



CNS1: SYNAPSE

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SYNAPSE

DEFINITION:

FUNCTIONAL JUNCTION BETWEEN TWO NEURONS IS CALLED SYNAPSE.

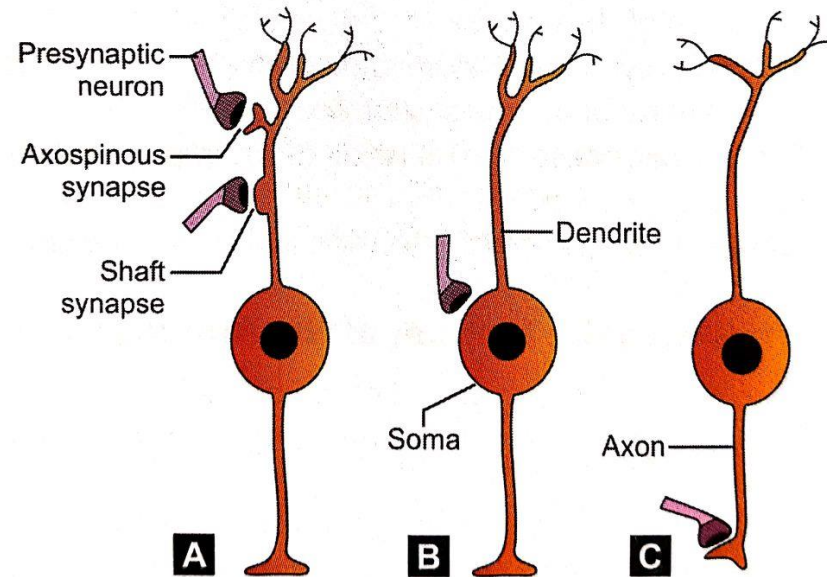
NO ANATOMICAL CONTINUITY

ONLY PHYSIOLOGICAL (FUNCTIONAL)
CONTINUITY

CLASSIFICATION

– ANATOMICAL CLASSIFICATION:

- * Axosomatic: Between axon, and cell body of nxt neuron
- * Axodendritic: Bet. Axon, and dendries of nxt neuron
- * Axoaxonic: Bet. Axon, and axon of nxt. Neuron
- * Dendrodendritic: Bet. Dendrite, and dendrite of nxt. neuron



Figs. 116.1A to C: Types of synapses. (A) Axodendritic synapse; (B) Axosomatic synapse; (C) Axoaxonic synapse.

Comprehensive Textbook of
Medical Physiology, Dr. G K Pal

• FUNCTIONAL CLASSIFICATION

1 Chemical: Neurotransmitter

Type I: excitatory

Type II: inhibitory

2) Electrical: gap junctions or low resistance bridges +nt.

invertebrates, cardiac and smooth muscles.

3) Conjoint: Chemical + Electrical

Structure of synapse

- PRESYNAPTIC TERMINAL
 - SYNAPTIC CLEFT
 - POSTSYNAPTIC TERMINAL
-

– PRESYNAPTIC TERMINAL:

- * Axon terminal
- * Synaptic vesicles: Neurotransmitter
- * Mitochondria
- * Presynaptic membrane: dense bodies, Ca⁺⁺ channels

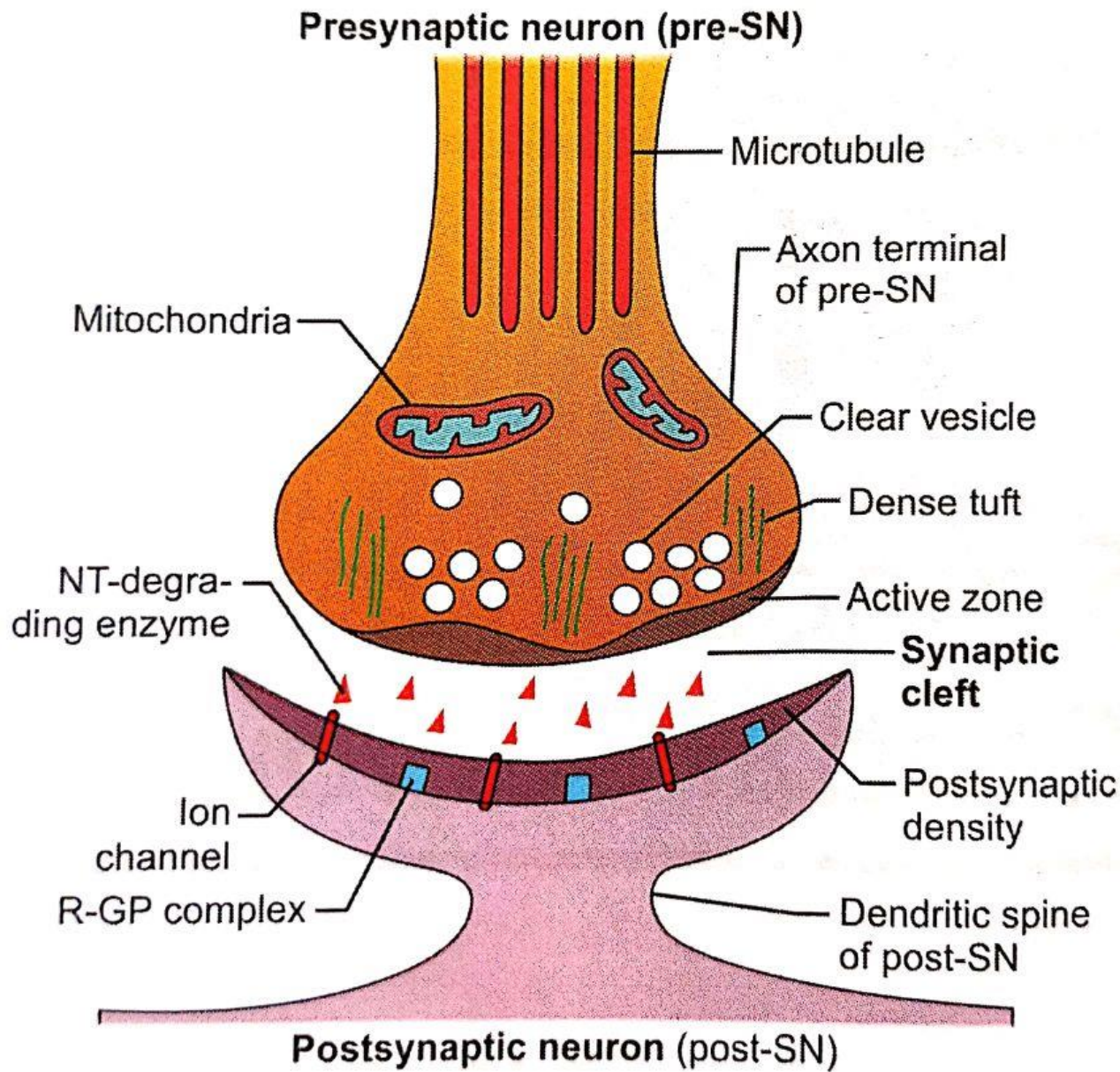


Fig. 116.2: Functional anatomy of an axodendritic synapse. Note the components of presynaptic (Pre-SN) and postsynaptic (Post-SN) neurons. (NT: Neurotransmitter; R-GP: Receptor-G protein).

– **SYNAPTIC CLEFT:** 200 Angstrom

ECF.

Enzymes, ions.

– **POSTSYNAPTIC TERMINAL:**

Postsynaptic membrane

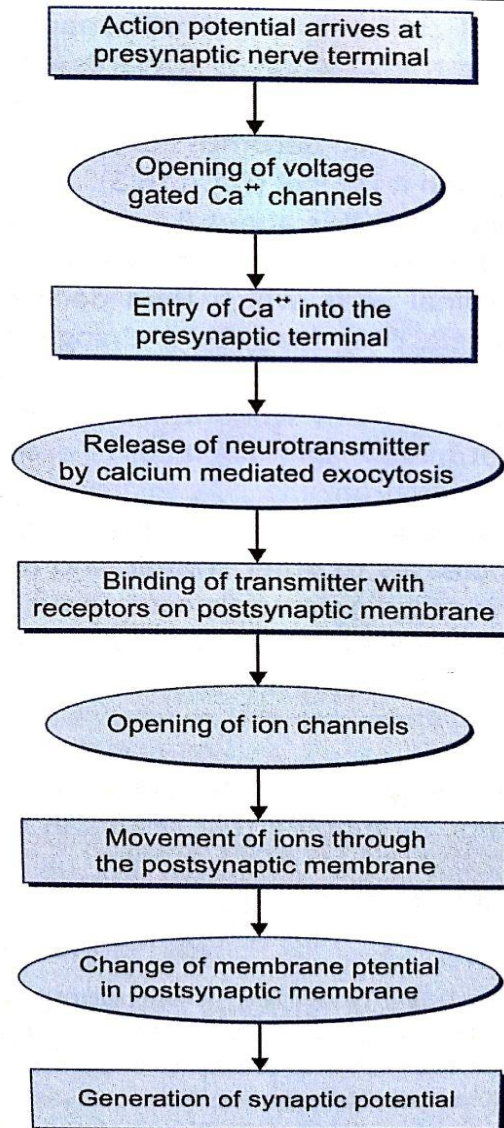
Receptor proteins:

1) Ion channel type: ligand gated

2) Enzyme type

MECHANISM OF SYNAPTIC TRANSMISSION

Flowchart 116.1: Steps of synaptic transmission.



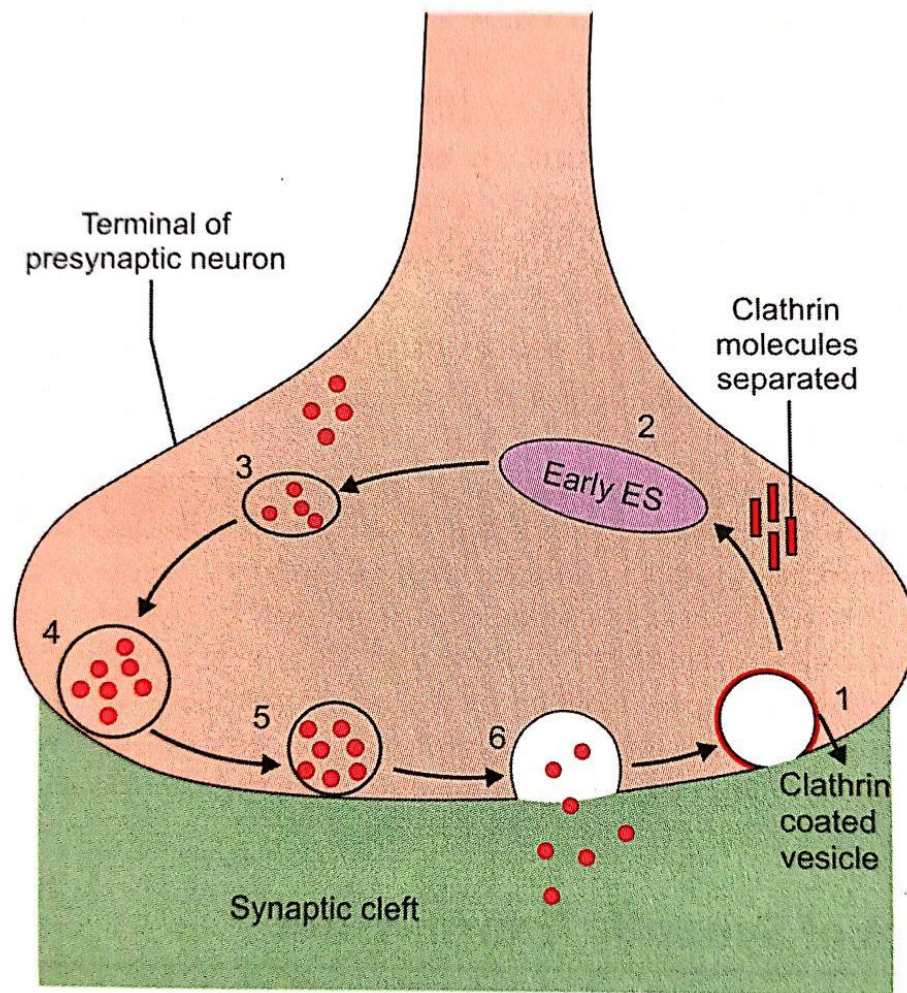


Fig. 116.3: Recycling of neurotransmitter vesicle in presynaptic neuron terminal. This occurs in six steps. 1. The vesicle that has discharged its content into the synaptic cleft is coated with clathrin and get detached from the membrane; 2. Clathrin is separated from the vesicle and vesicle fuses with early endosomes (Early ES); 3. New vesicle is formed from early ES, which picks up NT from nerve terminal; 4. Docking of vesicle containing NT with the membrane of presynaptic neuron terminal; 5. Priming of the vesicle; 6. Discharge of NT into synaptic cleft from vesicle by calcium-mediated exocytosis.

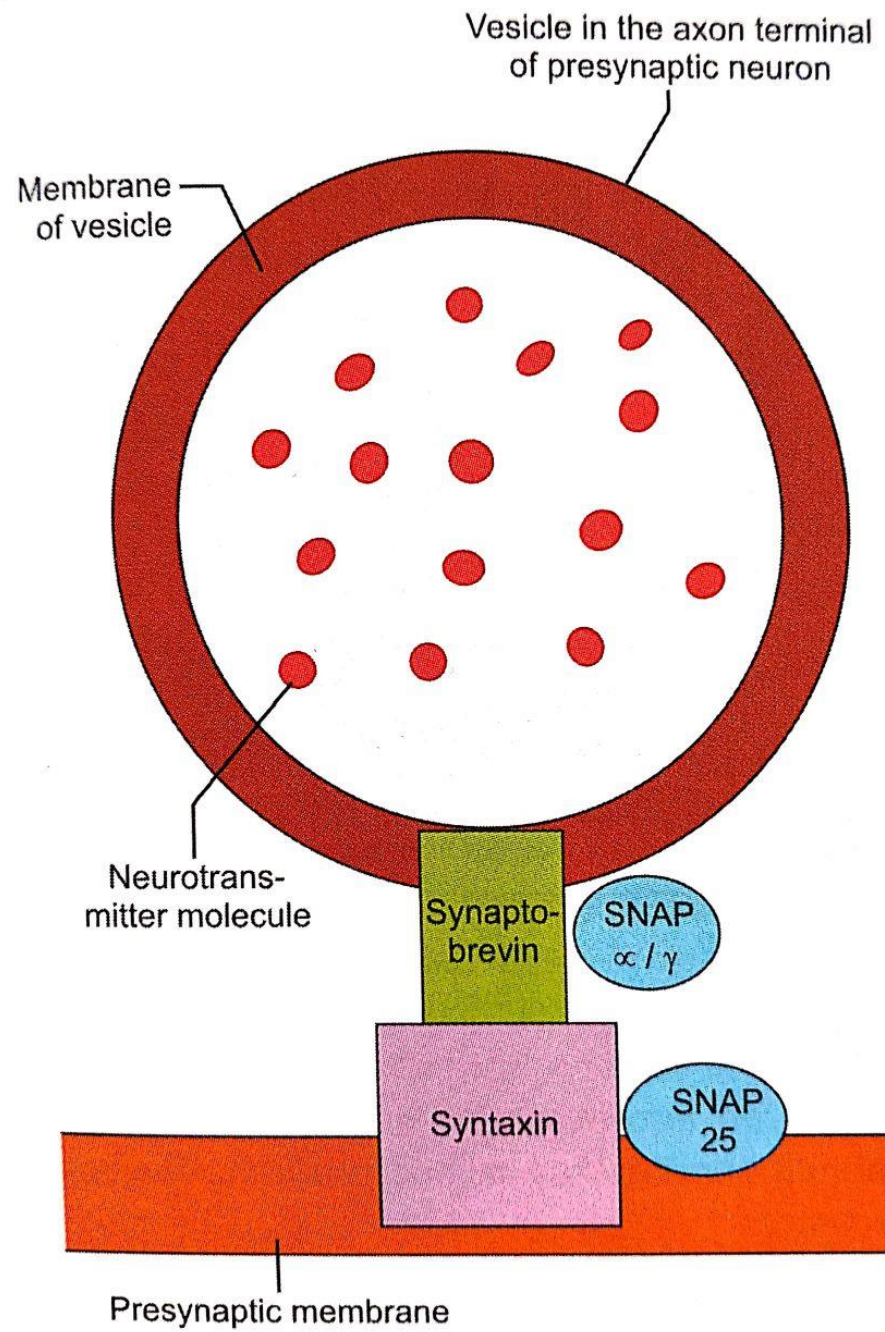


Fig. 116.4: Priming and docking. Note the role of syntaxin and synaptobrevin



Post synaptic receptor potential can be:

EPSP: excitatory post synaptic potential OR

IPSP: inhibitory post synaptic potential

If EPSP is more, AP develops at axon hillock : due to opening of voltage gated Na^+ channels.

AP propagates along the axon to the axon terminal.

FUNCTIONS OF SYNAPSE

– TO TRANSMIT IMPULSES FROM ONE NEURON TO ANOTHER NEURON.

* **EXCITATORY SYNAPSE:**

EPSP: EXCITATORY POST SYNAPTIC POTENTIAL

* **INHIBITORY SYNAPSE:**

IPSP: INHIBITORY POST SYNAPTIC POTENTIAL

EPSP



- EXCITATORY POST SYNAPTIC POTENTIAL
- EXCITATORY NEUROTRANSMITTER
- OPENING OF Na^+ OR Ca^{++} CHANNELS
- CLOSURE OF K^+ OR Cl^- CHANNELS

MECHANISM

- NEUROTRANSMITTER ACTIVATES THE CONVERSION OF ATP TO c-AMP.
- CONVERSION OF INACTIVE PROTEIN KINASE TO ACTIVE FORM.
- EITHER c-AMP OR ACTIVATED PROTEIN KINASE OPENS UP APPROPRIATE IONIC CHANNELS
- DEPOLARIZATION OF THE POST SYNAPTIC MEMBRANE CALLED EPSP.

RECIPROCAL INHIBITION

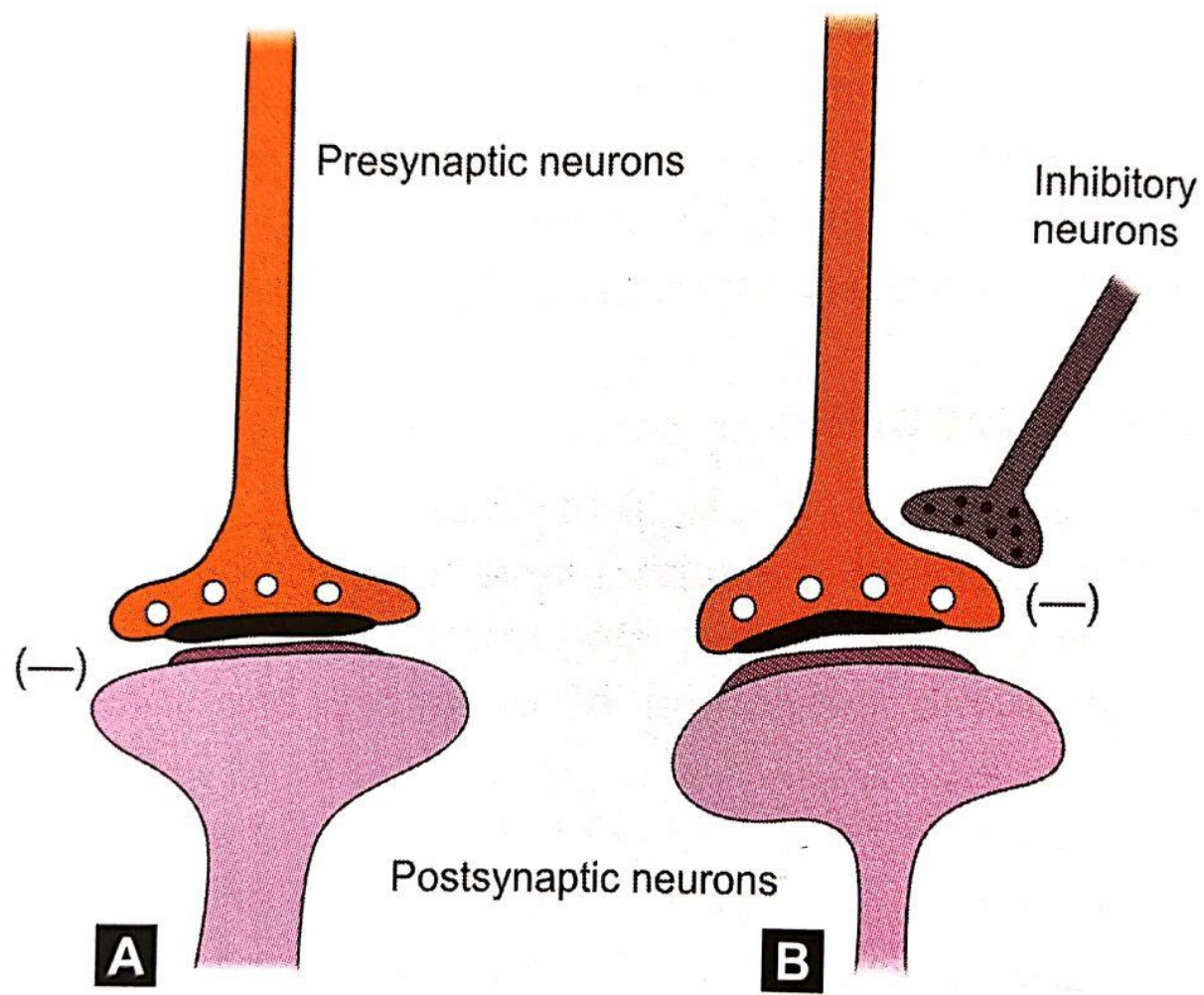
- During muscular activities like walking, running etc which require stimulation of one group of muscles and inhibition of antagonistic group of muscles (e.g. stimulation of flexors and inhibition of extensors at the same time).
- INHIBITION, RESTRICTS ACTIVITY OF POSTSYNAPTIC NEURON OR MUSCLE.

SYNAPTIC INHIBITION

- IPSP: INHIBITORY POST SYNAPTIC POTENTIAL
- INHIBITORY PRE SYNAPTIC POTENTIAL
- RENSHAW CELL INHIBITION OR NEGATIVE FEEDBACK INHIBITION
- FEED FORWARD INHIBITION

INHIBITORY POST SYNAPIC POTENTIAL

- INHIBITORY NEUROTRANSMITTER
- OPENING OF K^+ OR Cl^- CHANNELS
- RMP BECOMES MORE NEGATIVE
- INTERNEURON: GOLGI BOTTLE NEURONS
- IN SPINAL CORD: NT-GLYCINE



Figs. 116.7A and B: Direct (A) and indirect (B) inhibitions at synapses. In direct inhibition, an inhibitory presynaptic neuron directly inhibits postsynaptic neuron by releasing inhibitory neurotransmitter. In indirect inhibition, presynaptic neuron is not inhibitory, but, another inhibitory neuron projects to the axon terminal of presynaptic neuron that inhibits the release of neurotransmitter from it (therefore, the transmission is inhibited). Minus sign depicts inhibition.

Presynaptic inhibition: indirect

- THIS TYPE OF INHIBITION OCCURS AT PRESYNAPTIC TERMINALS BEFORE THE SIGNALS EVER REACH THE SYNAPSE.
- AXOAXONIC SYNAPSE
- INHIBITORY NT: GABA
- HYPERPOLARISATION: OPENING OF K^+ OR Cl^- CHANNELS

RENSHAW CELL INHIBITION

- Some motor neurons in the spinal cord give collaterals in the spinal cord.
- make excitatory synapses with interneuron called as Renshaw cells.
- Renshaw cells in turn inhibit the same motor neurons which have given rise to collateral.
- Glycine is the neurotransmitter released at the end of Renshaw cells.
- **NEGATIVE FEEDBACK MECHANISM**

RENSHAW CELL INHIBITION

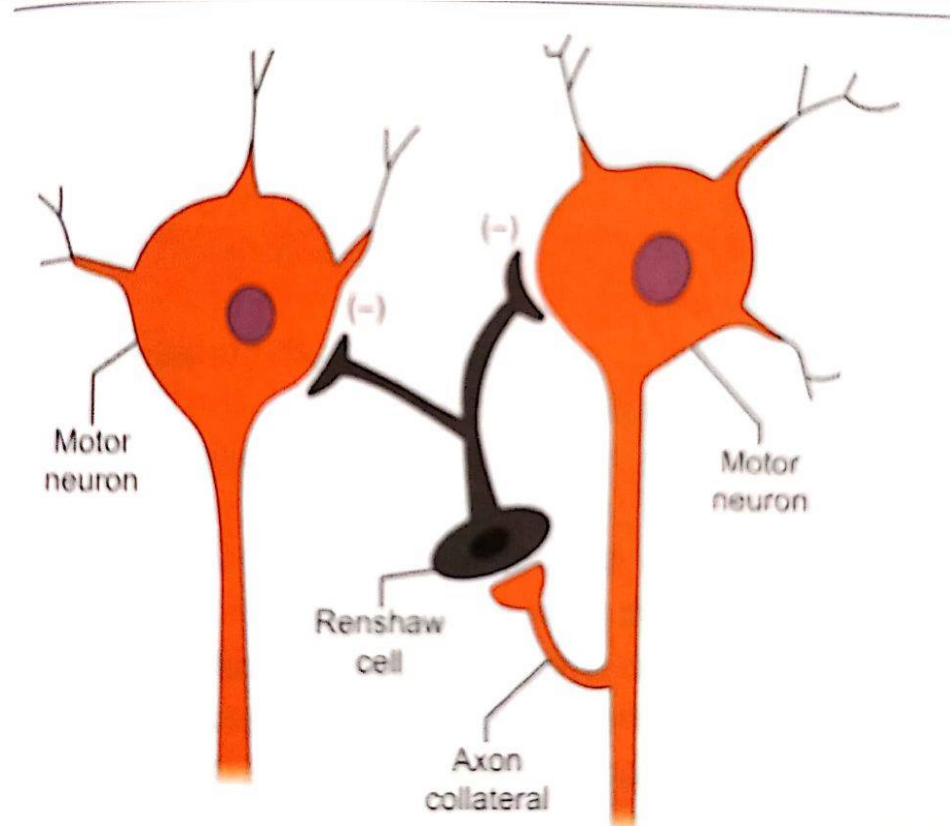


Fig. 116.8: Negative feedback inhibition by Renshaw cell. Collateral from spinal motor neuron ends on an inhibitory interneuron (Renshaw cell) that in turn inhibits (minus sign) the discharge of the same motor neuron. Collateral from Renshaw cell also inhibits neighboring motor neuron, which is called lateral inhibition.



FEED FORWARD INHIBITION:

- Present in the cerebellum.
- Cerebellum has a unique neuronal circuit consisting of granule cells, basket cells and purkinjee cells.
- **ALLOWS BRIEF AND PRECISE TIMED EXCITATION.**

PROPERTIES OF SYNAPSE

- ONE WAY CONDUCTION
- SYNAPTIC DELAY
- FATIGUE
- SUMMATION: SPATIAL & TEMPORAL
- CONVERGENCE & DIVERGENCE
- AFTER DISCHARGE
- OCCLUSION
- SUBLIMINAL FRINGE
- SYNAPTIC PLASTICITY: POST TETANIC POTENTIATION, HABITUATION, SENSITIZATION, PRESYNAPTIC INHI.
- REVERBERATION
- RECIPROCAL INHIBITION

ONE WAY CONDUCTION

- ONLY FROM PRESYNAPTIC TO POSTSYNAPTIC TERMINAL
- BELL AND MAGENDIE LAW
- BECAUSE, ONLY PRESYNAPTIC TERMINAL CONTAIN NT,
- AND POSTSYNAPTIC TERMINAL HAS RECEPTORS.
- IMPORTANCE: FOR ORDERLY CONDUCTION OF IMPULSE IN ONE DIRECTION

SYNAPTIC DELAY

– TIME LOST BY IMPULSE TO PASS FROM PRE TO POSTSYNAPTIC TERMINAL

– 0.5 ms.

– IMPORTANCE:

* ONE OF CAUSES OF LATENT PERIOD OF REFLEX ACTIVITY.

* NO. OF SYNAPSES CAN BE ESTIMATED

SYNAPTIC FATIGUE / habituation

- REPEATED STIMULUS OF POSTSYNAPTIC NEURON, DECREASES RESPONSE AND THEN DISAPPEARANCE.
- TEMPORARY.
- CAUSE: DEPLETION OF NT, INACTIVATION OF Ca^{++} CHANNELS, ACCUMULATION OF WASTE.
- FIRST SEAT OF FATIGUE IN INTACT AND EXPERIMENTAL PREPARATION: Synapses in NS.

summation

spatial summation.

when the presynaptic terminals, which are nearly situated on a membrane, are stimulated simultaneously their effects are summed. It is called

temporal summation.

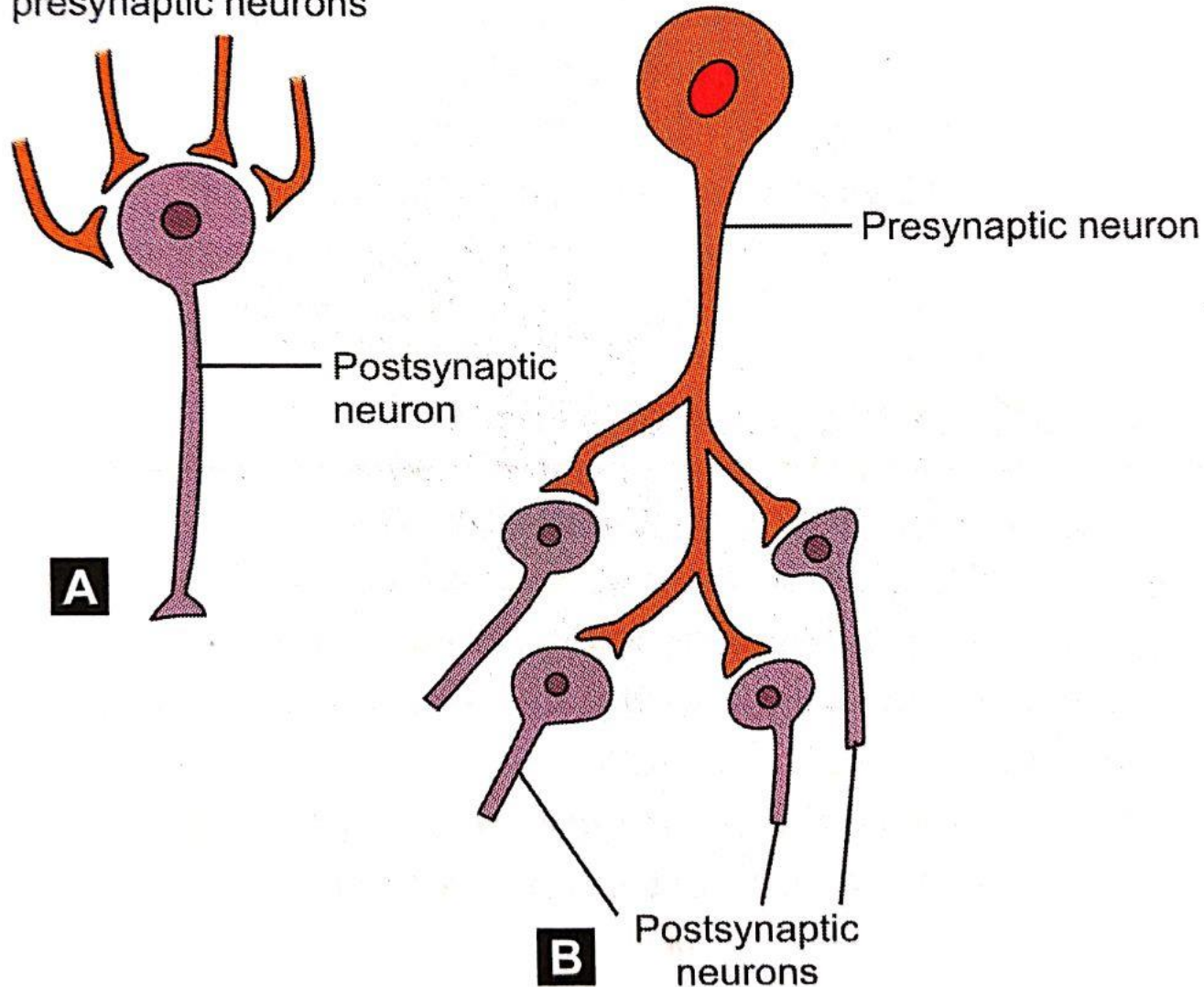
when a single presynaptic terminal is repetitively and rapidly stimulated, the effects produced by impulses are added. It is called



CONVERGENCE: COMING TOGETHER
IN SPINAL CORD

DIVERGENCE: DISTRIBUTING

Axon terminals of
presynaptic neurons



Figs. 116.6A and B: Convergence (A) and divergence (B). In Convergence, many presynaptic neurons project to one postsynaptic neuron, and in divergence, one presynaptic neuron projects to many postsynaptic neurons.

subliminal fringe and occlusion

when the presynaptic terminals are separately stimulated, sum of their effects is less than the effect, which is produce when they are simultaneously stimulated. This property is called

subliminal fringe

when the presynaptic terminals are separately stimulated, sum of their effects is more than the effect, which is produce when they are simultaneously stimulated. This property is called

occlusion

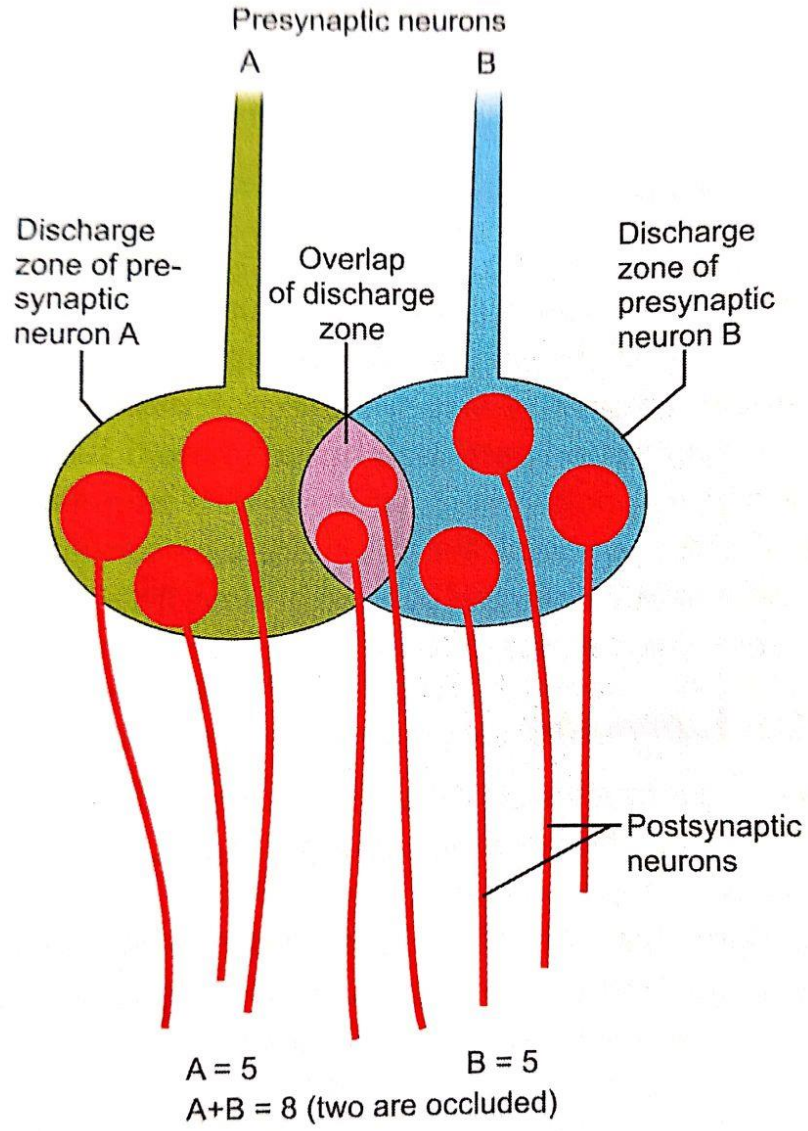


Fig. 116.9: Mechanism of occlusion. Activation of presynaptic neuron A stimulates a set of five postsynaptic neurons and activation of presynaptic neuron B stimulates another set of five postsynaptic neurons, when A and B are activated separately. But, as two presynaptic neurons share their discharge zone (common area of A and B), simultaneous activation of A and B results in activation of eight postsynaptic neurons instead of ten. This is called occlusion.

SYNAPTIC PLASTICITY

<p>Post tetanic potentiation</p>	<p>When a presynaptic neuron is stimulated with a single stimulus, followed by stimulation with a volley of stimuli for 2 sec and then again with single stimulus, the second stimulus produces larger postsynaptic response than the first stimulus.</p>
<p>Inhibition</p>	<p>post synaptic (direct) / presynaptic /renshaw cell / reciprocal inhibition of sherrigton</p>
<p>Habituation</p>	<p>if the stimulus is non-injurious or unimportant and applied repeatedly , the response progressively becomes lesser and lesser and may cease (decreased NT and Ca^{++})</p>
<p>Sensitization</p>	<p>if the stimulus is injurious or important and applied repeatedly , the response progresively becomes more and more.</p>

AFTER DISCHARGE

- When a presynaptic neuron is continuously stimulated with high frequency the post synaptic neuron responds.
- But if the stimulation is suddenly removed, the response in the postsynaptic neuron continues in spite of stoppage of stimulus. This is called as after discharge.
- It occurs due to presence of **reverberating circuits**.

Reverberation

- Passage of impulse from presynaptic neuron and again back to presynaptic neuron leading to continuous stimulation of presynaptic neuron.
- REBERVERATING CIRCUIT
- Reverberation of impulse through the same circuit again and again.
- This is prevented to some extent in the form of fatigue.

Effects of acidosis and hypoxia:

- Hypoxia (lack of oxygen): CNS neurons cannot tolerate oxygen lack resulting in depression of neurons.
- Alkalosis: Increases neuronal excitability and can lead to convulsions.
- Acidosis: Neuronal activity is depressed. Can lead to coma.



**THANK
YOU**