PROPERTIES OF SYNAPSE
(Essay/Short essay)

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Properties of synapse

1. One way conduction
2. Synaptic delay
3. Convergence
4. Divergence
5. Summation
6. Occlusion
7. Facilitation
8. Subliminal fringe effect
9. Synaptic Fatigue
10. After discharge
11. Synaptic inhibition
12. Synaptic plasticity
    a. Post tetanic potentiation
    b. Habituation
    c. Sensitisation
    d. Long term potentiation
    e. Long term depression

1. One way conduction

- Synapses permit impulse conduction only from pre to post synaptic neuron →because NT is present only in pre synaptic terminal
- Receptors of neurotransmitter is present in postsynaptic neuron
- Axons can conduct in both directions
- Antidromically conducted impulse dies out after depolarizing cell body
- Necessary for orderly conduction of impulses
- Bidirectional in electric synapse

2. Synaptic delay

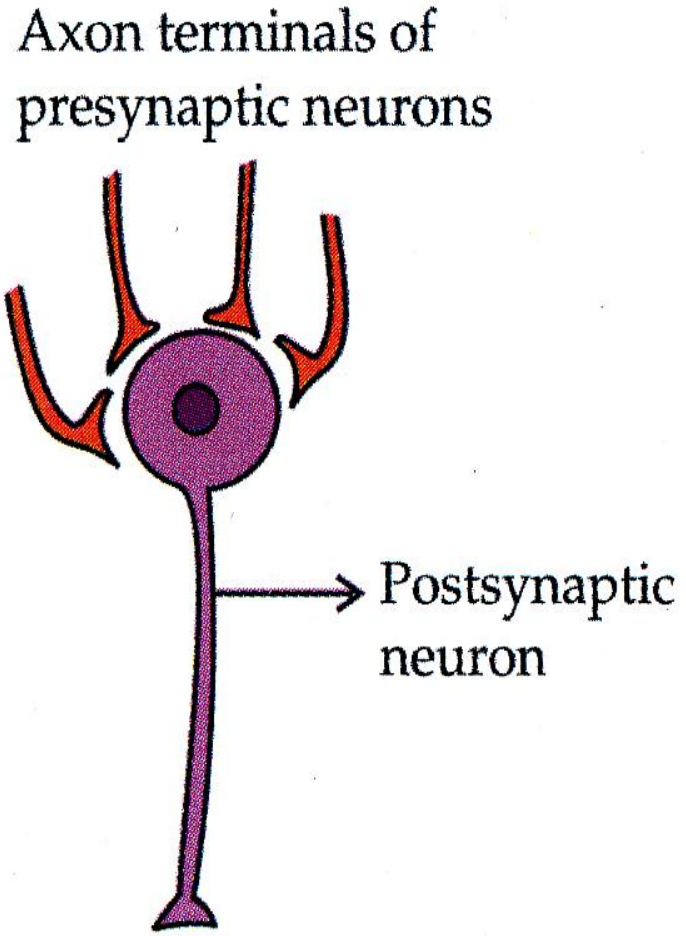
- Time delay between impulse transmission at the synapse
- Delay which occurs between arrival of an impulse at Presynaptic terminal & the occurrence of response in post synaptic neuron
- Usually about 0.5 ms
- Can be used to find the number of synapses in a pathway

Causes of synaptic delay

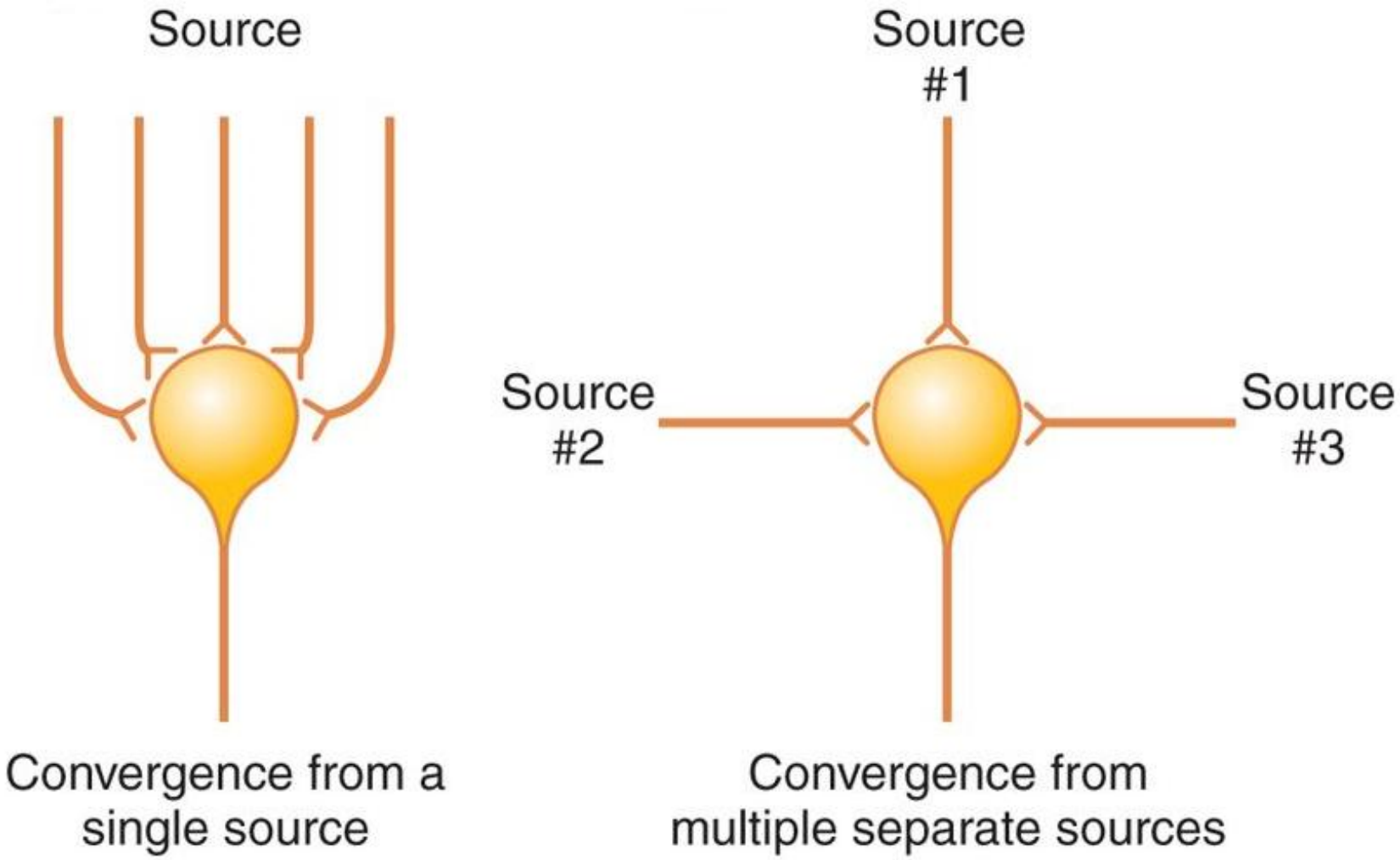
- Time taken for
  - Release of NT
  - Diffusion across synaptic cleft
  - Its action on receptor
  - Diffusion of ions
  - Change in membrane potential

3. Convergence

- Many pre synaptic terminals converge on a single post synaptic neuron

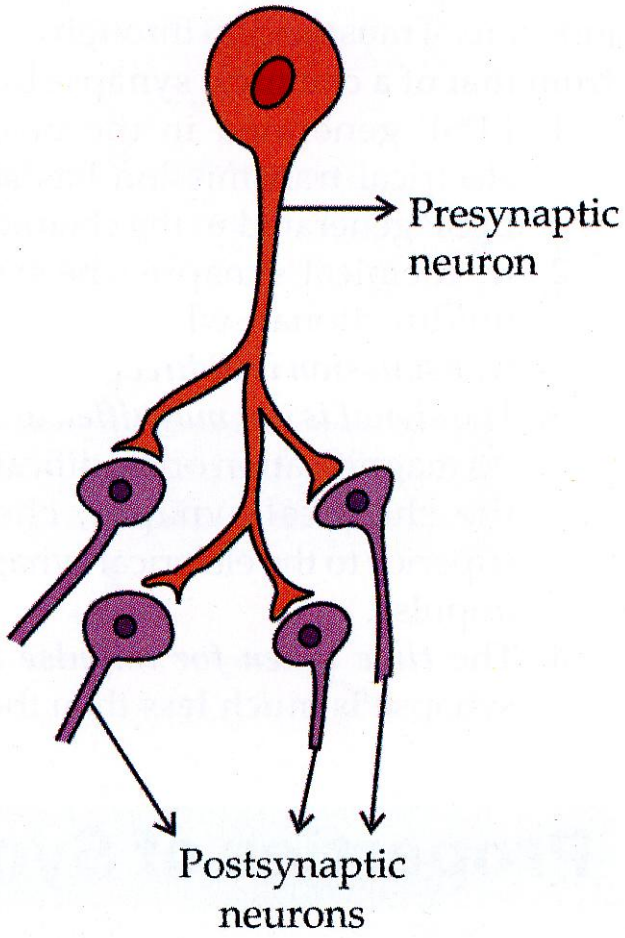


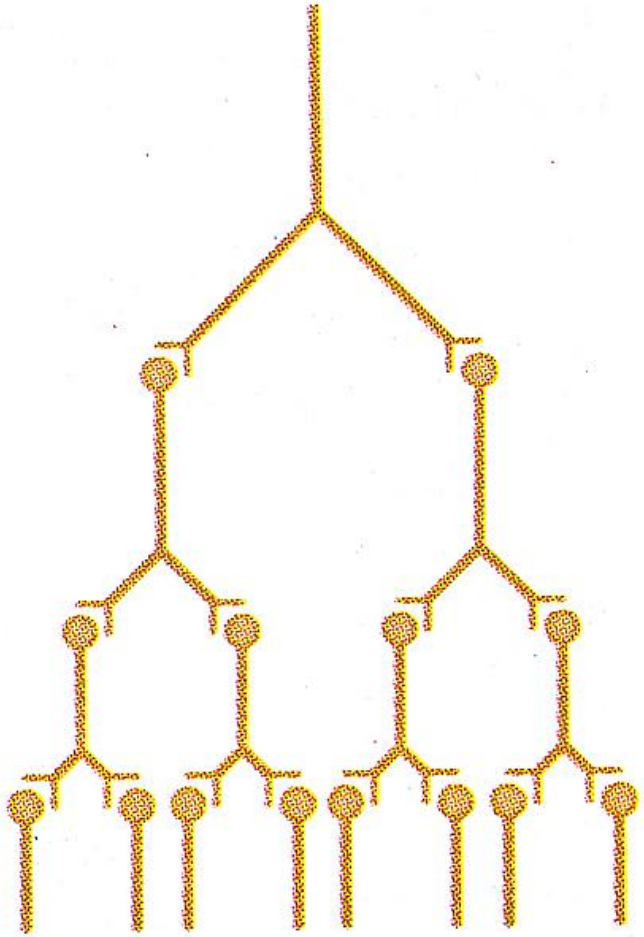
- Input can be from a **single source** (multiple terminals of same neuron) or from **different sources**



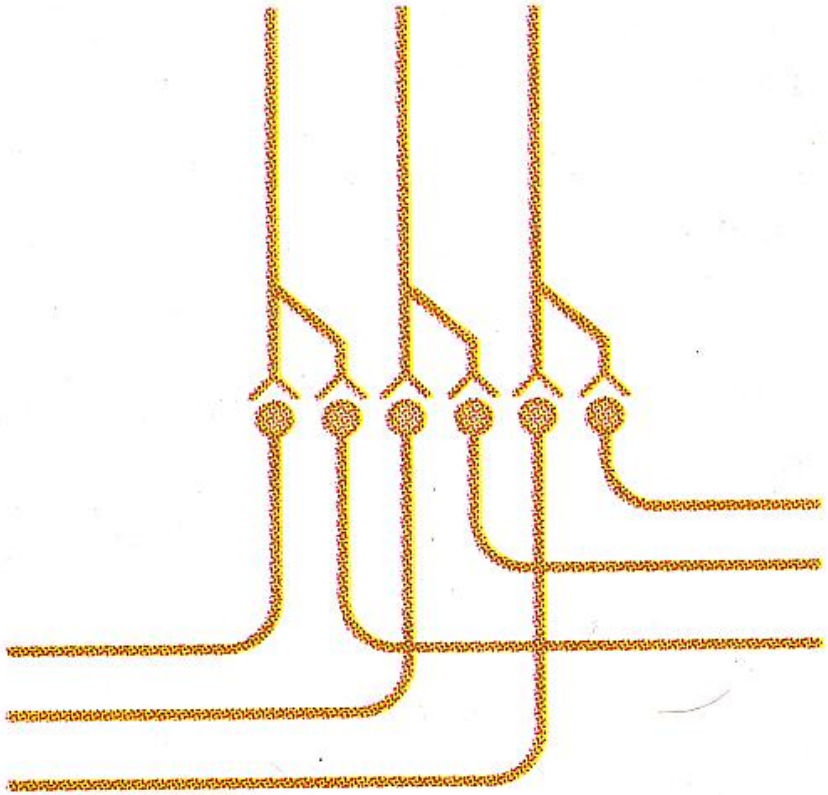
4. Divergence

- A single presynaptic neuron divide into many branches & connect with many postsynaptic neurons





Divergence in same tract

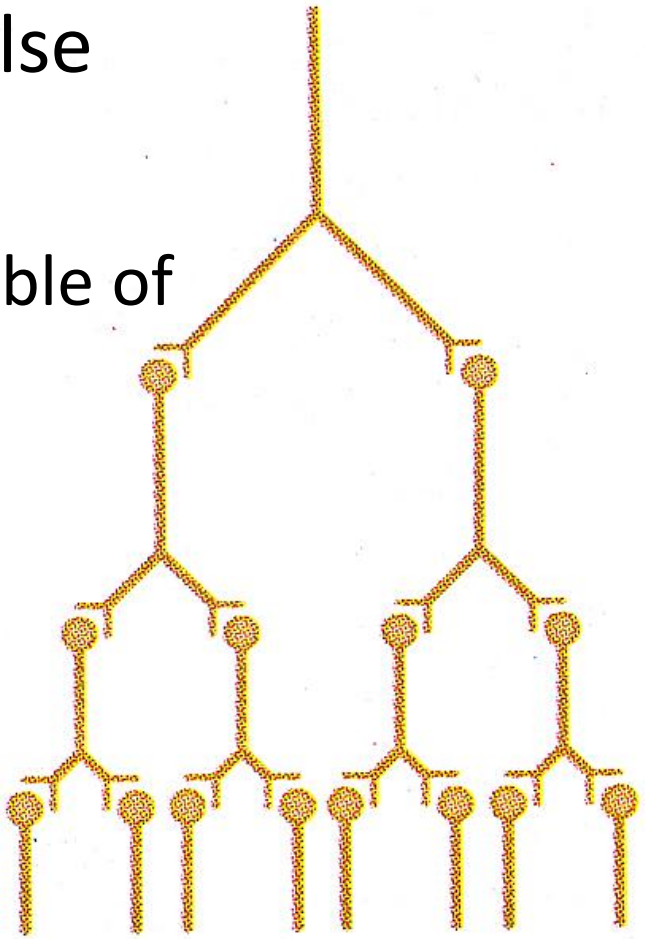


Divergence in multiple tracts

- Postsynaptic neurons may travel in the **same tract or multiple tracts**

Same tract

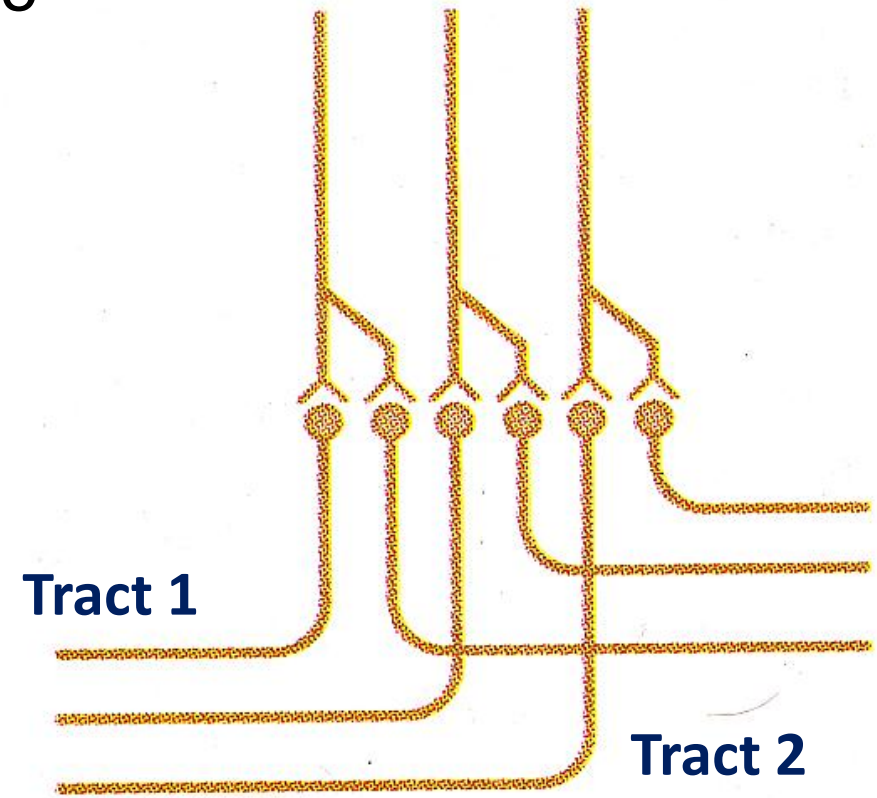
- Helps in amplification of an impulse
- Characteristic of pyramidal tract
  - A single large pyramidal cell is capable of exciting as many as 10,000 muscle fibres



Divergence in same tract

Multiple tracts

- Signal is transmitted in 2 directions from the same pool
- Information transmitted up dorsal column of SC passes into cerebellum and to thalamus & cerebral cortex



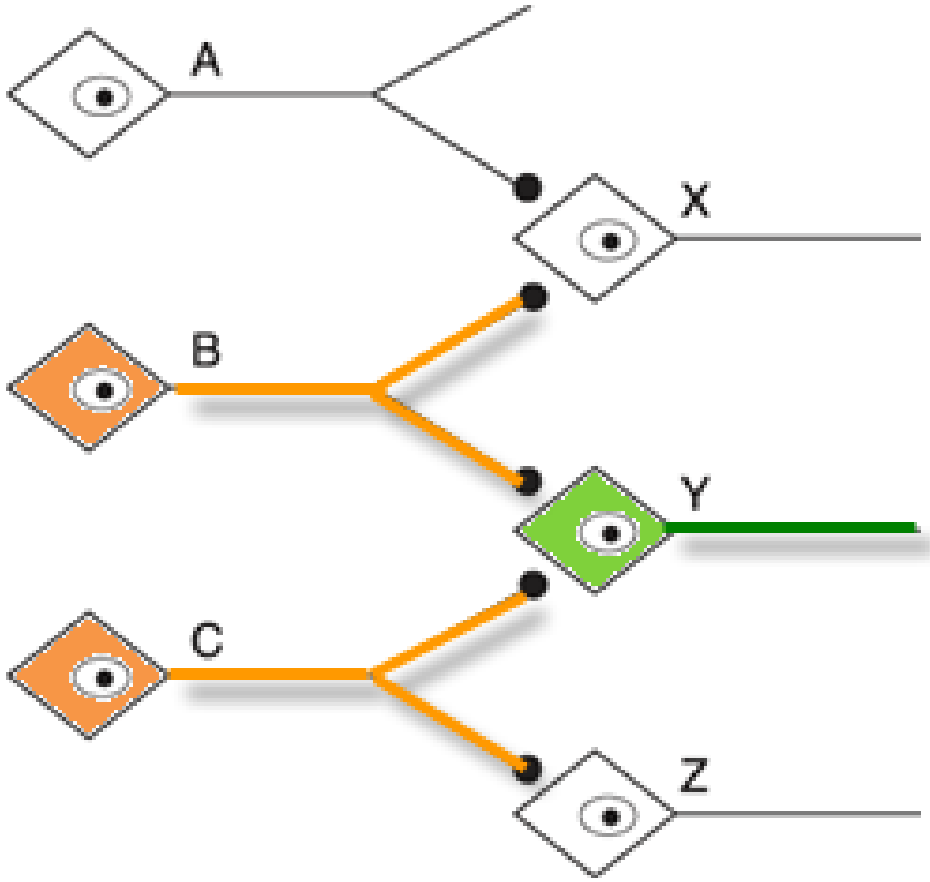
Divergence in multiple tracts

5. Summation

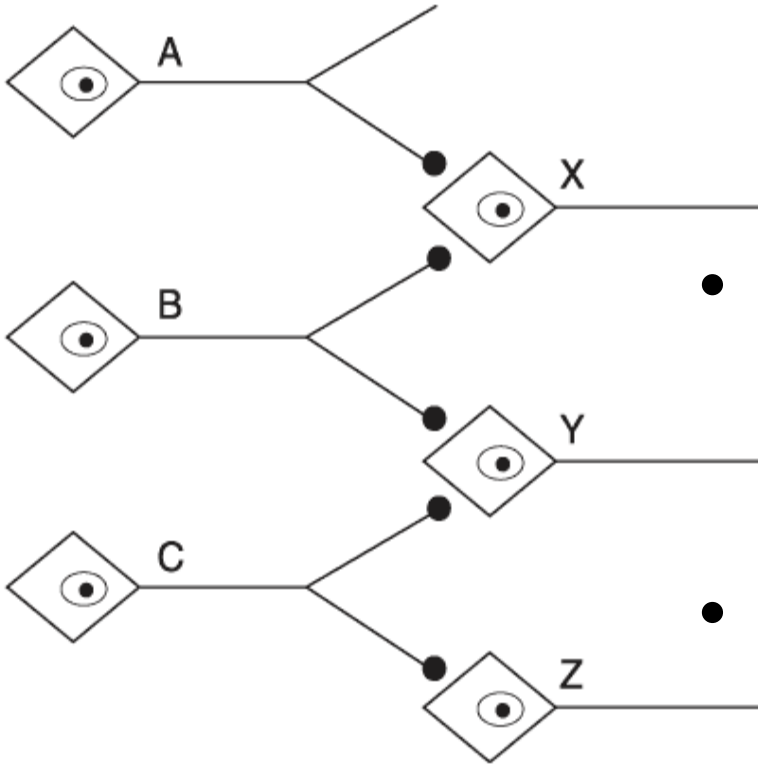
- **Spatial summation**
  - When activity is present in more than one synaptic knob at the same time, **spatial summation** occurs
- **Temporal summation**
  - Repeated successive stimuli at short intervals –before previous EPSPs have decayed →**temporal summation**

6. Occlusion

- Situation in which **response to simultaneous stimulation of 2 presynaptic N is less than the sum total of response obtained when they are stimulated separately**

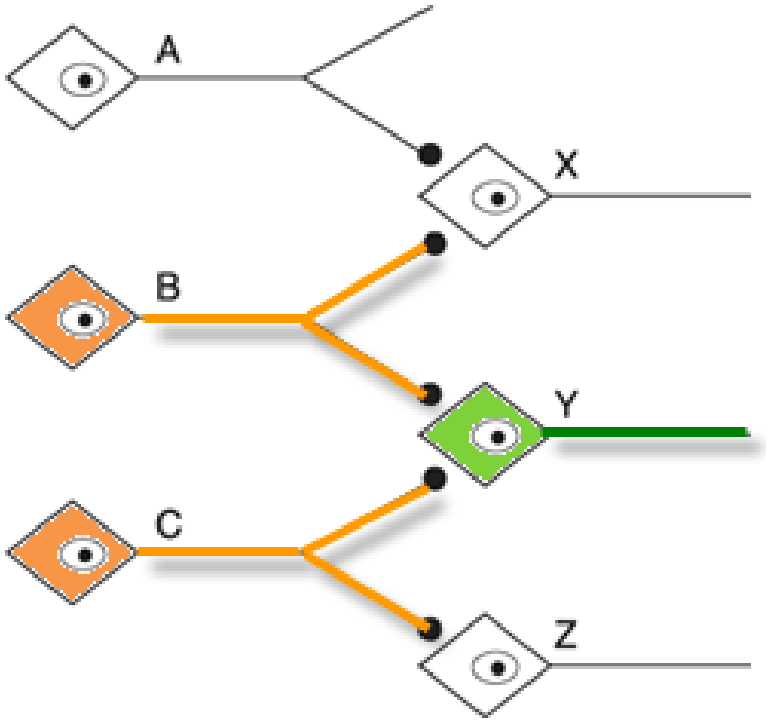


Occlusion



- If B is repeatedly stimulated, due to temporal summation, AP can occur in X & Y (2 N)
- If C is repeatedly stimulated, AP can occur in Y & Z (2 N)
- If B & C are simultaneously stimulated, AP are produced in X, Y & Z (3 N)

- Response to stimulation of B & C together is not as great as the sum of responses to stimulation of B & C separately →because B & C both end on neuron Y

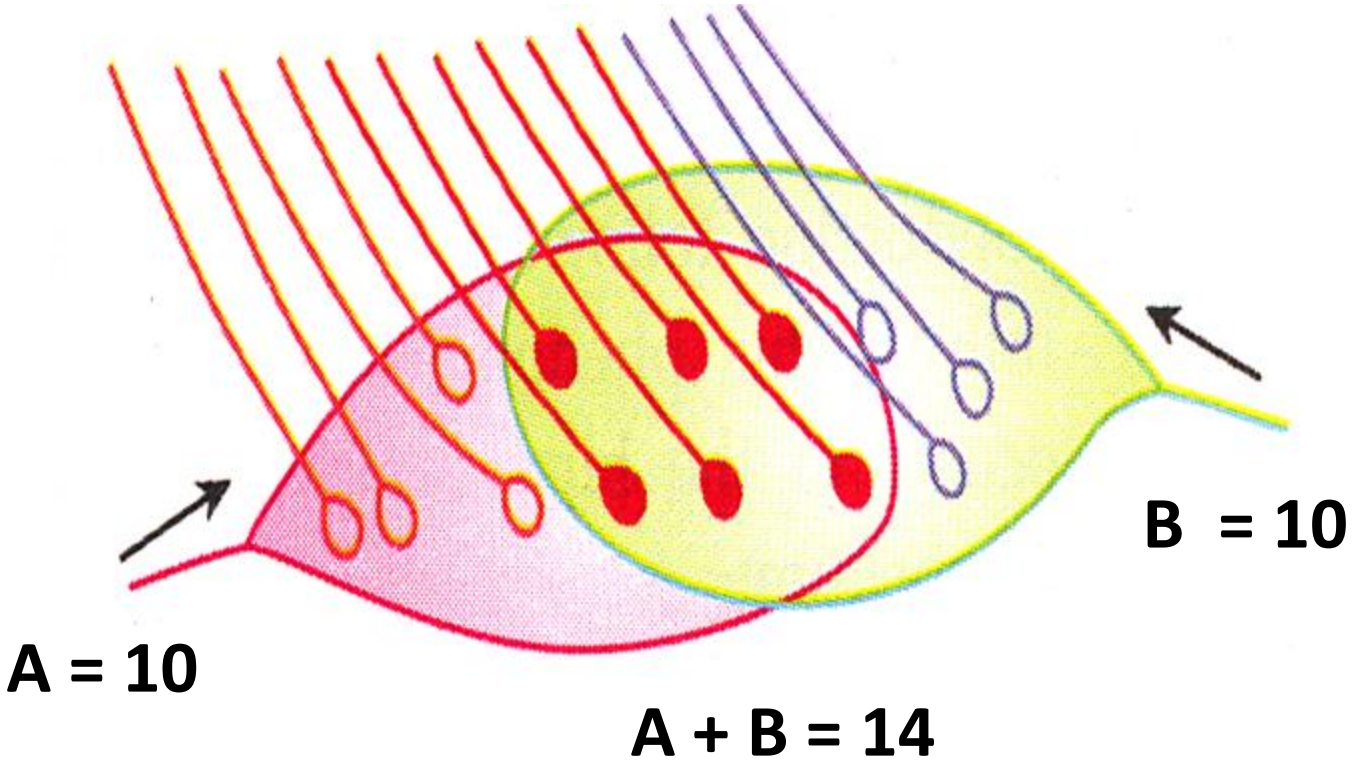


B =2

C =2

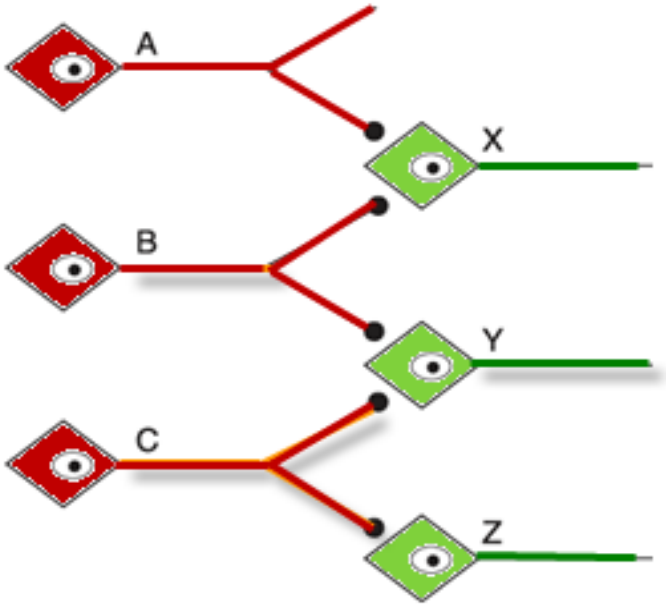
but B + C = 3

- This decrease in expected response, due to pre synaptic fibers sharing post synaptic neurons is called **occlusion**



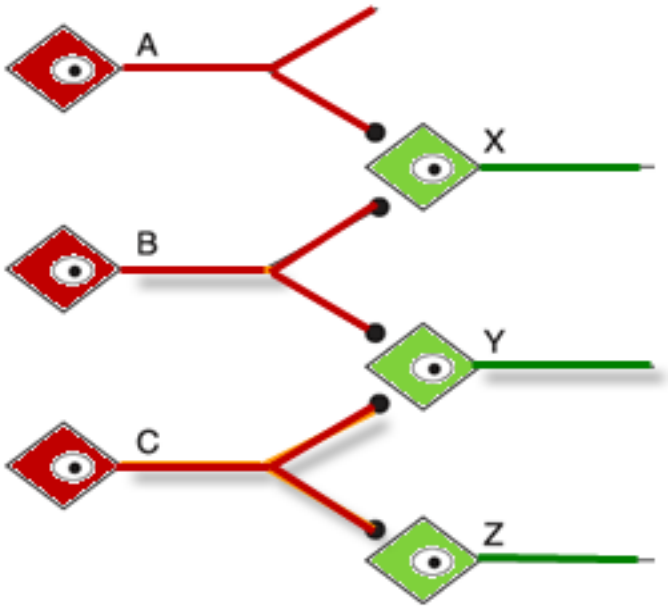
7. Facilitation

- Stimulation of A or B →EPSP in X
- Stimulation of A +B →EPSPs in X will sum & may reach threshold →AP in X
- Effect of depolarisation caused by impulse in A is facilitated by that due to activity in B →**spatial facilitation**



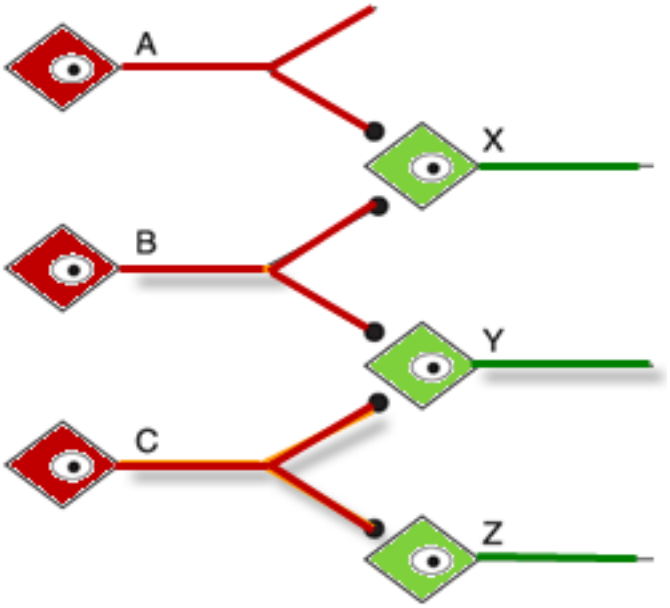
8. Subliminal fringe

- Subliminal- below threshold
- Fringe- border
- Postsynaptic neuron are said to be in subliminal fringe if they are not discharged by activity of presynaptic neuron but their excitability increased



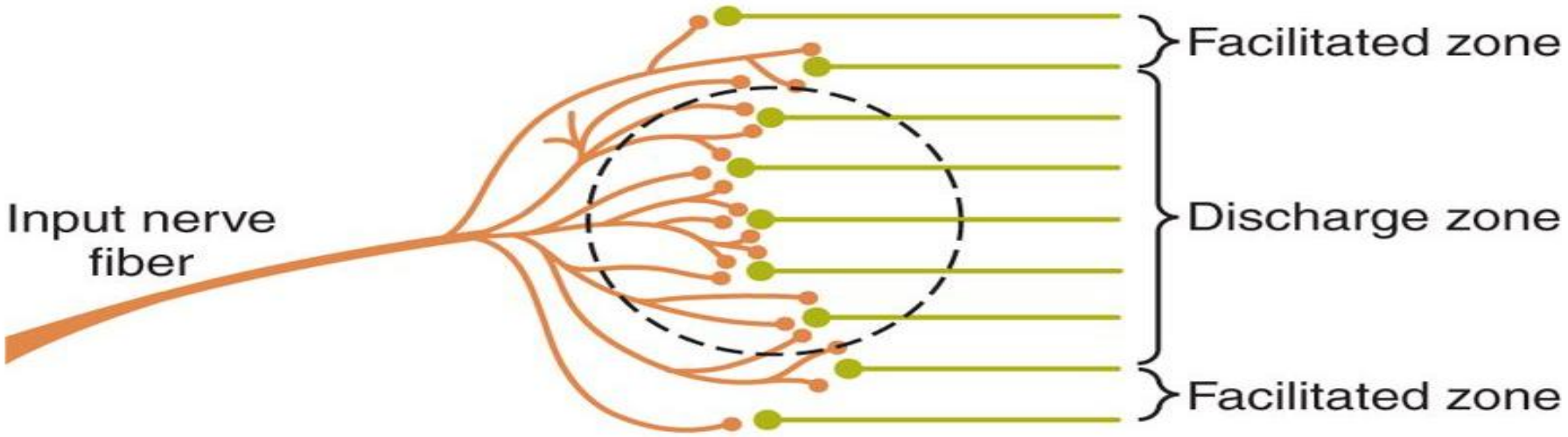
Subliminal fringe

- Activity in A + B → AP in X and ↑ excitability in Y →but Y not fired
- Now it is easier for activity in C to fire Y
- Y is said to be in subliminal fringe of X



Subliminal fringe

- Neurons with many active knobs terminating on them are in the **discharge zone** & stimulation of pre synaptic neuron causes them to discharge
- Neurons with few active knobs terminating on them are in the **subliminal fringe** & they do not discharge but have increased excitability by activity in pre synaptic neuron



9. Synaptic fatigue

- Repeated stimulation of Presynaptic neuron → gradual ↓ & finally disappearance of post synaptic response

- Reasons
  - Exhaustion of NT
  - Gradual inactivation of Ca^{2+} channels → ↓ Ca^{2+} influx
  - Progressive inactivation of postsynaptic receptors
  - Slow development of abnormal concentration of ions inside post synaptic neuron
- Protective mechanism against excess neuronal activity

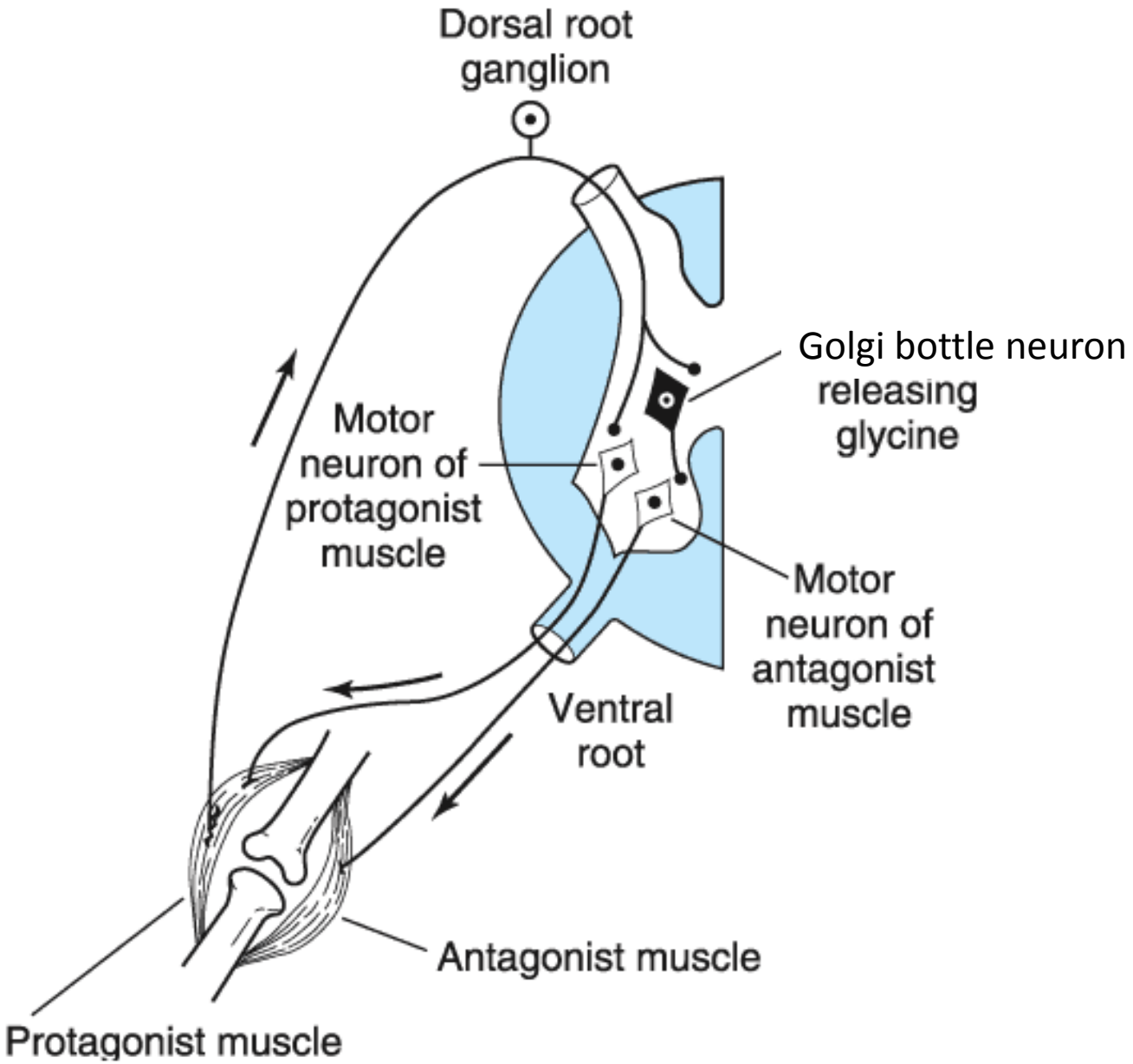
10. Synaptic inhibition (Short essay)

- **Post synaptic Inhibition**
  - **Indirect**
  - **Direct**
    - Direct post synaptic
    - Negative feedback
    - Feed forward
- **Pre synaptic Inhibition**

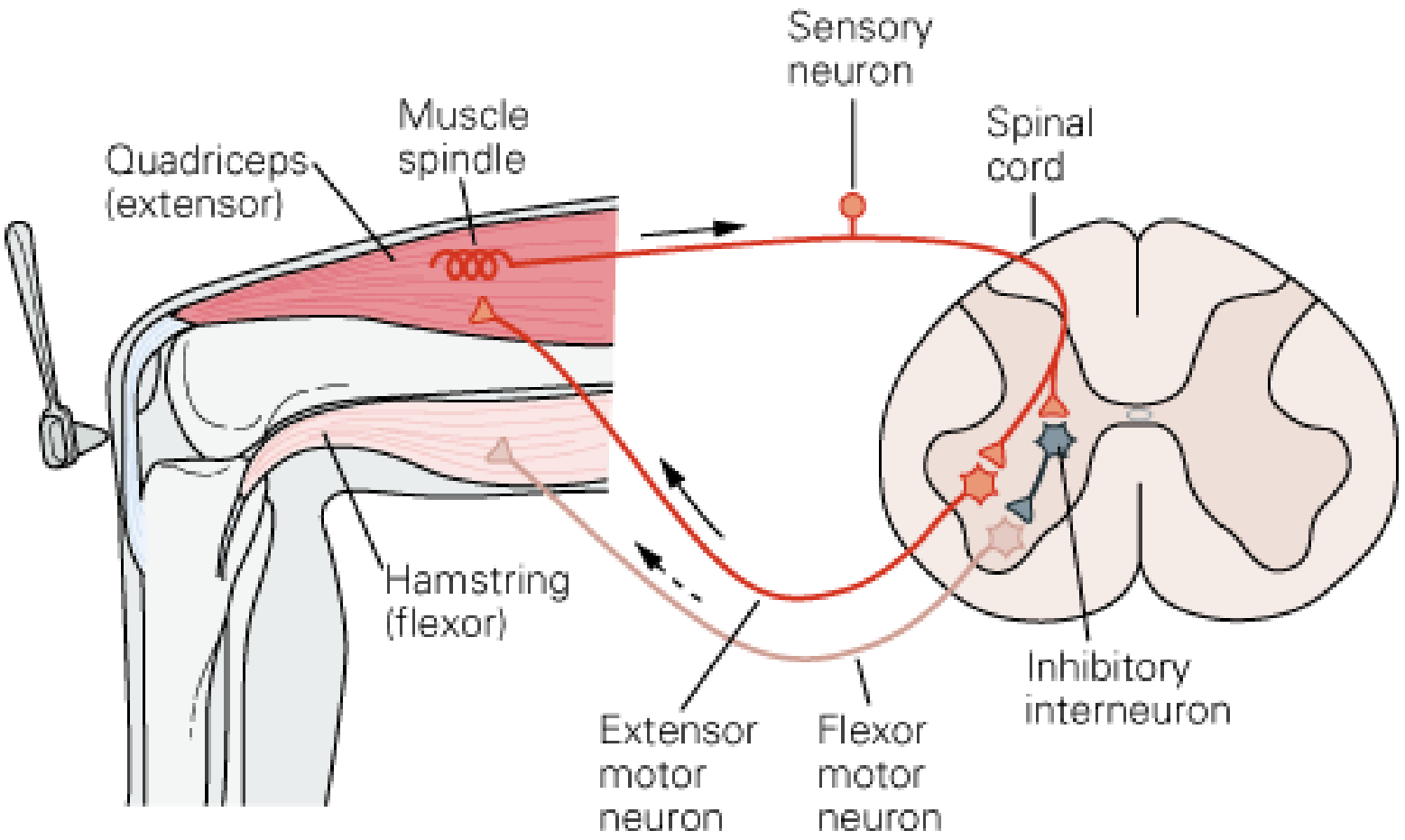
Indirect Post synaptic inhibition

- **Indirect** –inhibition due to effects of previous post synaptic N discharge
- IPSP is not produced
- Mechanisms
  - Post syn.N can be refractory to excitation because it is in refractory period of a previous impulse
  - During hyperpolarisation of a previous impulse

Direct post synaptic inhibition



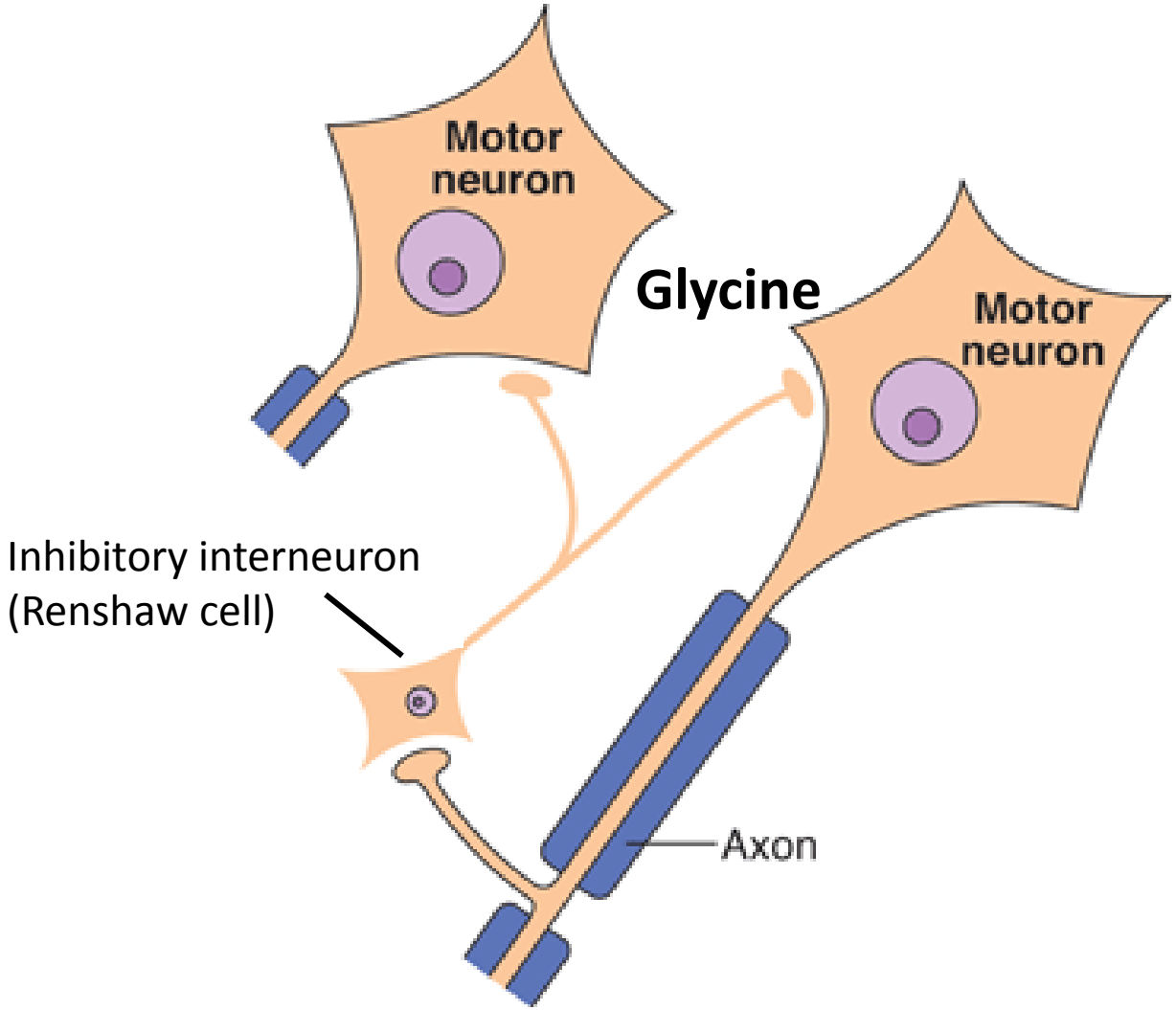
Reciprocal innervation



Direct post synaptic inhibition

- Occurs during the course of an IPSP
- Due to release of inhibitory NT from presynaptic N
- Afferent from stretch receptors in muscle spindle →pass directly to spinal motor N supplying the same muscle
- Impulses in afferents cause EPSPs & AP in post synaptic motor N supplying the same ms

Negative feedback inhibition of a spinal motor neuron via an inhibitory interneuron (Renshaw cell).

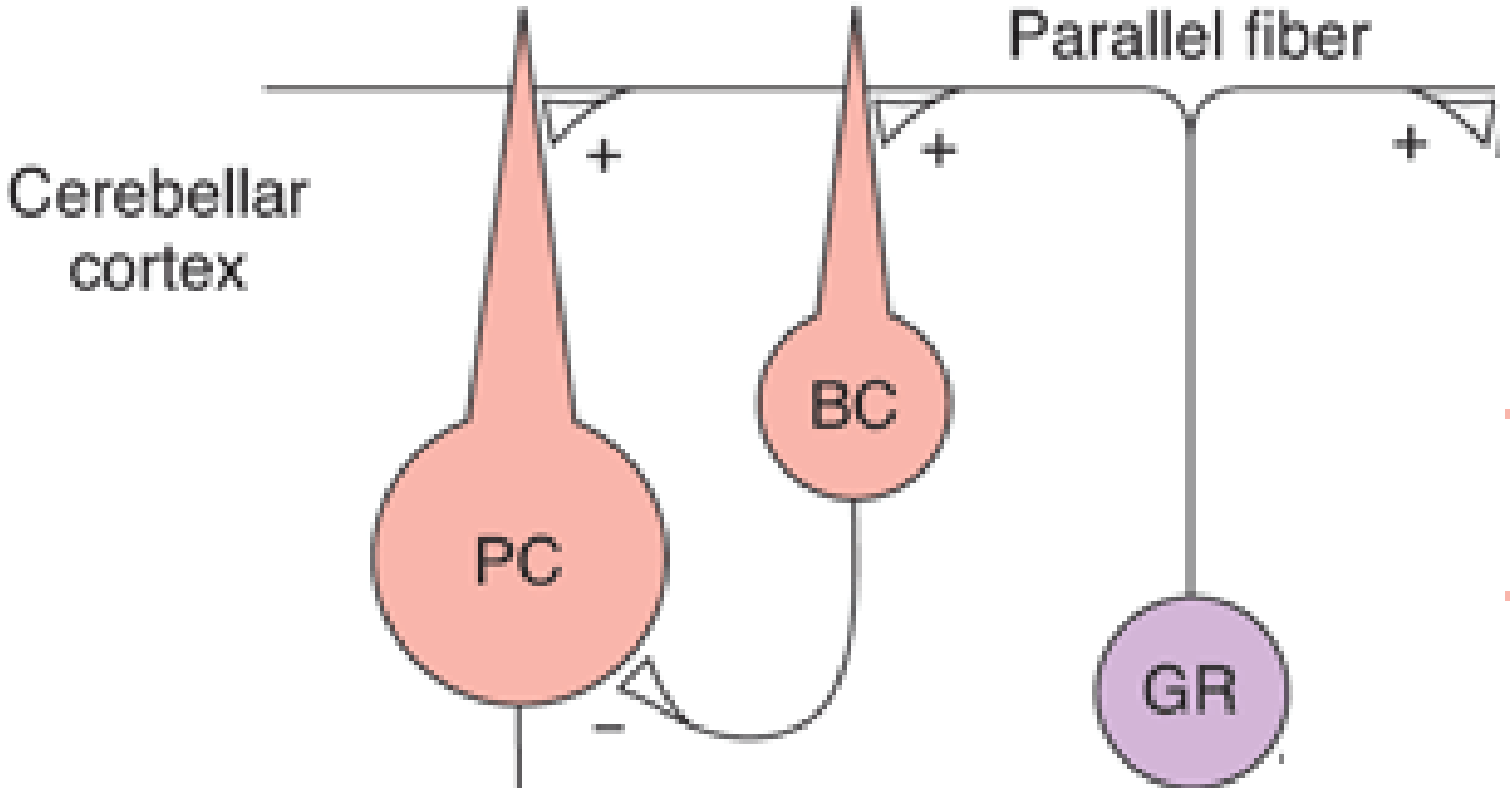


Negative feedback inhibition (Renshaw cell inhibition)

- Neurons inhibit themselves in a negative feedback manner
- Seen in spinal cord, cerebral cortex, limbic system
- Each spinal motor N gives off a recurrent collateral which synapses with an inhibitory interneuron –**Renshaw cell**
- Renshaw cell in turn synapses with cell body of same motor N & also other spinal motor N

- When the motor N is stimulated, impulses pass through the collateral to Renshaw cell also
- This cell secretes Glycine which slows / stops discharge of motor N

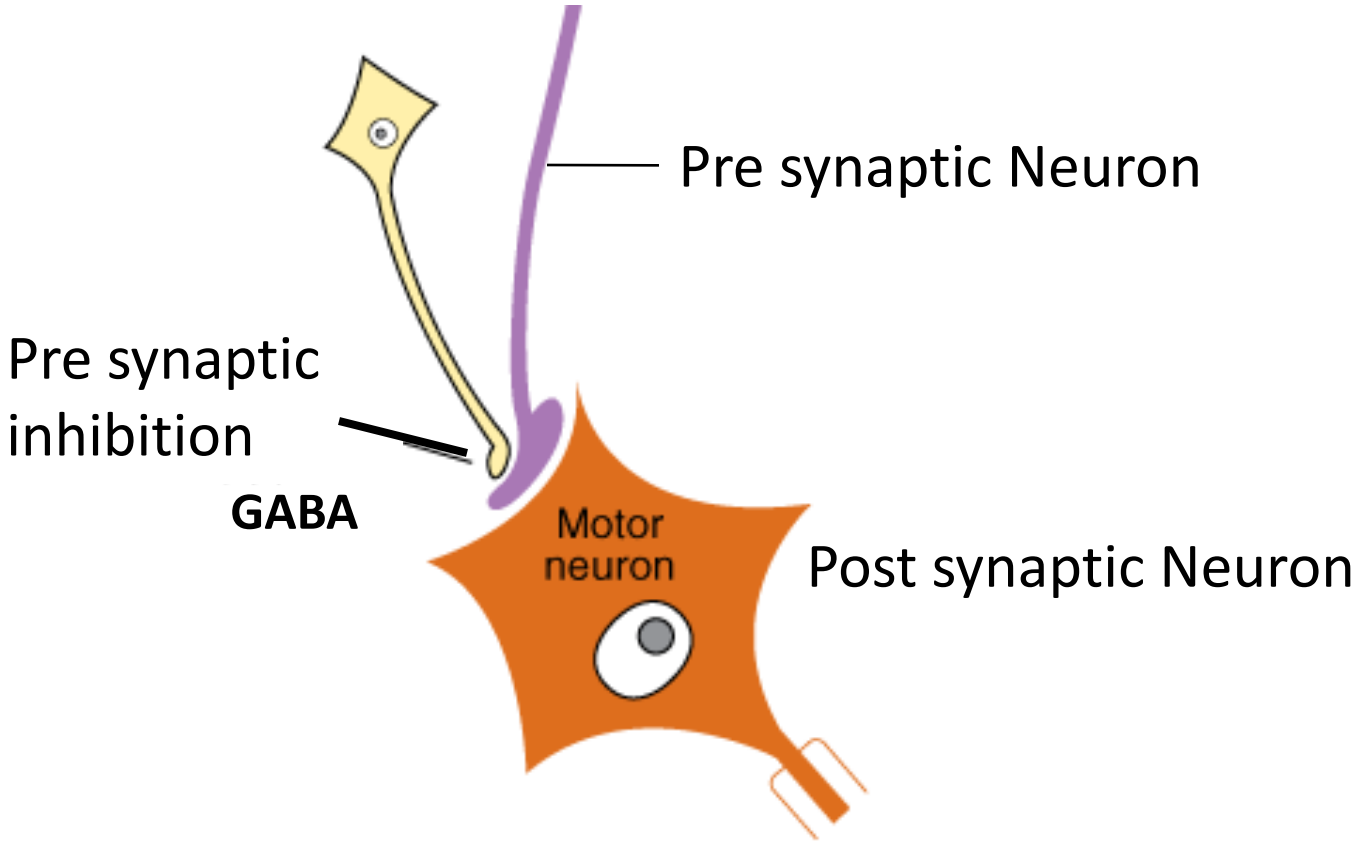
Feed forward inhibition –cerebellar cortex



Feed forward inhibition

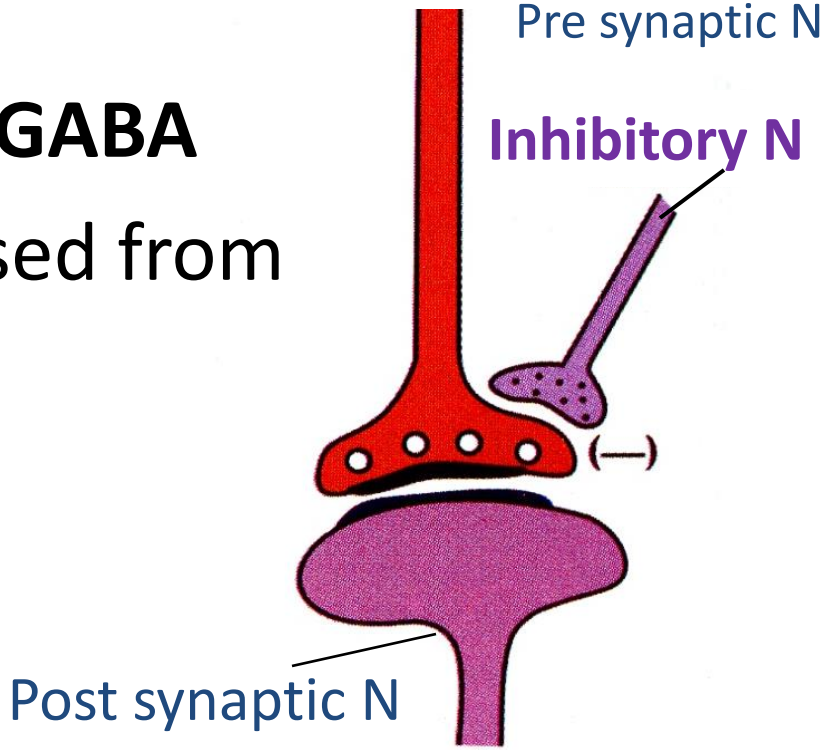
- Seen in cerebellar cortex
- Branches of **Granule cells** called parallel fibres (afferent fibres) excite both **basket cells & Purkinje cells**
- But stimulation of Basket cells produce IPSPs in Purkinje cells
- This limits the duration of excitation of Purkinje cells & output from Purkinje cells is controlled

Presynaptic inhibition



Presynaptic inhibition

- Occurs at presynaptic terminal before the signal reaches the synaptic area
- Mediated by inhibitory neurons that form axo - axonal synapse with excitatory pre synaptic terminal
- Releases inhibitory NT –eg:**GABA**
- ↓ the quantity of NT released from presynaptic terminal



Three mechanisms of **pre synaptic inhibition**

1. Activation of Presynaptic N -↑ Cl^- influx →↓size of AP reaching pre synaptic terminal→ ↓ Ca^{2+} influx→ ↓excitatory NT release
2. Opening of VG K^+ channels –↑ K^+ efflux → ↓ Ca^{2+} influx → ↓ excitatory NT release
3. Direct inhibition of NT release independent of Ca^{2+} influx into excitatory presynaptic ending

- Presynaptic inhibition
  - **In pain pathways**
  - Collaterals from ascending touch pathways →pre synaptic inhibition at the dorsal horn - (gating of pain) → so when touch fibers are activated pain is inhibited
  - Descending central analgesic pathway terminate on afferent pain fibers at the dorsal horn gate → pre synaptic inhibition of pain fibers

Significance of synaptic inhibition

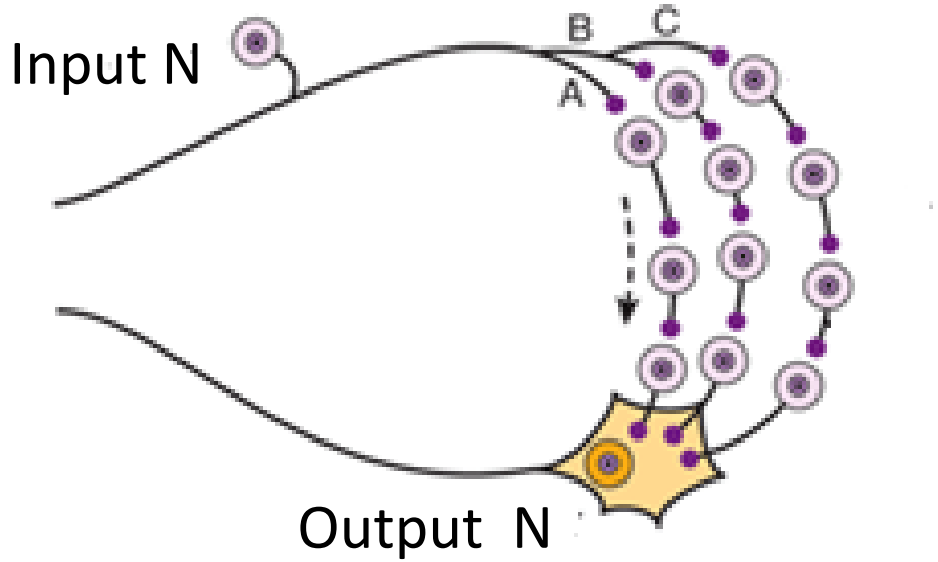
- Synaptic inhibition offers restriction over neurons & muscles so that they can react properly & accurately
- Inhibition helps to select proper no: of impulses to be transmitted & to block excess ones
- Minimizes sideways spread and mixing of signals in sensory tracts

11. After discharge

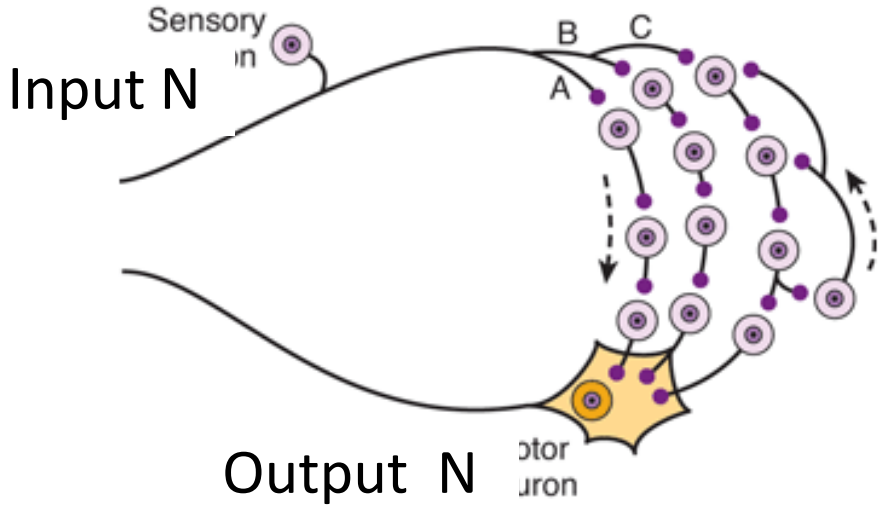
- Prolonged output discharge even after incoming signal is over
- Can last a few ms to as long as many minutes
- It can be
  - Synaptic after discharge
  - Parallel circuit type
  - Reverberating (Oscillatory) circuit type

- **Synaptic after discharge**
  - Post synaptic electrical potential which can last for many ms
  - Occurs when NT is long acting
  - As long as the post synaptic N is excited → it transmits a continuous train of output impulses

- Parallel circuit type of after discharge
  - Many parallel pathways from presynaptic N converge on postsynaptic N
  - Impulses reach output N one after the other as there is synaptic delay of 0.5ms at each synapse
  - Output N discharges repeatedly & for long duration



- Reverberating (Oscillatory) circuit type
  - Common & important in brain & SC
  - Such circuits are caused by positive feedback within the neuronal circuit
  - Some branch pathways turn back on themselves via collaterals to re excite the input of the same circuit
  - Consequently, once stimulated, the circuit may discharge repetitively for a long time.
  - Cessation of activity occurs as a result of fatigue



12. Synaptic plasticity (Short essay)

- Refers to the short or long term strengthening / weakening of synaptic conduction on the basis of past experience
- Can be due to changes in pre synaptic or post synaptic neuron
- Of great interest as they represent forms of learning & memory

Synaptic plasticity

- Post tetanic potentiation
- Habituation
- Sensitization
- Long term potentiation
- Long term depression

Post tetanic potentiation

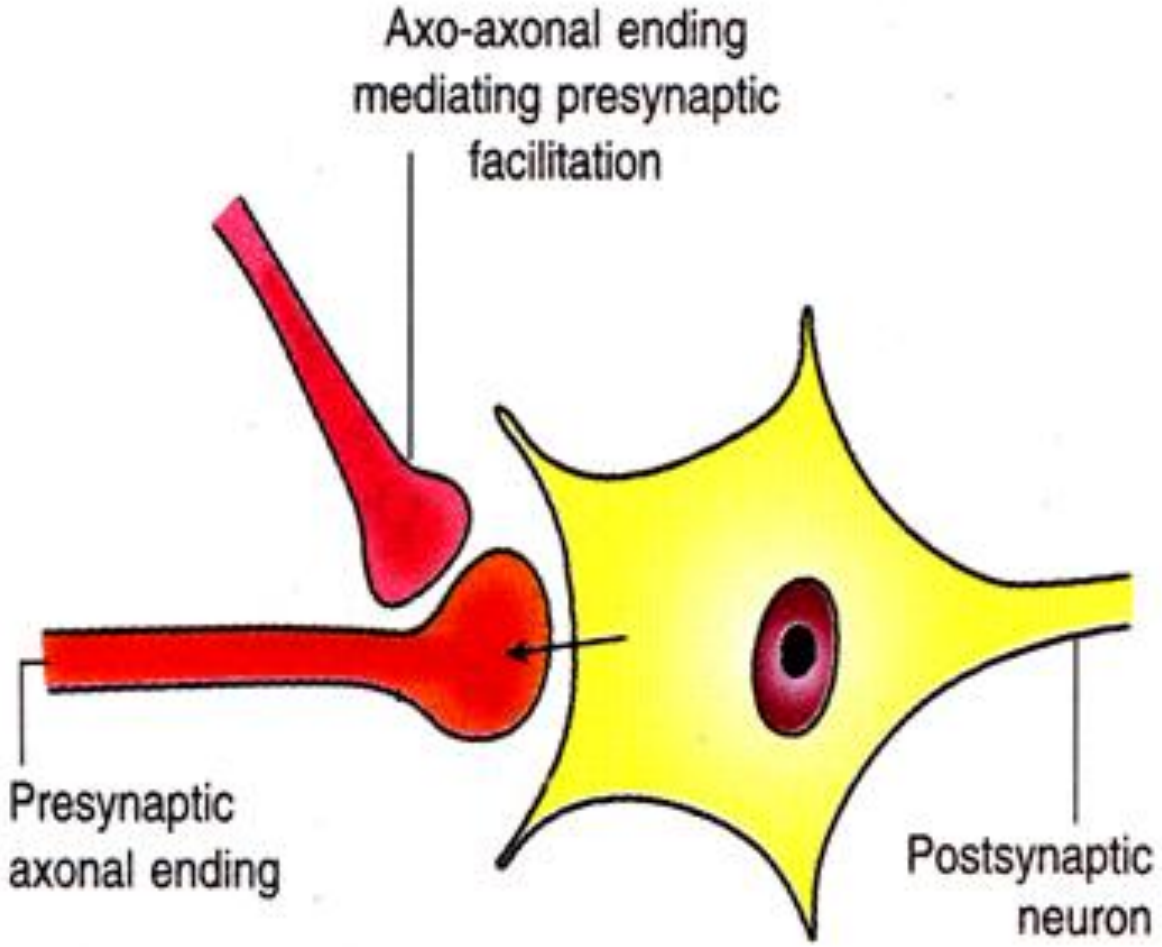
- Production of enhanced postsynaptic potentials in response to a stimulus
- Occurs during a brief period of time after cessation of a train of tetanizing stimuli applied to pre synaptic neuron
- Enhancement lasts up to 60 sec
- Tetanizing stimulation of pre synaptic neuron → ↑ Ca^{2+} accumulation in **pre synaptic terminal**

Habituation

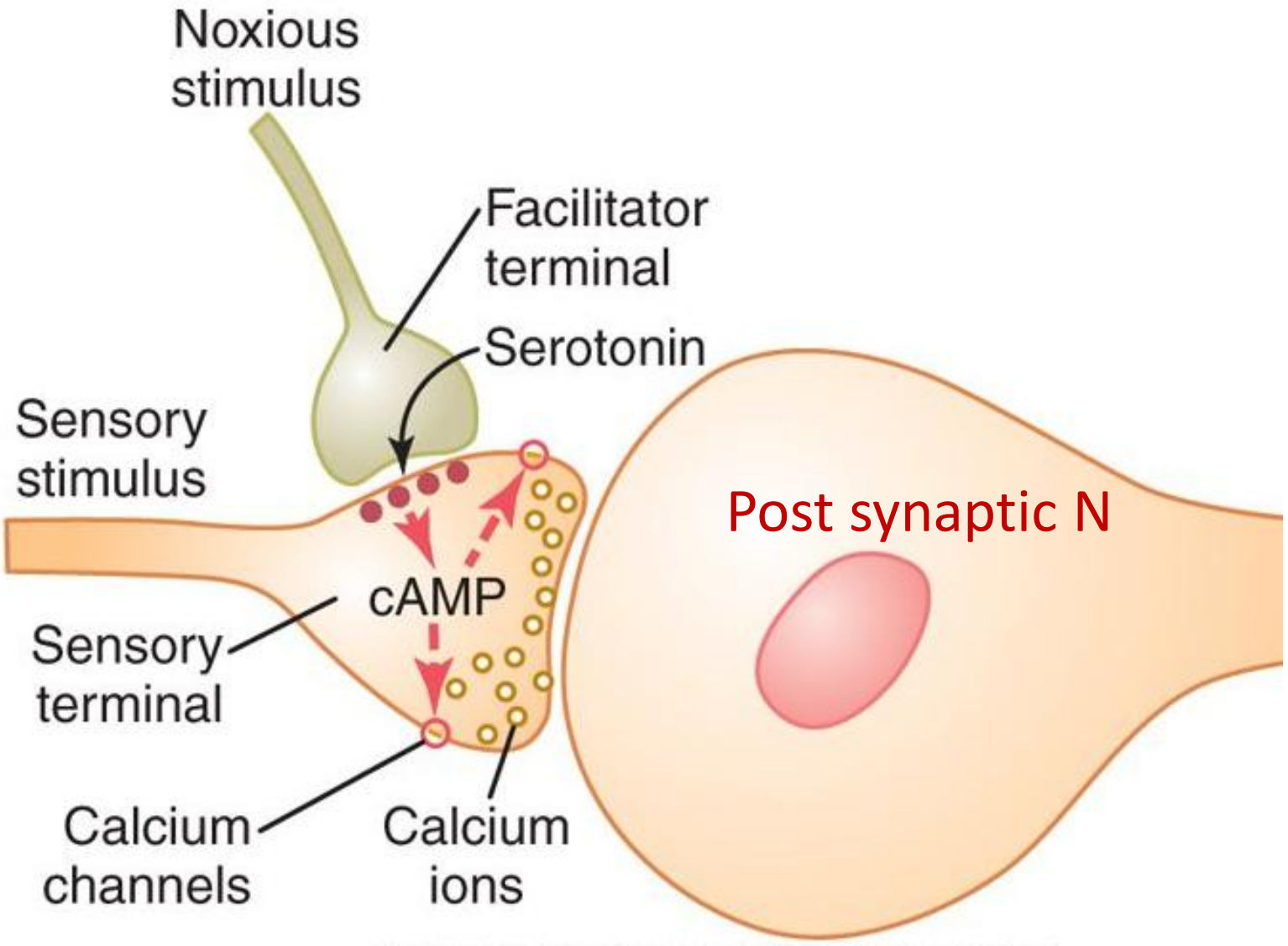
- Same as synaptic fatigue
- Repeated benign stimulation of Presynaptic neuron → gradual ↓ & finally disappearance of post synaptic response
- Can be short term or can be long term if the benign stimulus is repeated many times

- Reasons
  - Exhaustion of NT
  - Gradual inactivation of Ca^{2+} channels → ↓ Ca^{2+} influx
  - Progressive inactivation of postsynaptic receptors
  - Slow development of abnormal concentration of ions inside post synaptic neuron
- Protective mechanism against excess neuronal activity

Sensitization



Sensitization



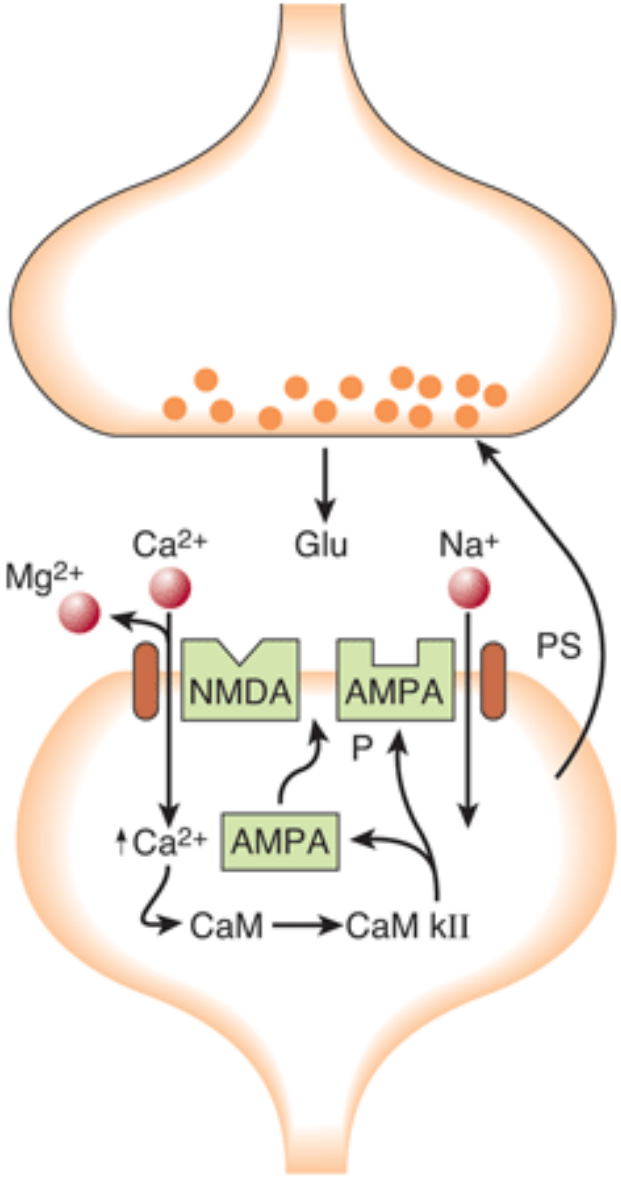
Sensitization

- Opposite to Habituation
- Sensitization is the **prolonged occurrence** of **augmented post synaptic response** when a benign stimulus to which an animal has become habituated is paired once or several times with a noxious stimulus
- Noxious stimulus →release of **serotonin** from axo –axonal ending on pre synaptic sensory neuron → pre synaptic facilitation
- Benign stimulus repeated → habituation
- Benign stimulus + noxious stimulus → sensitization - become more sensitive to benign stimulus

Mechanism of sensitization

- Serotonin binds to receptors on presynaptic N→ activates adenylyl cyclase → ↑ cAMP →activates protein kinase →phosphorylates K^+ channels & closes them → ↓ K^+ efflux →prolongs AP → ↑ Ca^{2+} influx in pre synaptic terminal → ↑ NT release →prolonged augmented postsynaptic response
- Sensitization can be a **transient** response
- Or can be **prolonged** response if reinforced by additional pairing of benign & noxious stimuli
- Then it exhibit features of –short term or long term memory

Long term potentiation (LTP)



- Rapid, persistent enhancement of post synaptic potential in response to pre synaptic stimulation, after a brief period of rapidly repeated stimulation of presynaptic Neuron
- Can last for days
- Due to **↑ Ca^{2+} in post synaptic neuron**

- Long -term potentiation also involves protein synthesis and growth of the presynaptic and postsynaptic neurons and their connections
- Eg in hippocampus- long term memory

Long term depression (LTD)

- LTD present in same fibers as LTP
- Opposite of LTP
- Characterized by ↓ in synaptic strength
- Due to slower stimulation of pre synaptic neurons → smaller rise in intra cellular Ca^{++} in post synaptic terminal
- Maybe the mechanism by which learning occurs in cerebellum

Other factors affecting synaptic transmission

- ✓ Acidosis – greatly depress neuronal activity
- When pH falls from 7.4 to 7 it can cause coma
- ✓ Alkalosis – greatly increase neuronal excitability
- When pH rises from 7.4 to 7.8 it can cause epileptic seizures
- Hyper ventilation can precipitate seizures in pre disposed individuals

- ✓Hypoxia –neuronal excitability is highly dependent on oxygen supply
  - Cessation of O_2 supply for even a few sec →complete inexcitability of some neurons
  - Decreased cerebral blood flow can cause unconsciousness within 3-7 sec

- ✓ Drugs –caffeine , theophylline, theobromine can ↑ neuronal excitability by decreasing threshold for excitation
- Strychnine increase excitability- by inhibiting action of inhibitory neurotransmitters esp Glycine
- Anesthetics decrease excitability - ↑neuronal threshold