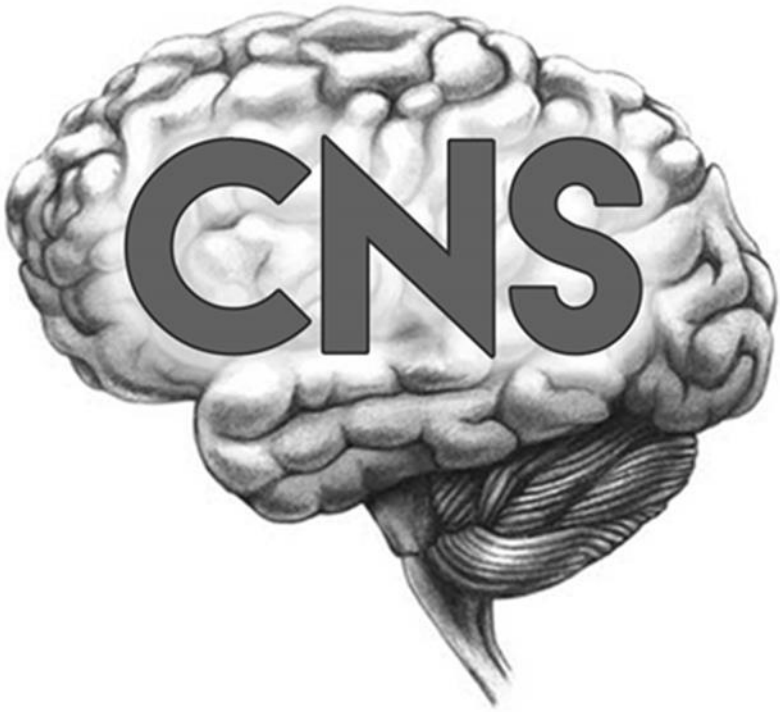

PHYSIOLOGY

☑ Sheet ○ Slide ○ Handout

Number

10

Subject

Sensory System

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Date: 27/2/2017

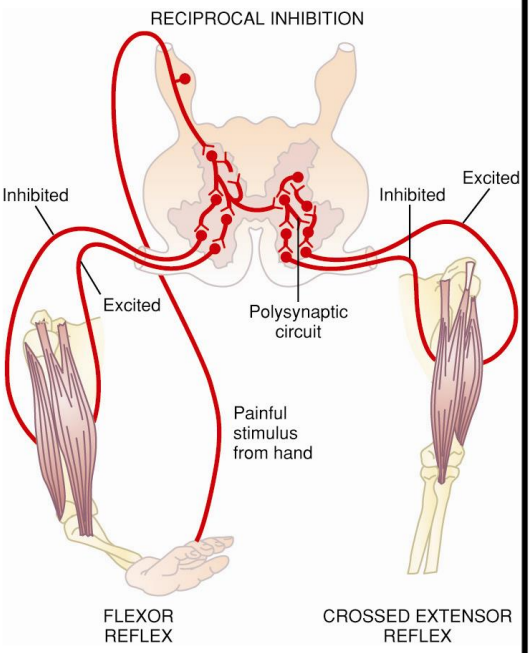
Price:

- This sheet is written according to sec. 1 recording.

Revision from the last lecture

☆ Crossed extensor reflex:

- This reflex occurs on the contralateral side of flexor reflex, which is opposite in action (develop extension on the other side to prevent the body from falling down)



- The crossed extensor reflex is slower than the flexor reflex, because of something called synaptic delay, the duration of which is around 0.5 milliseconds in every synapse, so it makes sense for the crossed extensor reflex to be slower since it has more synapses.

- A pain stimulus starts in the pain receptors in the skin and the impulse travels through the sensory neuron to the spinal cord, in the spinal cord it ascends or descends 1 or 2 segments via ascending and descending interneurons, the flexors are then excited and the extensors are inhibited ipsilaterally, while the opposite happens contralaterally, notice that there are too many synapses.
    - This dual stimulatory and inhibitory activity is called reciprocal innervation.
- The crossing from one side to the other happens anterior to the central canal, this is very important as the central canal might enlarge (a disease called **Syringomyelia**, enlargement of the central canal) and it might destroy these crossed fibers so you might lose the crossed extensor reflex.
- *Notice that the stimulus that caused the flexor reflex is the same one that caused the crossed extensor reflex.

Syringomyelia is a condition characterized by a fluid-filled cavity or cyst known as a syrinx that forms within the spinal cord.

Syringomyelia is a chronic condition and a syrinx can expand over time compressing or destroying the surrounding nerve tissue.

After Discharge:

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

Example: when staring at the sun briefly then closing your eyes, you may still see the last image because of this after discharge “EPSP” *(receptor potential is still above threshold),* And as long as the cortex is still receiving this action potential it will “think” that there’s still an image,

*The more the After Discharge, the longer is the signal, the longer is the reflex.

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

Nerd's Area

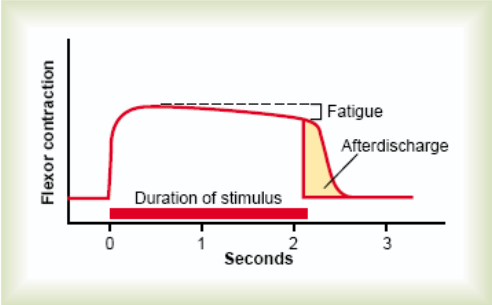


Figure 54–9

Myogram of the flexor reflex showing rapid onset of the reflex, an interval of fatigue, and, finally, afterdischarge after the input stimulus is over.

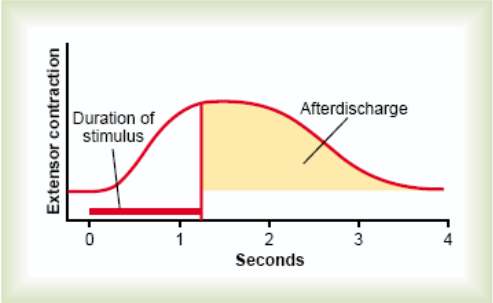


Figure 54–10

Myogram of a crossed extensor reflex showing slow onset but prolonged afterdischarge.

This is the myogram of the crossed extensor and flexor reflexes:

- The flexor reflex is faster than the crossed extensor reflex, because as we said, it doesn't contain as many synapses.
- Notice how the after discharge is much longer in the crossed extensor reflex than it is in the flexor reflex, because it contains more synapses.

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

Babinski reflex:

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

Reflexes that Cause Muscle Spasm:

- Rigid abdomen ➔ emergency case, occurs due to perforated appendix "visceral reflex" (muscle will spasm and become rigid)

**pay attention to this as it's not caused by basal ganglia or decerebrate rigidity!!*

Sensory System

Transducers:

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

labeled line principle. A hypothesis to explain how different nerves, all of which use the same physiological **principles** in transmitting impulses along their axons, are able to generate different sensations.

- these receptors are sensitive to “adequate stimulus”
- in another words: the sensory receptors/neurons are sensitive to **quality** not quantity of the stimulus.

The **adequate stimulus** is a property of a sensory receptor that determines the type of energy to which a sensory receptor responds with the initiation of sensory transduction. Sensory receptor are specialized to respond to certain types of stimuli.

For example: touch receptor’s threshold for touch is very small yet to other stimulus high, which means that they can be stimulated by other stimulus but it's very hard to do so because they are specific for touch ,

Can pain receptors be stimulated by thermal stimulus?

Yes, but we need high temperature to do so, around 55-60

This is what we call adequate stimulus !

Coding Mechanism:

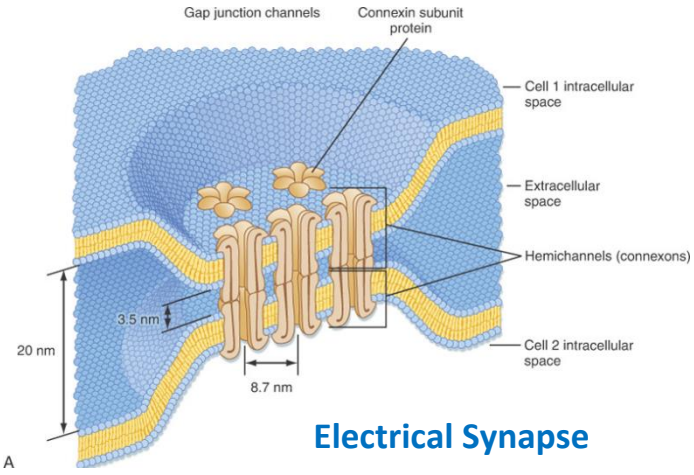
What should the cortex know about the stimulus that has reached it?

- The Intensity, location and modality “type of the sensation”

❖ Modality is coded by type of the type of receptor and the special pathway.
❖ Location is coded by special pathway, and the he body is represented on the cerebral cortex sensory part (postcentral gyrus), It is also upside down and the representation size is proportional to the number of receptors “density of receptors“
  » Density of receptors refers to the **finesse of sensation** (accuracy of two points discrimination), like in fingertips if there was two points 1mm apart we can still feel them as 2
  » While in the back we can't discriminate between these two points if they were less than 50 mm (5cm) apart!, due to low density receptors in that part of the body
  » that's why the back is represented in the cortex in a very small part while the face and fingers have very large areas
  » so higher number of receptors leads to “accurate sensation"

The Synapses:

There are two types of synapses:
Electrical and **Chemical**



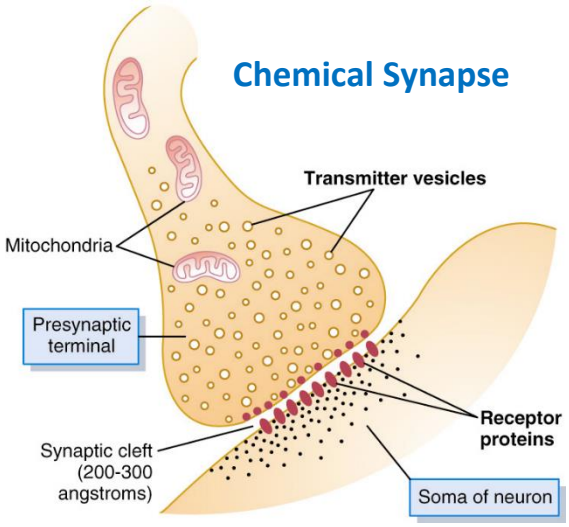
Electrical Synapse

Electrical synapses:

- there is physical continuity between the pre- and post- synaptic neurons
- they have "voltage gated" channels between them "as gap junction", responsible for ionic current spreading between the two cells
- when these channels open, the ions go to both sides "bidirectional" according to their gradient, (two way transmission)
- no neurotransmitters involved, and there's no synaptic delay
- These electrical synapses are found in the CNS only! "not found in the PNS"

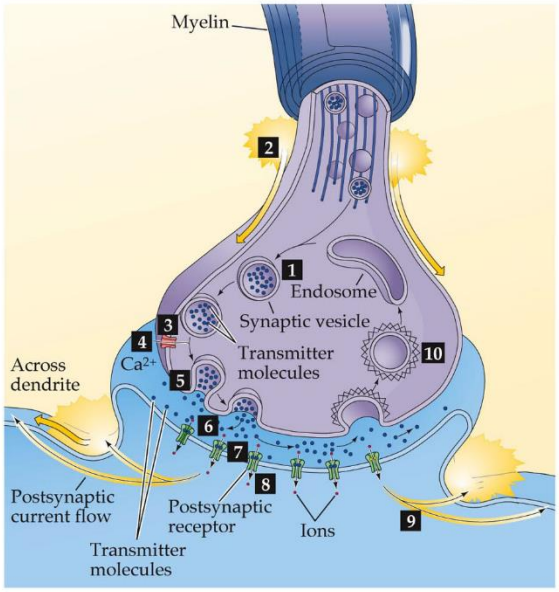
Chemical synapse:
"the classical well know synapse"

- Axo-axonic, axo-dendritic and axo-somatic
- there is synaptic cleft and synaptic delay
- it is one way information transferring synapse *"from pre to post synaptic neuron"*



- Once there's Action potential in the presynaptic neuron neurotransmitters will be released, and when they pass through the synaptic cleft and reach their receptors in the post-synaptic membrane they change its permeability! (either increase or decrease) resulting in hyperpolarization or depolarization
- if it was depolarizing, it causes EPSP *"excitatory postsynaptic potential"*, gets the membrane closer to threshold,
- if it was hyperpolarizing, it causes IPSP *"inhibitory postsynaptic potential"* → moves the membrane away from threshold "inhibitory!"

Mechanism (in details):



Action Potential occurs in presynaptic membrane ➔ Ca^{++} channel open ➔ Ca^{++} move inside according to electrochemical gradient ➔ vesicle fuse to membrane ➔ neurotransmitters released ➔ go to receptors ➔ causes either EPSP or IPSP occurs

Ca inside the neurons is about 10^{-7} M

Outside "in blood + interstitium" 10^{-3} "$2.5*10^{-3}$ is the outside but half of it is bound to albumin so we care about the free part only which is near $1*10^{-3}$"

↳ NeuroTransmitters:

There are two classes of neurotransmitters

a- rapidly acting small molecules (acetylcholine, epinephrine, norepinephrine, GABA, glycine, glutamate aspartate and NO gas)
b- neuropeptides or neuromodulators

- Each neuron has only one type of rapidly acting neurotransmitters
- but it may have many types of neuromodulators!

What is the function of neuromodulators?

- They modulate the action of neurotransmitters
- Neuropeptides ➔ modulators

Ex: enkephalins (leucine or methionine, substance P, vasoctive intestinal peptides etc…

When we talked about the Basal ganglia, we said that the Substantia Nigra produce dopamine & substance P, substance P is a neuromodulator that modulates the action of dopamine "prolong its action!".

also in globus palidus (straitum) GABA is produced and enkephalins

Neurotransmitters recycling:

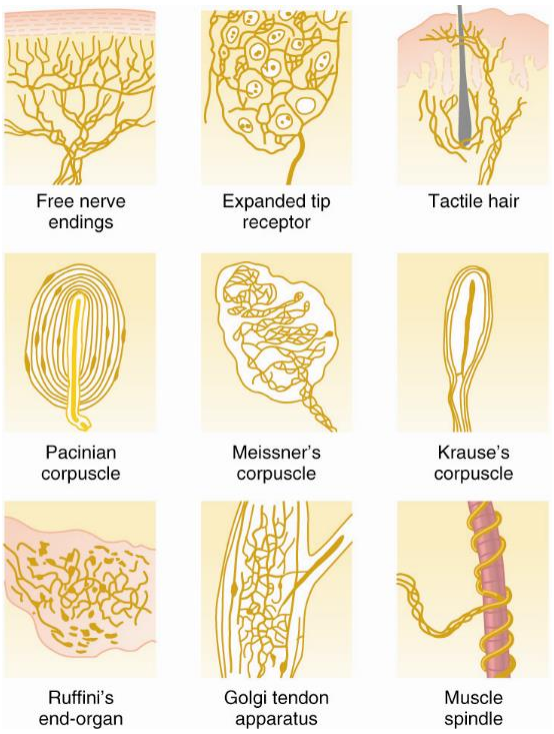
I. Reuptake of transmitters by presynaptic neuron etc
II. Enzymatic breakdown: estrase, mono amine oxidase "MAO"
III. Diffusion

- And neuromodulators they are peptides so they are recycled "degraded" by peptidases

Receptors:

Can be classified by their site (location) "inside or outside":

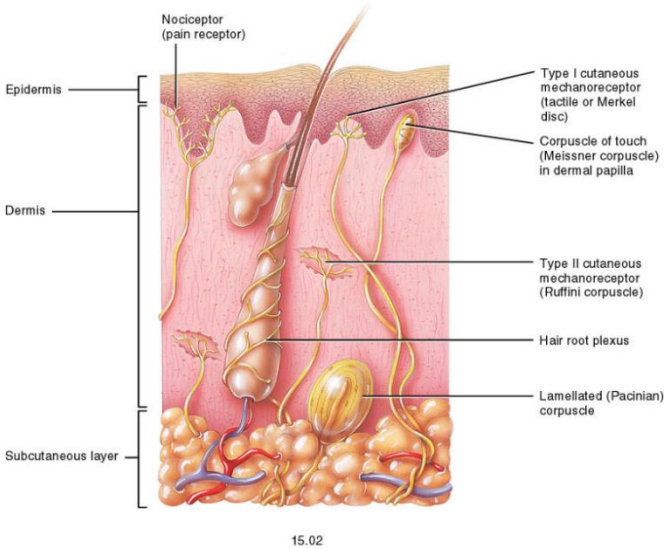
A. **Exteroceptors**: outside at or near the surface (pressure, touch, pain & temperature)
B. **Interoceptors:** of the viscera!
C. **Proprioceptors:** sense of position, like the Muscle spindle, golgi tendon and large tactile receptors around the joints



Or classified by types of sensation (modality):

A. Mechanoceptors: touch, pressure, sounds…
B. Chemoceptors: chemicals, CO2, O2, taste, smell…
C. Thermoceptors: temperature
D. Nociceptors: pain
E. Electromagnetic receptors (photoceptors)

- Generally receptors are of two types, either **separate** from the afferent neuron **OR** the **terminal part** of the afferent neuron
- But either way they are <u>non-excitable</u> tissues!
- Which means that they can't generate action potential (the only excitable tissues are muscles and neurons)



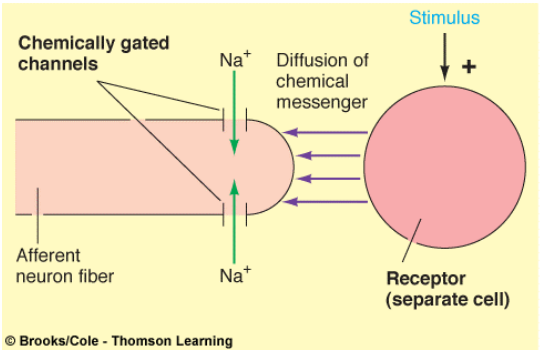
When these receptors respond to stimulus:

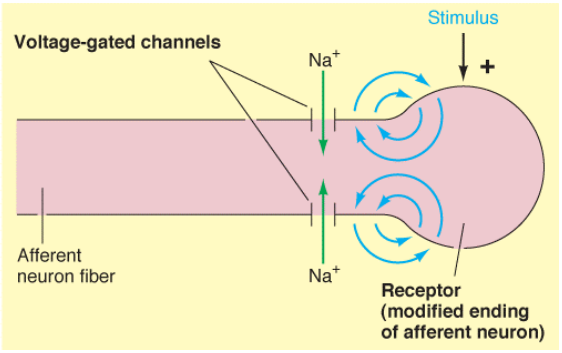
- **Change**! in the permeability occurs "either increase or decrease in permeability"
- which leads to change in the potential of receptor's membrane "**called <u>receptor potential</u> or <u>generator potential</u>"**

- Receptor potential and generator potential are vaguely the same, the only difference is that we call this potential generator potential **if the receptor is <u>part of the afferent neuron,</u> and if it's not part of it (separate) we call it receptor potential**

Example of separate and non-separate receptors:

a) crista ampullaris & b) utricle and saccule of the maculae in the vestibular system, the hair cells and the afferent fibers of the vestibular part of Vestibulocochlear nerve are separate from each other
c) while touch receptors are the terminal part of the nerve "non-separate"

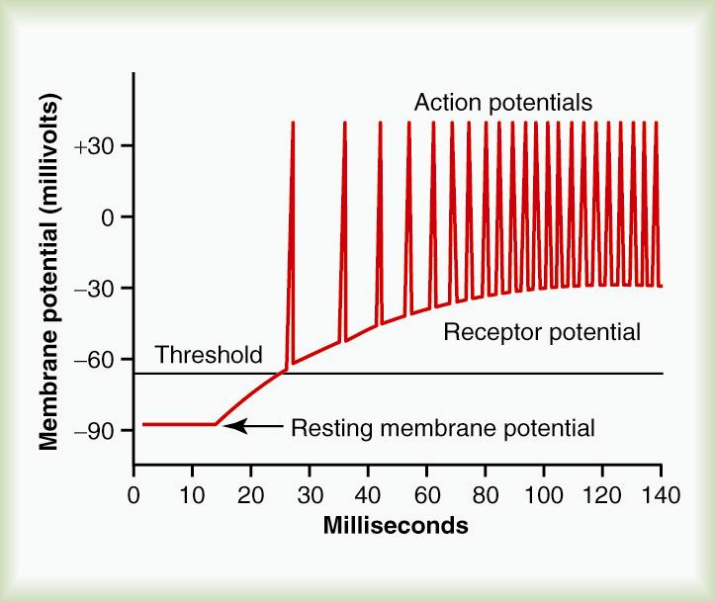




- Now, the stimulus opens **<u>stimulus gated channels</u>** , *"mostly sodium channels"* which causes depolarization!
- So the receptor potential might depolarization potential!
- If the sodium channels were initially open and the stimulus closed them, the receptor potential then will be hyperpolarizing
- So receptor potential could be either one of both!

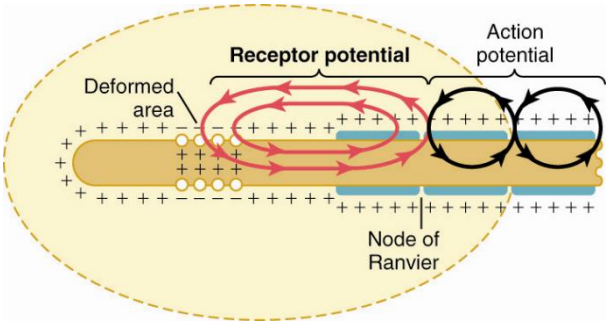
- So now we said that receptors has modality and sensitivity to the intensity of stimulus

  - Modality → specificity of the receptor to transduce the signal → "the labeled line principle"

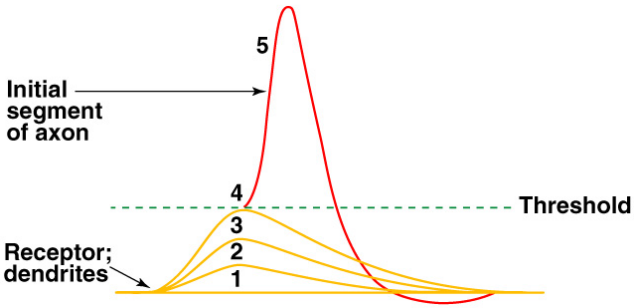


  - Stronger stimulus causes larger receptor potential!
  - Which Causes the membrane potential to rise instead from -70 → -40 for example, to rise from -70 → -20, which means more Action Potential
  - If the threshold was -40, and membrane potential reached -40, it generates action potential, and that action potential time is 10ms for example (hypothetically!) *"actually it is around 0.1-0.2 ms"* but for easier calculations let's consider it 10ms
  - Now if membrane potential was equal to threshold it gives 10ms, so the rate of discharge will be 1000ms/10ms ➔ 100 AP/s (1000ms is 1 second) *"this calculation means that there will be 100 action potentials each second!"*
  - If the receptor potential was above the threshold, it will stimulate the neuron at the relative refractory period!
  - if it reached 5ms, the rate will be 200 AP/s, and so on until we reach the absolute refractory period (let's consider it here as 1 ms) then the rate will be 1000 AP/s "maximum", and more increment won't increase this rate anymore, rendering it useless!
  - Now the rate of discharge depends on how large is the receptor potential is!
  - the larger the potential, the greater the rate,
  - and the stronger the stimulus the larger is the receptor potential,
  - this all means that (the strength of stimulus is coded for the number of impulses that reach the cortex) "aka rate",

Receptor excitation mechanism:



- mechanical deformation which stretches membrane and opens ion channels causes receptor potential
- application of chemical which opens ion channels
- change in temperature which alter permeability of ten membrane through changing metabolic rate
- electromagnetic radiation that changes the membrane characteristics



- Action potential is generated at the **first node of Ranvier**!
- first stimulus doesn't generate action potential because it doesn't reach the threshold
- yet it is **facilitative** , along with the 2nd & 3rd, and the 4th stimulus will cause action potential

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These are some quick information concerning the next lecture!
(Have a quick look at them!)

"The mechanism of receptor potential is the same as EPSP"

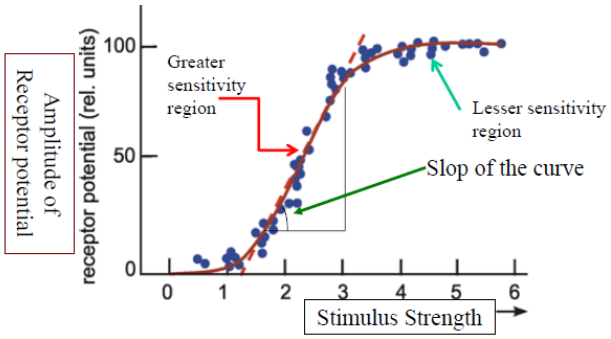
» (Higher receptor potential ➔ higher frequency of action!)
» Because we are closer to the relative refractory period, Relative refractory period is special that it can be stimulated, but the stimulus need to be stronger to do so!
» So we can increase the strength of stimulus until we reach the absolute refractory period, no benefit after that.
» Stimulus is stronger ➔ amplitude of receptor potential is higher!

What is the amplitude?

- We start membrane potential from -70, if it reaches -30 the amplitude is 40, and if it's -20 amplitude is 50 and so on…

- So now, high amplitude means strong stimulus!

- Also, high amplitude means high rate or frequency (number of impulses), because we are stimulating during the relative refractory period!
- So the high number of impulses to the cortex will make it consider this stimulus or sensation as intense (strong)



How about the location?

- As mentioned earlier, each part of our body is represented in the cortex and the representation of the sensory is similar to the motor counterpart
- Upside down and representation is proportional to the density of receptors

Types of neurons in the sensory system are A and C

A → I II III myelinated

C → IV unmyelinated

Le Fin.

"you're never too important to be nice to people"

– John Batiste

Done by: *Omar Saffar*