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Physiology of synapses and receptors

PHYSIOLOGY
CNS Block
Team 443

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Editing File

Objectives:

1. Define synapses and enumerate functions of synapses.
2. Classify types of synapses: anatomical & functional.
3. Draw and label structure of synapses.
4. Describe Synaptic transmission & neurotransmitters.
5. Explain the fate of neurotransmitters.
6. Describe the properties of synaptic transmission
7. Explain electrical events at synapses (EPSPs & IPSPs).
8. Explain factors affecting synaptic transmission.

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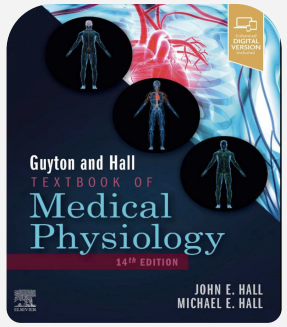


Highly recommended!

it's a Long one
but very Helpful!!

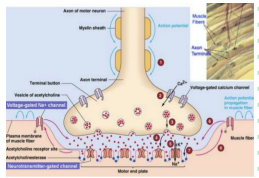
Synapse:

A connection (junction) where the axon or some other portion of one cell (**presynaptic cell**) terminates on the dendrites, soma, or axon of another neuron (**Postsynaptic cell**).

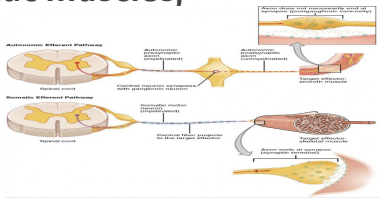
General Facts abouts Synapses:

- A connection between a neuron and a second cell, in the CNS this other cell is also a neuron, while in PNS this other cell maybe either a neuron or an effector cell eg;- gland or muscle.
- Information is transmitted in the central nervous system mainly in the form of nerve action potentials, called nerve impulses, though a succession of neurons.
- The CNS contains more than 100 billion neurons.
- Junctions (synapses) outside the CNS :

Neuromuscular junction between nerve and skeletal muscle



Contact between autonomic neurons and smooth (example: GIT muscles), cardiac muscles, and gland (Other effector).



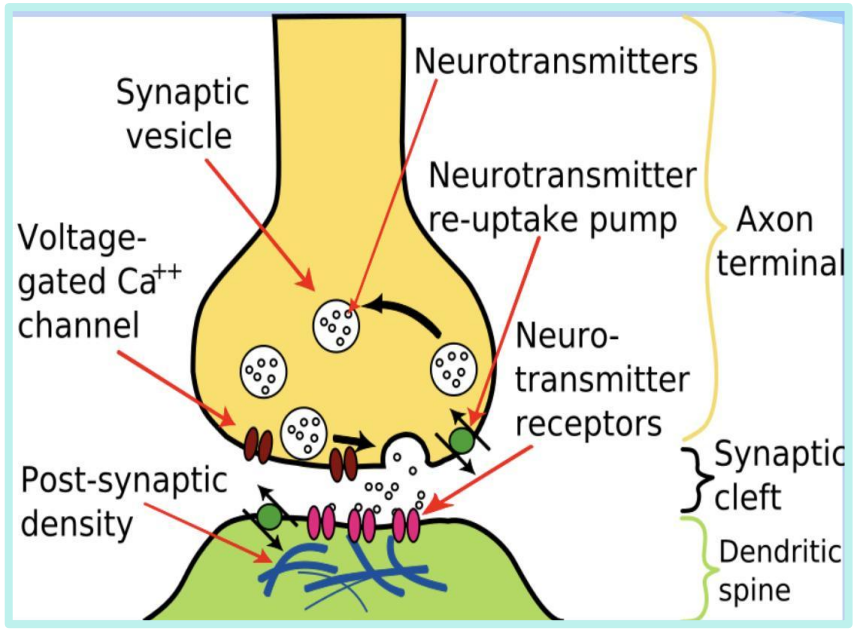
The synapse is between two neurons and the junction is between a neuron and other structure.
The more synapse in the neuron the higher the function quality .

Synaptic Structure:

1. **Synaptic knobs (presynaptic terminal):** It has synaptic vesicles (neurotransmitters).

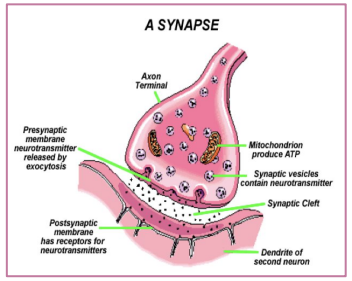
2. **Synaptic cleft:** It is the space between the axon terminal and sarcolemma. It has a width of 200-300 angstroms.

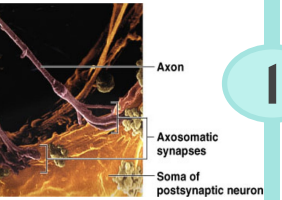
3. **Postsynaptic membrane:** It has receptors for neurotransmitters



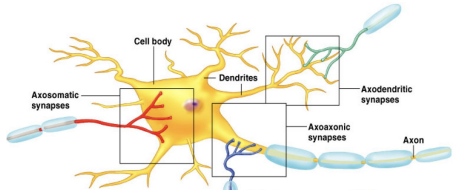
Synaptic Types

Anatomical





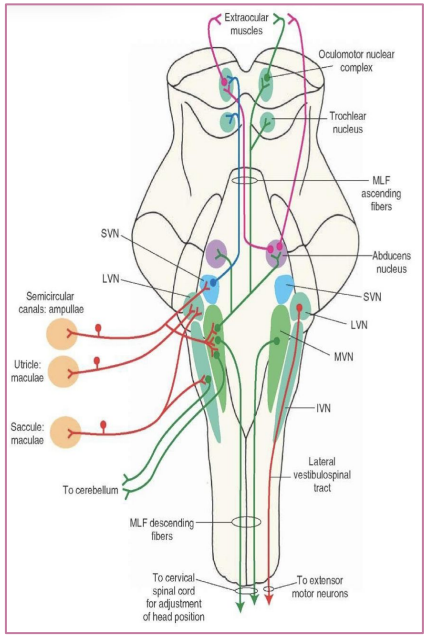
1. **Axosomatic:** Synapses between the axon of one neuron and the soma of another. (common in cerebellum)
2. **Axodendritic:** Synapses between the axon of one neuron and the dendrite of another.(common in cerebrum 98%)
3. **Axoaxonic:** Axon to axon.



4. **Dendrodendritic:** Dendrite to dendrite.
5. **Dendrosomatic:** Dendrites to soma.

Functional

1. **Chemical (Most)**
2. **Electrical**
3. **Conjoint (Few): Both electrical and chemical. Example: Neurons in lateral vestibular nucleus.**



CHEMICAL SYNAPSE

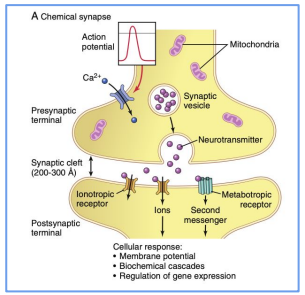
-Mechanism: first neuron secretes a chemical substance called **neurotransmitter** at the synapse to act on **receptor** on the next neuron to excite it, inhibit or modify its sensitivity.

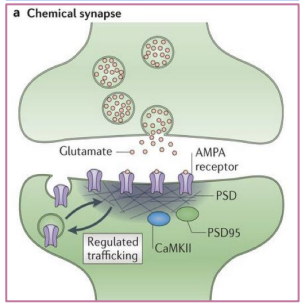
-There is cleft NTs

-Almost all synapses in the CNS

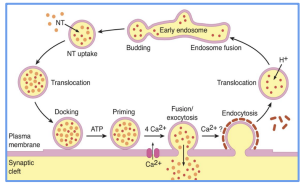
-**Amount** of NTs released depends upon **frequency** of AP (3 types of vesicles)

-more than 50 known transmitters





NTs is synthesized in the **body** and are released from synaptic vesicles (can be detected by immunohistochemistry):



there are two ways to release the NTs:

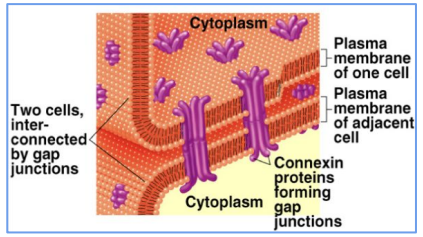
1- Vesicles fuse with axon membrane and NTs are released by **exocytosis**.

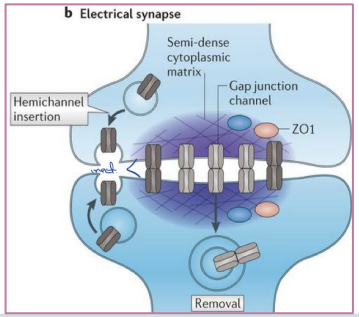
2-The synaptic vesicle can discharges its contents through a small hole in the cell membrane, then the opening reseals rapidly and the main vesicle stays inside the cell (**kiss-and-run discharge**).

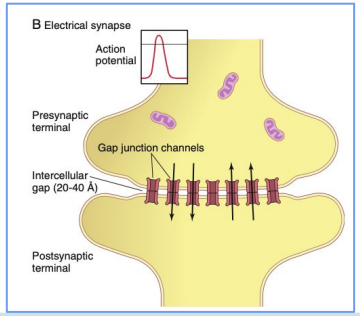
ELECTRICAL SYNAPSE

-Mechanism; Membranes of the pre- and postsynaptic neurons come **close together** and **gap junctions** form → low membrane borders which allow passage of **ions**.

-Correspond to gap junctions found in other cell types







-Impulses can be regenerated in adjacent cells electrically coupled through **GAP JUNCTIONS** composed of 12 connexin proteins without interruption.

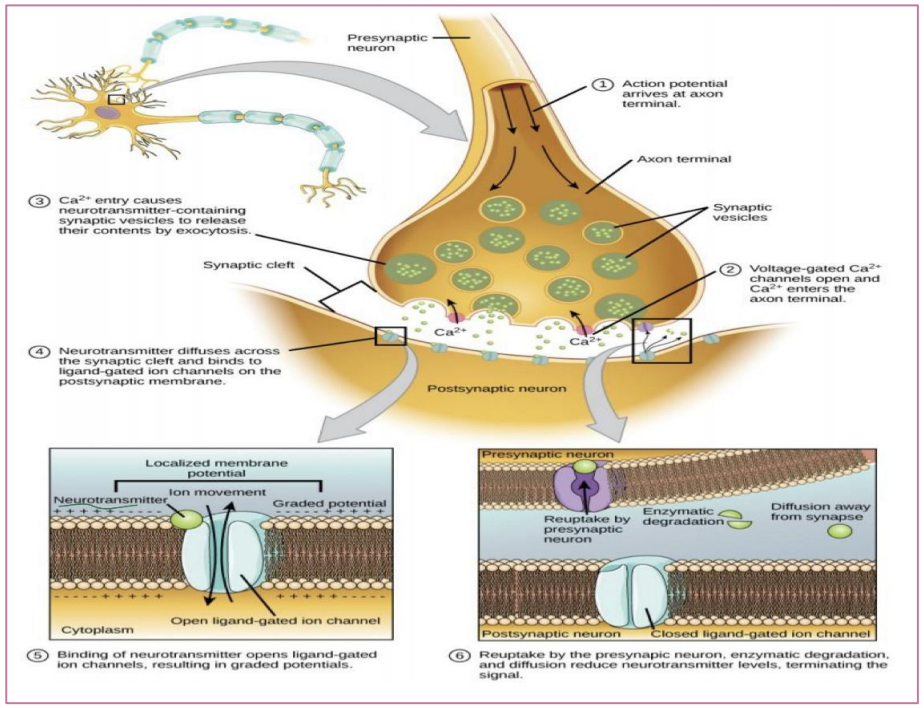
Examples: Smooth and cardiac muscles, brain, and glial cells.

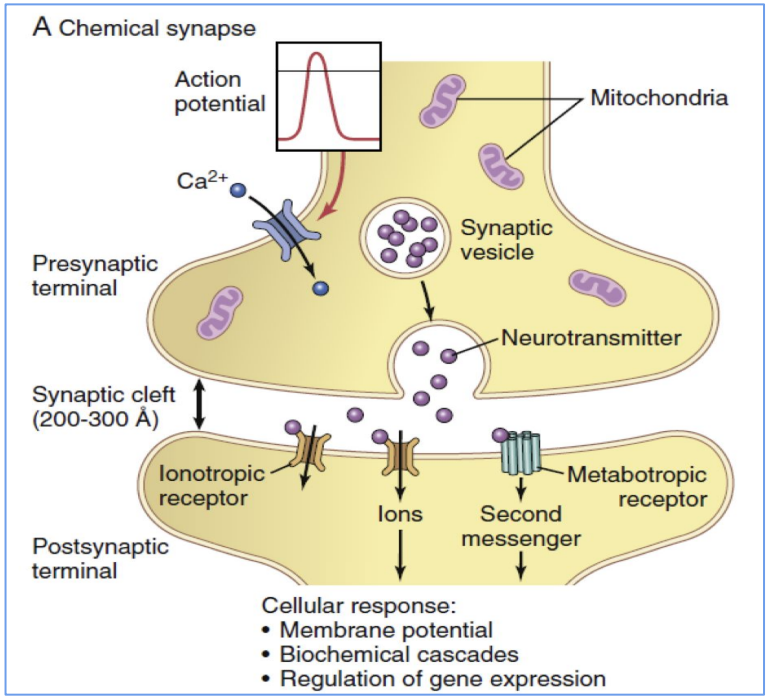
-**Electrical synapses** are present throughout the central nervous system specifically in neocortex, hippocampus, thalamic reticular nucleus, locus coeruleus, inferior olivary nucleus, striatum, cerebellum, olfactory bulb, retina, and spinal cord.

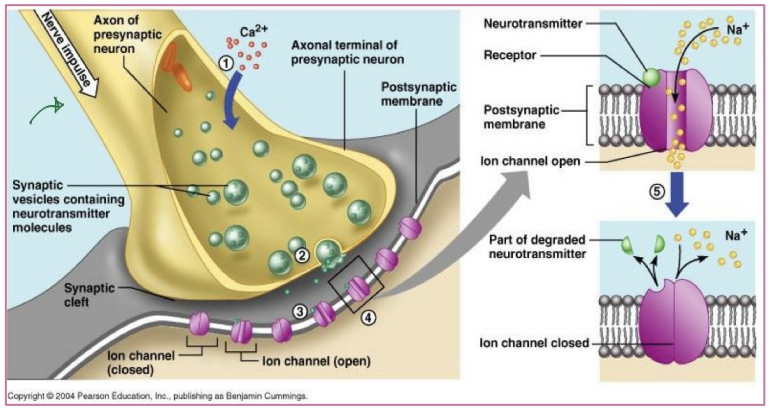
CHEMICAL SYNAPSE	ELECTRICAL SYNAPSE
→ Two separate cells that **do not** touch → Terminal button is separated from postsynaptic cell by synaptic cleft **20-40 nanometer** (200-300 A). → Exhibits **synaptic delay**, e.g.: at NMJ reveal a delay of **0.5 to 4.0 millisecond** → Slower → Act on receptors which are specific → More complex behaviors →The response may not be the same as the source. → The response in the postsynaptic neuron is **variable**.	→ Gap junctions are intercellular connection that **directly connect** the cytoplasm of cells → Cells approach within about **3.8 nm** of each other (20-40A) so the impulses can be regenerated without interruption in adjacent cells. → **Almost no delay** in transmission. → Faster, many neurons fire synchronously → No need for receptors to recognize chemical messengers → simple behavior → The response is always the same sign as the source. → signal in the postsynaptic neuron is the **same or smaller** than that of the originating neuron
-**"One-Way"** conduction at chemical synapses.(unidirectional) -More common than electrical synapses	- The **bidirectional** transmission of electrical synapses permits them to help coordinate the activities of large groups of interconnected neurons and promotes synchronous firing of a group of interconnected neurons. The bidirectional transmission is important to the **coordination** between the organs, also it is important in complex processes. - **For example, in mental attention, emotions and Memory, arousal from sleep**

Events in \ mechanism of chemical synaptic transmission

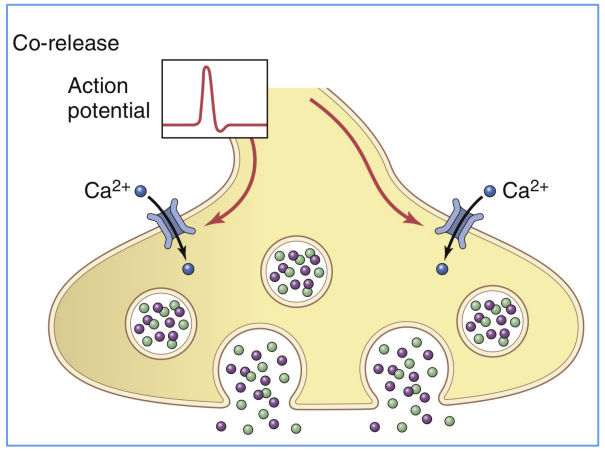
- AP travels down axon to bouton.
- VG Ca2+ channels open & Ca2+ diffuses inside bouton.
- Ca2+ triggers rapid fusion of synaptic vesicles at "docking site" and release of NTs (directly related to Calcium ions that inter the cell), [Ca2+ activates calmodulin, which activates protein kinase that aid in the fusion of synaptic vesicles]
- NTs (ligands) are released and diffuse across synaptic cleft & act on receptor proteins in postsynaptic cell membrane to produce effects. (inhibition or excitation of the postsynaptic membrane)
  - ((depending on the type of the neurotransmitter i.e. excitatory or inhibitory))

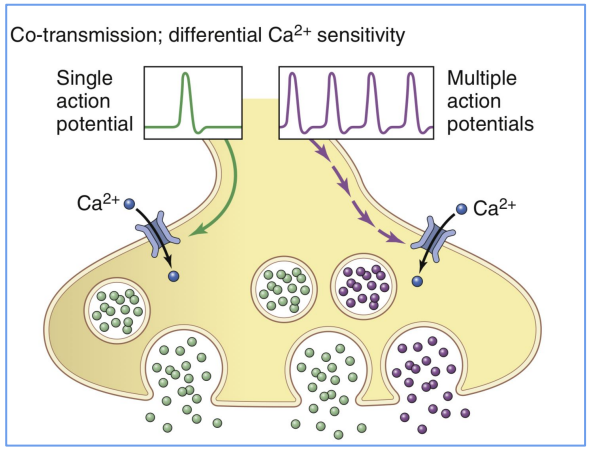


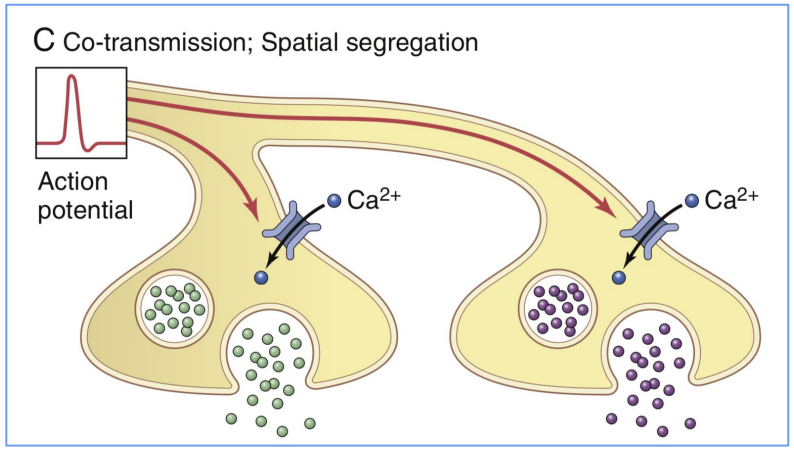




Co-release and Co-transmission.







Co-Release	Co-transmission
(green and purple) are stored in the same set of synaptic vesicles and released together; Serotonin and glutamate → sleep wake cycle	the transmitters are stored in different populations of synaptic vesicles with differential release mediated by different calcium ion (Ca2+) sensitivities. It can also rely on the spatial segregation of vesicle populations to different boutons

Some neurons have more than one NT ,so when the different NTs are in the same vesicle and they are released in the same time then it is called co-release ,and if the NTs are in different vesicles and they get released in a different APs then it is called co-transmission. Some different NTs get released in different AP powers

Mechanism Of Synaptic Transmission

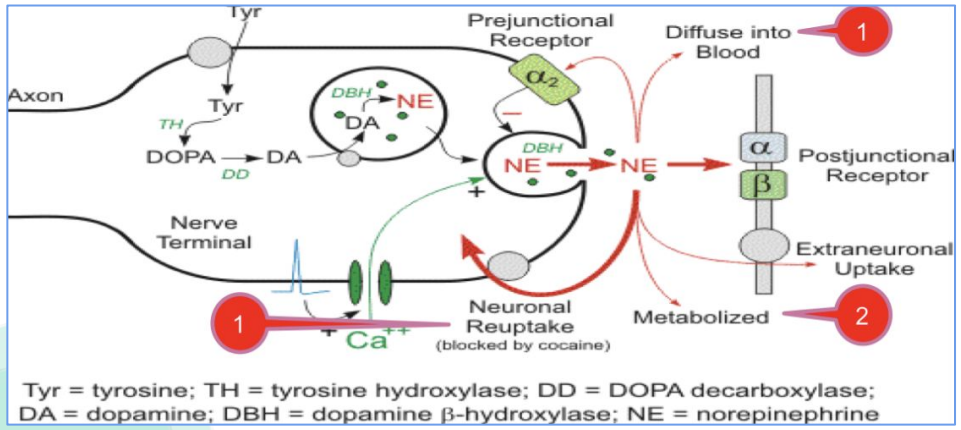
1. Action potential leading to opening of voltage-gated calcium channels.
2. Ca2+ enters bouton down concentration gradient.
3. Ca2+ activates calmodulin, which activates protein kinase. Protein kinase aid in the fusion of synaptic vesicles.
4. Inward diffusion triggers rapid fusion of synaptic vesicles with the presynaptic terminal membrane and release of NTs.
5. Neurotransmitter release at docking site and diffuse across synaptic cleft.
6. Binding to postsynaptic receptors (inhibition or excitation) of the postsynaptic membrane (Depending on the type of the neurotransmitter, i.e. excitatory or inhibitory).
7. Information is transmitted in the central nervous system mainly in the form of nerve action potentials, called Nerve impulses, through a succession of neurons, one after another.
   - NT release is rapid because many vesicles form fusion-complexes at "docking site."

Fate of Neurotransmitter

After a transmitter substance is released at a synapse, it must be removed by:

1. Diffusion out of synaptic cleft into surrounding fluid.
2. Enzymatic destruction e.g Ach esterase for Ach.
3. Active transport back into presynaptic terminal itself e.g norepinephrine. (Reuptake, involves: Na + -dependent membrane transporter).

There are two proteins, one is called syntaxin and the other is called synaptobrevin Syntaxin lines the synaptic vesicles while synaptobrevin lines the docking site, when synapsin is activated it links the two proteins together and only then the vesicles are moved to the docking site and released by exocytosis.



Transmitter act on postsynaptic neuron via "receptor protein"

Postsynaptic receptors have two components :

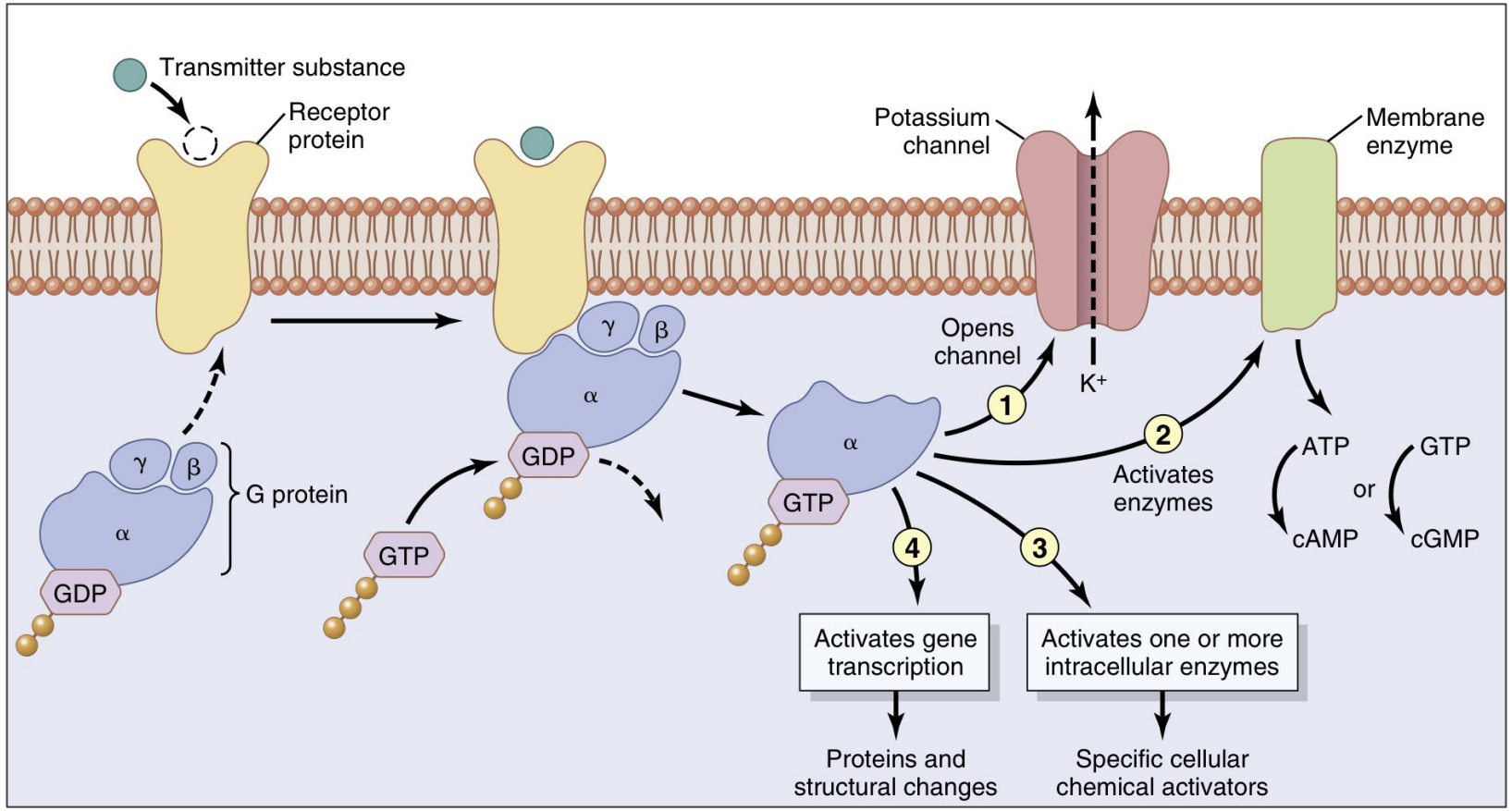
1- **Binding site** that face the cleft to bind the neurotransmitter
2- **Ionophore** (Intracellular component): It passes all the way through the membrane to the interior

- Receptor activation acts in one of two ways (It Is of two types) :

(1) By opening and gating ion channels directly in the postsynaptic neurons and allowing passage of specified types of ions through the membrane. (Ionotropic receptors) - cation channels (Na- most common-,K,Ca) : Opening of Na+ channels → increase membrane potential in positive direction toward threshold level of excitation → (+) neuron. - anion channels (Cl- Mainly-) Opening of Cl^- channels → diffusion of negative charges into the membrane → ↓membrane potential making it more negative → away from threshold level → (-) neuron.	(2) By activating a "second messenger" that is not an ion channel but a molecule that protrudes into the cell cytoplasm and activates one or more substances inside the postsynaptic neuron (Metabotropic receptors) This mechanism is important where prolonged post- synaptic changes are needed to stay for days, months ,years (memory). Effects: intracellular enzymes activation, gene transcription, etc…

Second messenger system in the postsynaptic neuron

It acts in 4 ways:



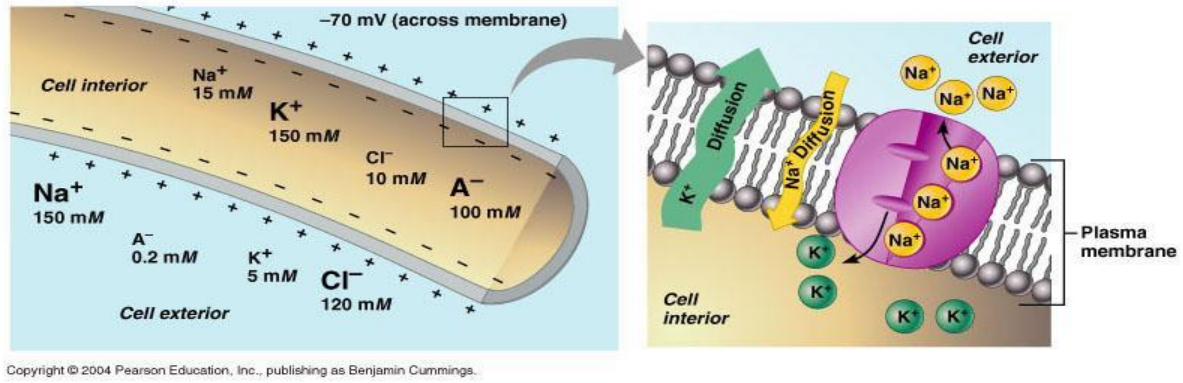
From Guyton:

The second messenger system whereby a transmitter substance from an initial neuron can activate a second neuron by first caus- ing a transformational change in the receptor that releases the activated alpha (α) subunit of the G protein into the second neuron's cytoplasm. Four subsequent possible effects of the G protein are shown, including the following:
I, opening an ion channel in the membrane of the second neuron;
2, activating an enzyme system in the neuron's membrane;
3, activating an intracellular enzyme system; and/or 4, causing gene tran- scription in the second neuron. Return of the G protein to the inactive state occurs when guanosine triphosphate (GTP) bound to the α subunit is hydrolyzed to guanosine diphosphate (GDP), and the β and γ subunits are reattached to the α subunit.

Electrical events in post-synaptic neurons

1. RMP of neuronal soma:

≃ -65mV i.e. less than sk. ms. [-70 to -90mV]. If the voltage is less negative → the neuron is excitable



2. Excitatory post-synaptic potential [EPSPs]

When excitatory neurotransmitter binds to its receptor on post-synaptic membrane → partial depolarization [↑ Na influx] of post-synaptic cell membrane immediately under presynaptic ending, i.e. EPSPs
If this potential rises enough to threshold level → AP will develop and excite the neuron.
This summation will cause the membrane potential to increase from -65mV to -45mV.
∴ EPSPs = +20mV which makes the membrane reach the firing level → AP develops at axon hillock.

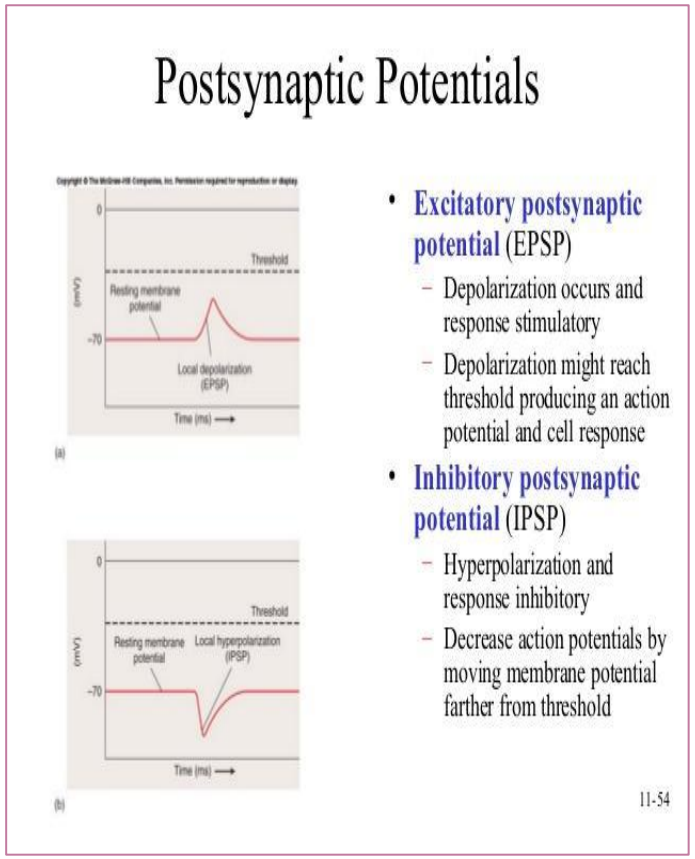
3. Inhibitory post-synaptic potentials (IPSPs):

When an inhibitory NT binds to its receptor on post-synaptic membrane, it causes hyperpolarization of the post-synaptic membrane.
Increasing membrane permeability to Cl^- of post-synaptic memb. (produced by inhibitory neurotransmitter) → ↓ excitability and m. potential (more negative)

Electrical events in post-synaptic neurons (EPSP & IPSP at Chemical Synapses)

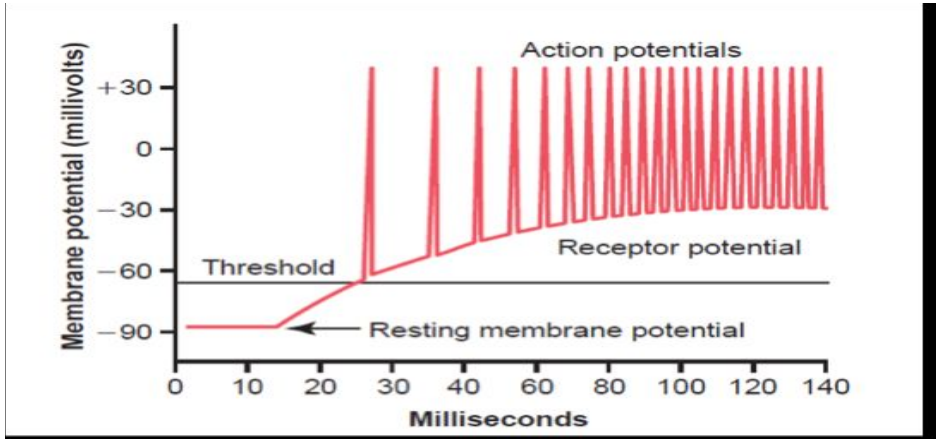
Male slides

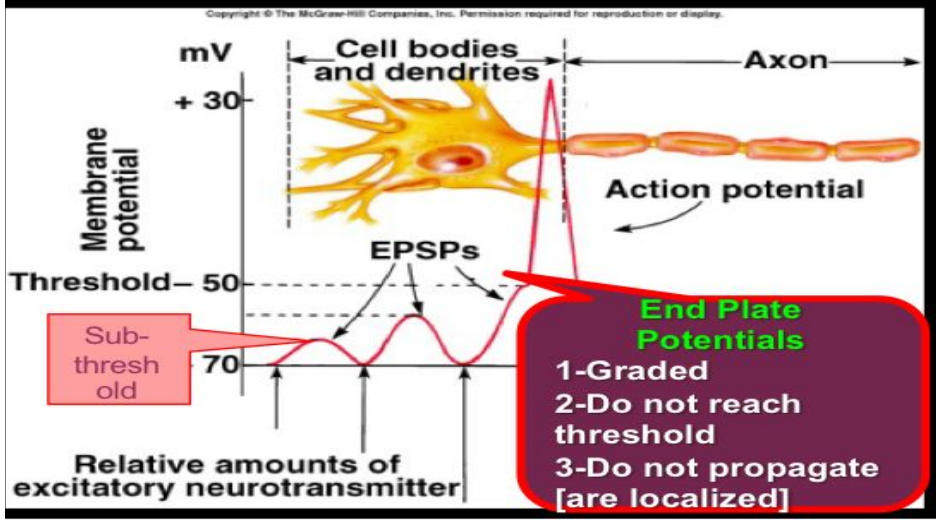
EPSP (excitatory postsynaptic potential)	IPSP (inhibitory postsynaptic potential)
I. Opening of Na channels (Positive charges) to threshold level (most common).	I. Opening of Cl (negative charges) ion channels through the postsynaptic neuronal membrane.
2. ↓ conductance of Cl or K channels, or both.	2. ↑ conductance of K ions out of the neuron.
3.Changes in the internal metabolism of the postsynaptic neuron to excite or to ↑ excitatory membrane receptors or ↓ inhibitory membrane receptors.	3. Activation of receptor enzymes that inhibit cellular metabolic functions that ↑ inhibitory membrane receptors or ↓ excitatory membrane receptors.



Generator potential (Receptor potential)

- Depolarization of receptor membrane leads to local circuit which initiates action potential at the first node of Ranvier on the nerve attached to the receptor.
- When the receptor potential rises above the threshold for eliciting action potentials in the nerve fiber attached to the receptor, then action potentials occur.
- Non propagated depolarizing potential.
-It is produced in un-myelinated nerve terminal.
- The more the receptor potential rises above the threshold level, the greater becomes the action potential frequency.





: Male doctor

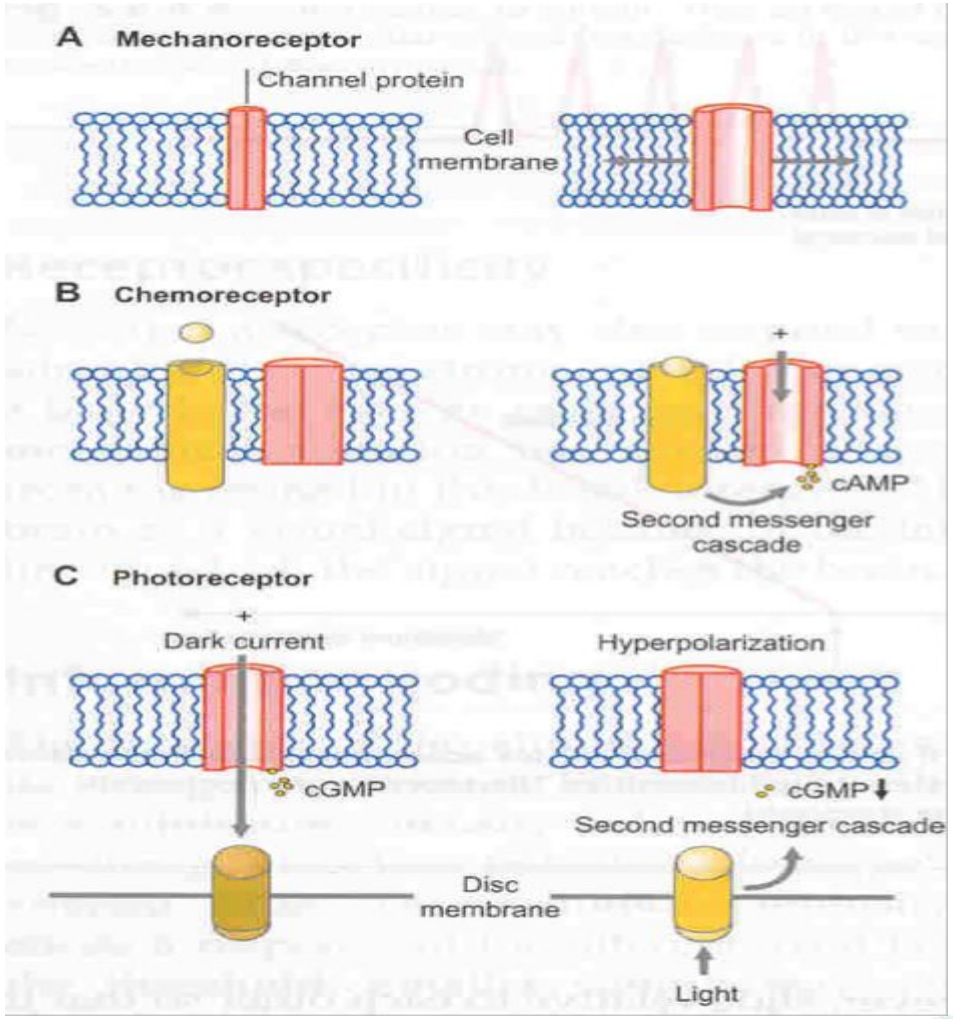
وش اللي يحدد اذا الـ **NT** كميته كثيره او قليله؟ حسب قوة الـ **AP** اختلاف القوة هو اللي يخلي الاوامر اللي من **CNS** في مستويات مختلفة.

Just to remind you, Action potentials cannot be graded (ALL or none role) so doctor probably meant (**frequency of Action potentials** NOT strength)

Mechanisms of receptor potentials:

Transmembrane potential: the basic cause of the change in membrane potential is a change in membrane permeability of the receptor, which allows ions to diffuse more or less readily through the membrane:

(I) by mechanical deformation of the receptor, which stretches the receptor membrane and opens ion channels.
(2) by application of a chemical (neurotransmitter) to the membrane, which also opens ion channels.
(3) by change of the temperature of the membrane, which alters the permeability of the membrane.
(4) by the effects of electromagnetic radiation, such as light on a **retinal** visual Receptor.

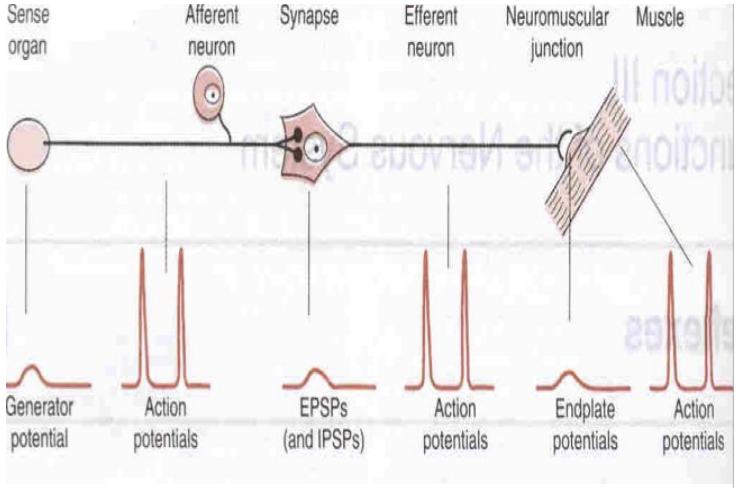


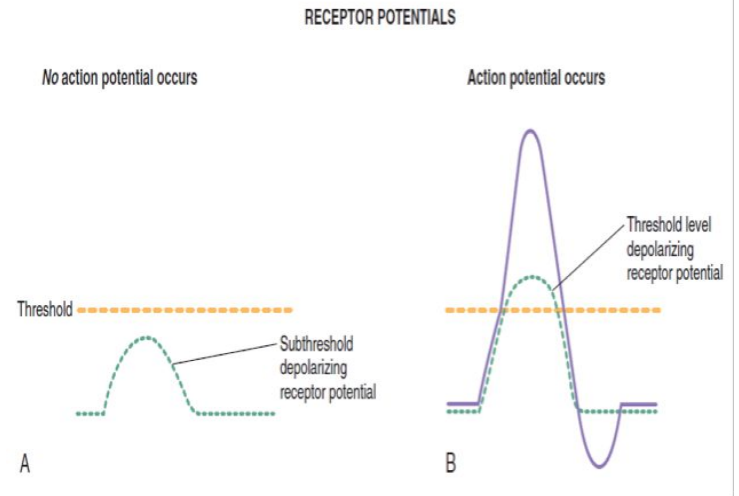
Differences between generator potential and action potential:

Receptor (Generator potential)	Action potential
In the Receptor	In the Sensory and motor nerve fiber
Graded	Not graded
Doesn't obey all or none rule	Obeys all or none rule
Can be summated	Not summated
Unpropagated	Propagated

- The frequency of action potential is proportionate to the intensity (magnitude) of the applied stimuli.

- In other way: the more the receptor potential rises above threshold level, the greater becomes the action potential frequency.



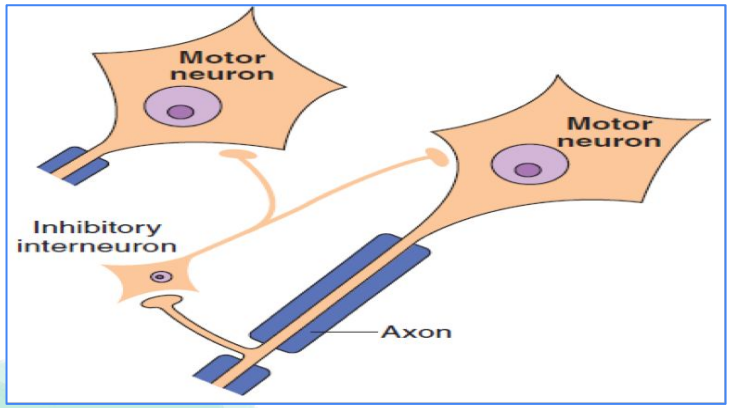


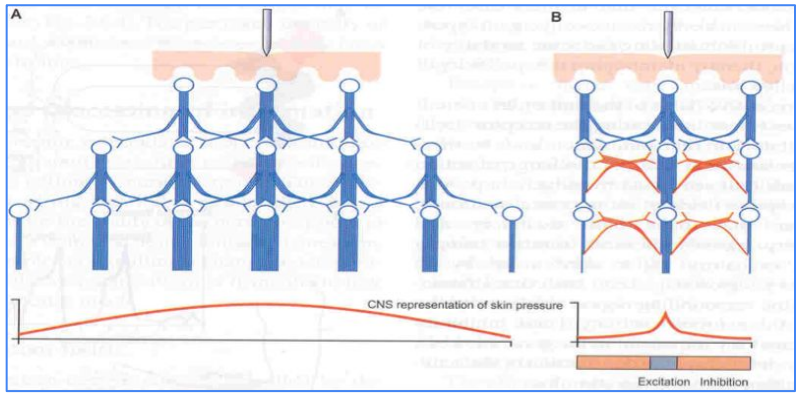
How EPSPs differs from Action potential? (Female slides only)

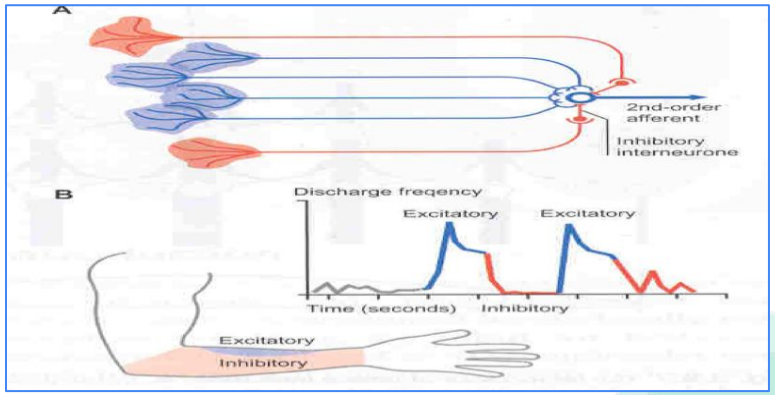
- Proportionate to the strength of the stimulus
- Can be summated
- If large enough to reach firing level→ AP is produced

Inhibitory interneuron (Lateral inhibition)

- Each stimulus is defined by a central zone of **excited** cells surrounded by a **field of inhibition.**
- Is important to localize the stimulus, a phenomenon called: two-point discrimination.
- Lateral inhibition depends on receptive field size. ex: receptive field for touch on fingertips is about 2mm compared with 40mm for the arm.
- In the absence of lateral inhibition, convergence and divergence at various levels blur the excitation distribution.
- Aside from the central excitatory signal, short lateral pathways transmit inhibitory signals to the surrounding neurons.
- These signals pass through additional interneurons that secrete an inhibitory transmitter (**Glycine**).







Processing of information (Transmission of signals)

- There is always a minimum intensity needed to elicit a response in the afferent axons (**threshold**).
- Small stimuli might provoke changes in the membrane potential of the receptor but will not elicit action potential in the nerve.
- Increase the strength of the stimuli by:

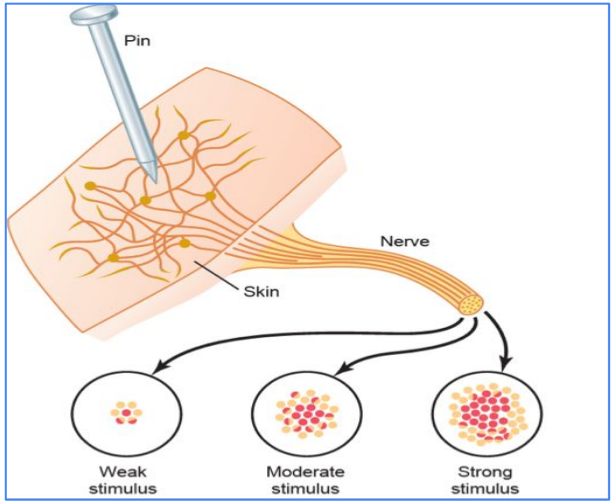
1) Spatial summation: several stimulus at the same time (using progressively greater numbers of fibers).
2) Temporal summation: increasing the frequency (increasing the frequency of nerve impulses in each fiber.

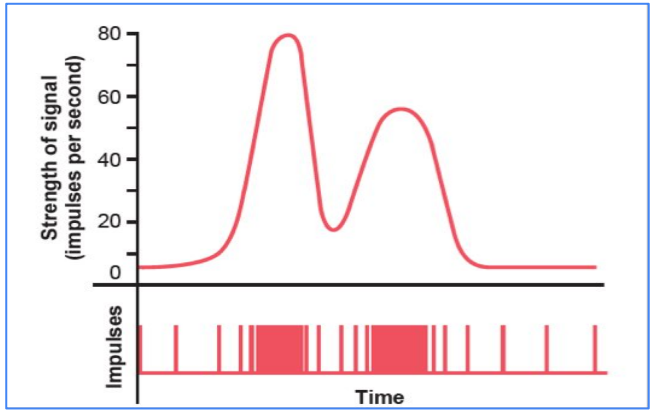
- Location of the stimulus is signaled by the activation of group of receptor.
- **Receptive field** (area of stimulation) **varies in size.**

e.g: fingertips have about 2300 receptors/cm2.

- **Importance of receptive field size:**

is a major factor to determine the ability to localize stimuli. And used clinically in 2 points discrimination Test.





Synaptic properties

For chemical Synapse

1. One-way conduction
2. Synaptic delay
3. Synaptic inhibition
4. Summation
5. Convergence and divergence
6. Fatigue

Synaptic properties For chemical Synapse

1 One-way conduction

Synapses generally permit conduction of impulses in one-way i.e. from pre-synaptic to post-synaptic neuron.

2 Synaptic delay (central delay)

Is the minimum time required for transmission across the synapse.
It is 0.5ms to 4ms for transmission across one synapse.
This time is taken by

- Discharge of transmitter substance by pre-synaptic terminal
- Diffusion of transmitter to post-synaptic membrane
- Action of transmitter on its receptor
- Action of transmitter to ↑ membrane permeability
- Increased diffusion of Na+ to ↑ post-synaptic potential

-Clinical importance: know number of synapses involved in neuronal pathways by time lag.

Synaptic properties For chemical Synapse

3 Synaptic inhibition:

Types

Direct inhibition (Postsynaptic inhibition)

Occurs when an inhibitory neuron (releasing inhibitory substance/ transmitter) (e.g.,glycine, GABA) from a presynaptic nerve terminal acts on a postsynaptic neuron leading to hyperpolarization due to opening of Cl^- [IPSPs] and/or K+ channels.

Example :

- Glycine at the level of the spinal cord to block pain impulses.
- Baclofen (GABA agonist)

Indirect inhibition (Presynaptic inhibition)

This happens when an inhibitory synaptic knob (Axoaxonic synapse) lie directly on the termination of a presynaptic excitatory fiber.
The inhibitory synaptic knob release a transmitter which inhibits the release of excitatory transmitter from the presynaptic fiber.
e. g. GABA (Pain modification)

Reciprocal inhibition

Inhibition of antagonist activity is initiated in the agonist muscle.

Impulses pass directly to the motor neurons supplying the same muscle and via branches to inhibitory interneurons that end on motor neurones of antagonist muscle.

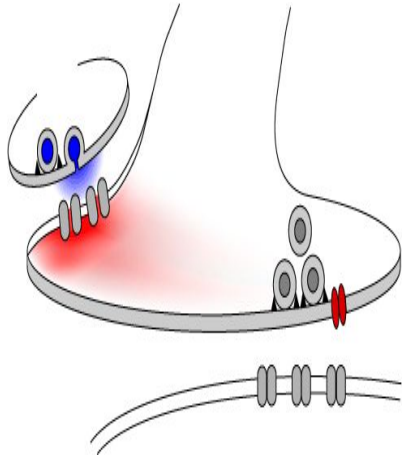
Inhibitory interneuron (Renshaw cells)

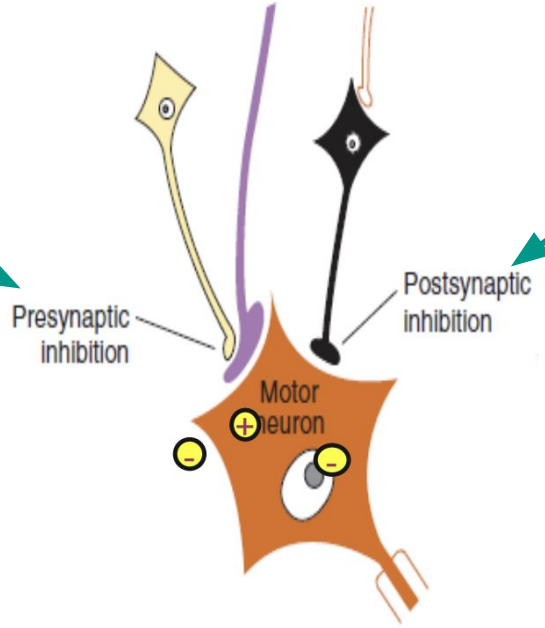
Negative feedback inhibitory interneuron of a spinal motor neuron
((Control the strength of contraction))

Synaptic properties For chemical Synapse

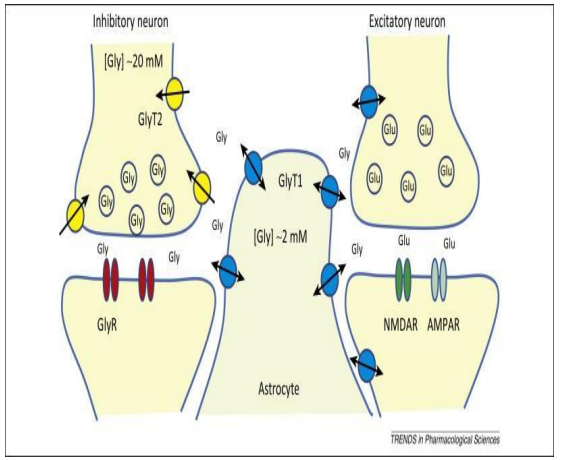
3 Synaptic inhibition:

Indirect inhibition (Presynaptic inhibition)



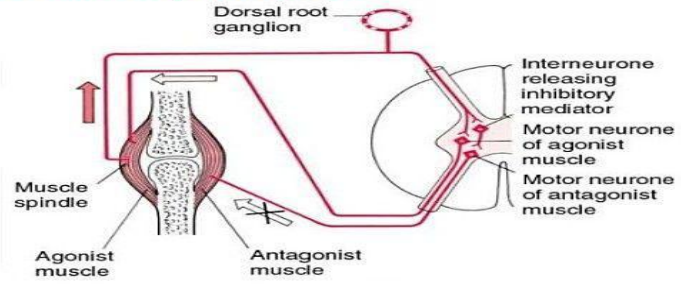


Direct inhibition (Postsynaptic inhibition)

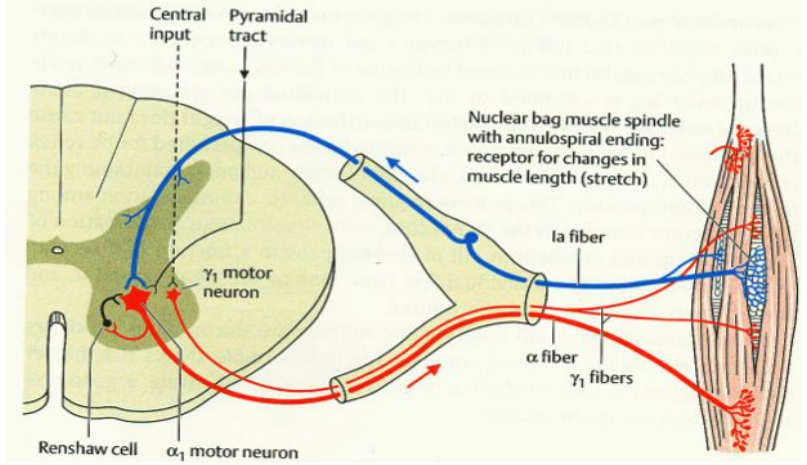


Reciprocal inhibition

When one muscle is contracted, its antagonist is automatically inhibited.



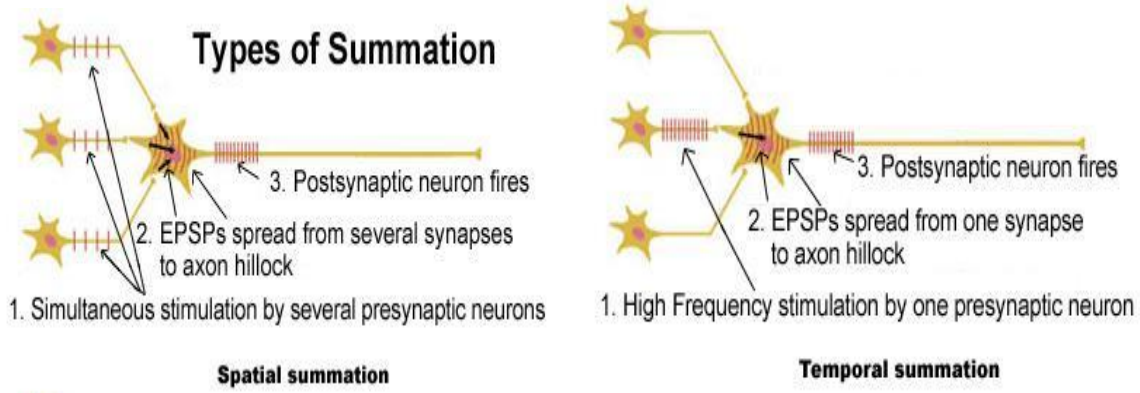
Inhibitory interneuron (Renshaw cells)



Synaptic properties For chemical Synapse

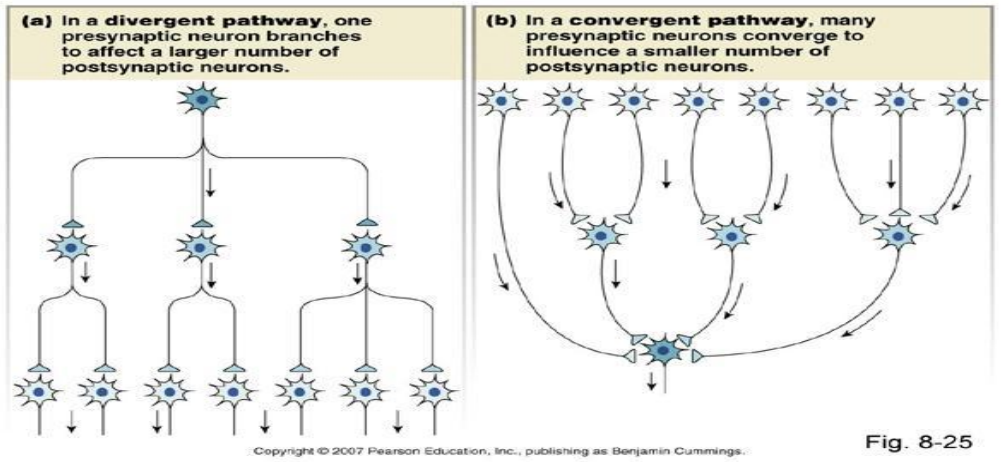
4 Summation

Spatial summation	Temporal summation
Eliciting an action potential in a neuron with input from multiple presynaptic cells.	When the frequency of stimulation increased from the same presynaptic fiver



5 Convergence and divergence

Convergence	Divergence
When many pre-synaptic neurons converge on any single post-synaptic neuron.	Axons of pre-synaptic neurons divide into many branches that diverge to end on many post-synaptic neurons.



Synaptic properties For chemical Synapse

6 Synaptic Fatigue

Synaptic fatigue means simply that synaptic transmission becomes progressively weaker the more prolonged and more intense the period of excitation. It is due to exhaustion of neurotransmitter.

- If the presynaptic neurons are continuously stimulated there may be an exhaustion of the neurotransmitter. Resulting in stoppage of synaptic transmission.
- Activity-dependent form of short term synaptic plasticity that results in the temporary inability of neurons to fire and therefore transmit an input signal (synaptic depression).
- The cause of this sudden cessation of reverberation is fatigue of synaptic junctions in the circuit.

Fatigue beyond a certain critical level lowers the stimulation of the next neuron.

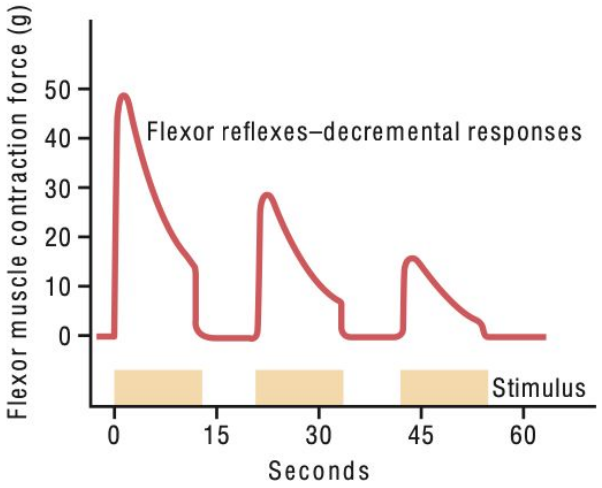


Figure 47-18. Successive flexor reflexes showing fatigue of conduction through the reflex pathway.

Mechanism of Synaptic fatigue:

- Exhaustion of the stores of transmitter.
- Inactivation of many of the postsynaptic membrane receptors.
- Abnormal ion concentrations in postsynaptic neuron.

Fatigue is a protective mechanism against excess neuronal activity. Epilepsy!!

Male slides

Patterns of synaptic transmission in neuronal pools

- DIVERGENCE
- CONVERGENCE
- RECIPROCAL INHIBITION
- AFTER DISCHARGE
- IRRADIATION
- REVERBERATORY CIRCUIT
- LOCAL SIGN

Patterns of synaptic transmission in neuronal pools

DIVERGENCE of signals

Divergence: is important for weak signals entering a neuronal pool to excite greater numbers of nerve fibers leaving the pool:

1. **Amplifying divergence: an input signal spreads to an increasing number of neurons as it passes through successive orders of neurons in its path. Corticospinal pathway in its control of skeletal muscles, with a single large pyramidal cell in the motor cortex capable, under highly facilitated conditions, of exciting as many as 10,000 muscle fibers.**
2. **Divergence into multiple tracts. The signal is transmitted in two directions from the pool. (Dorsal column tract).**

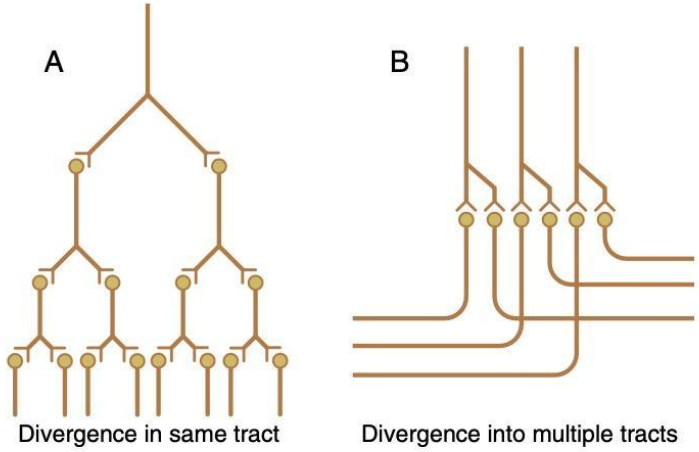


Figure 47-11 "Divergence" in neuronal pathways. **A,** Divergence within a pathway to cause "amplification" of the signal. **B,** Divergence into multiple tracts to transmit the signal to separate areas.

CONVERGENCE of signals

- **Convergence means signals from multiple inputs uniting to excite a single neuron.**
- **To make the stimulus more concentrated**

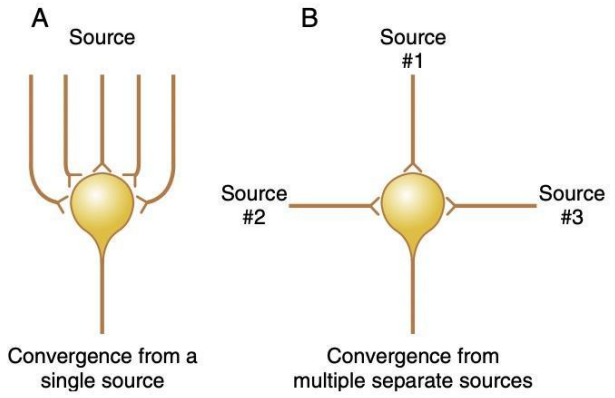


Figure 47-12 Convergence of multiple input fibers onto a single neuron. **A,** Multiple input fibers from a single source. **B,** Input fibers from multiple separate sources.

Patterns of synaptic transmission in neuronal pools

RECIPROCAL INHIBITION

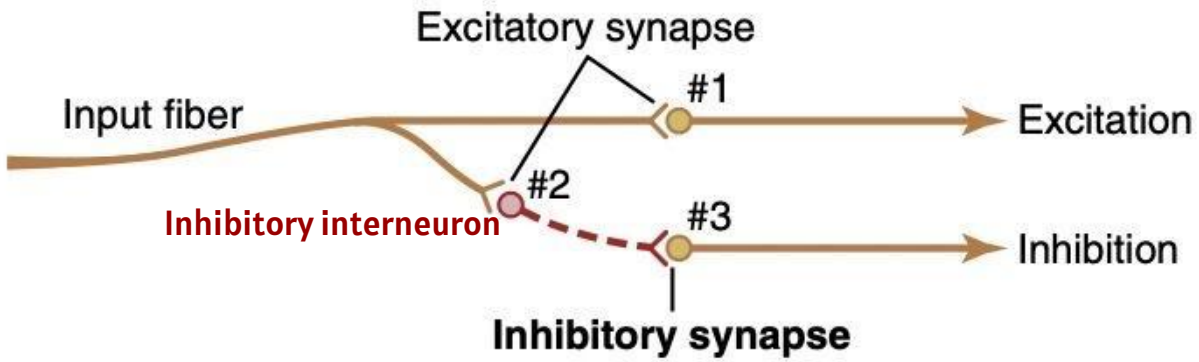


Figure 47-13 Inhibitory circuit. Neuron 2 is an inhibitory neuron.

Neuronal circuit with both excitatory and inhibitory output signals.
E.g.: controlling agonist & antagonistic pairs of muscles, in reciprocal inhibition circuit

Reciprocal inhibition: Inhibition of antagonist muscle when agonist is excited.

Negative feedback inhibition of a spinal motor neuron via an inhibitory interneuron. The axon of a spinal motor neuron has a recurrent collateral that synapses on an inhibitory interneuron that terminates on the cell body of the same and other motor neurons. The inhibitory interneuron is called a Renshaw cell and its neurotransmitter is glycine.

Male slides

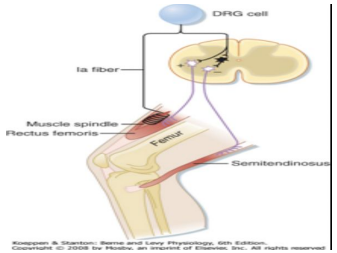
Patterns of synaptic transmission in neuronal pools

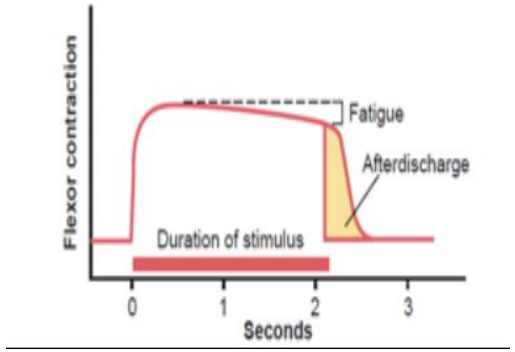
AFTER DISCHARGE

A prolonged maintained output discharge of Ach called after -discharge, lasting a few milliseconds or many minutes after the incoming signal is over. Due to:

I- EPSP can continue to excite the neuron to transmit (a series of continuous repetitive discharges) .

2- Reverberating circuits





REVERBERATORY CIRCUIT

Positive feedback within the neuronal circuit that feeds back to reexcite the input of the same circuit .The circuit may discharge repetitively for a long time.

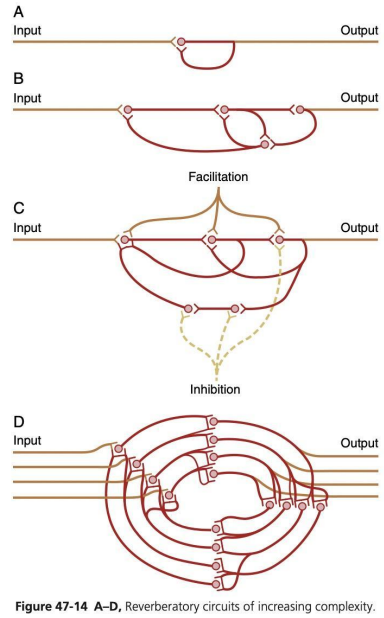


Figure 47-14 A–D, Reverberatory circuits of increasing complexity.

IRRADIATION

Spread of impulses up & down to different segments and motor neurons in the spinal cord. e.g. A strong stim. in sensory afferent irradiate to many segments of S.C due to divergence.

LOCAL SIGN

The response to the stimulus by the body will be determined by the location of the stimulus on the body. Noxious stimulus to the hind limb does not result in withdrawal of a forelimb

Factors affecting synaptic transmission

Change in internal environment:

1. Alkalosis (Increases neuronal excitability)

Causes cerebral epileptic seizures (Increased excitability cerebral neurons)
e. g. overbreathing in person with epilepsy, The over breathing blows off carbon dioxide and therefore elevates the pH of the blood momentarily/ Hyperventilation & epilepsy.

2. Acidosis (Depresses neuronal activity)

pH around 7.0 usually causes a coma (e. g. severe diabetic or uremic acidosis)/ Diabetes mellitus & coma.

3. Hypocalcemia: ↑ Neuronal excitability (Tetany).
4. Hypoglycemia: ↓ Neuronal activity.
5. Hypoxia: Depression of neurons

Diseases:

1. Tetanus: Inhibits release of GABA (Spastic) (opposite to Alcohol effect).
2. Botulism: Inhibits release of Ach (Flaccid).

Drugs:

1. Caffeine found in coffee, tea, depolarizes postsynaptic membrane, increases neuronal excitability, by reducing the threshold for excitation of neurons.
2. Strychnine: blocks inhibitory transmitters Glycine.
3. Sedatives & anesthetics: hyperpolarize (↑ threshold) postsynaptic membrane.
4. Alcohol: increases the inhibitory effects of the neurotransmitter GABA.
5. Morphine: acts on synapses that use endorphin neurotransmitters.
6. Cocaine: blocks the reuptake of dopamine.

TEST YOURSELF !

1- which of the following best describes Spread of impulses up & down to different segments and motor neurons in the spinal cord?

A) Irradiation
B) Spatial summation
C) Convergence
D) Reverberating

2- which of the following conditions increase synaptic excitation on neurons?

A) Alkalosis
B) Hypoxia
C) Dehydration
D) Acidosis

3- what is it called when an output neuron sends restimulation collaterals to the input nerve?

A) Divergence
B) Convergence
C) Reverberation
D) Reactivation

4- the minimum time required for transmission of signal through the synaptic space is?

A) 0.005ms
B) 0.05ms
C) 0.5ms
D) 0.8ms

Answers: A, A, C, C.

SAQ

Explain the fate of neurotransmitters?

1. Diffusion out of synaptic cleft into surrounding fluid.
2. Enzymatic destruction e.g Ach esterase for Ach.
3. Active transport back into presynaptic terminal itself e.g norepinephrine.

Enumerate the factors affecting synaptic transmission?

Answer in slide number 31.

What is the difference between the chemical synapse and the electrical synapse?

Chemical synapse	Electrical synapse
- Exhibits synaptic delay.	- Almost no delay in transmission.
Slower	Faster
Mostly unidirectional.	Mostly bidirectional.
The response may not be the same as the source.	The response is always the same sign as the source.

check the main slide for more..

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