



Synapses and Synaptic Transmission

Objectives

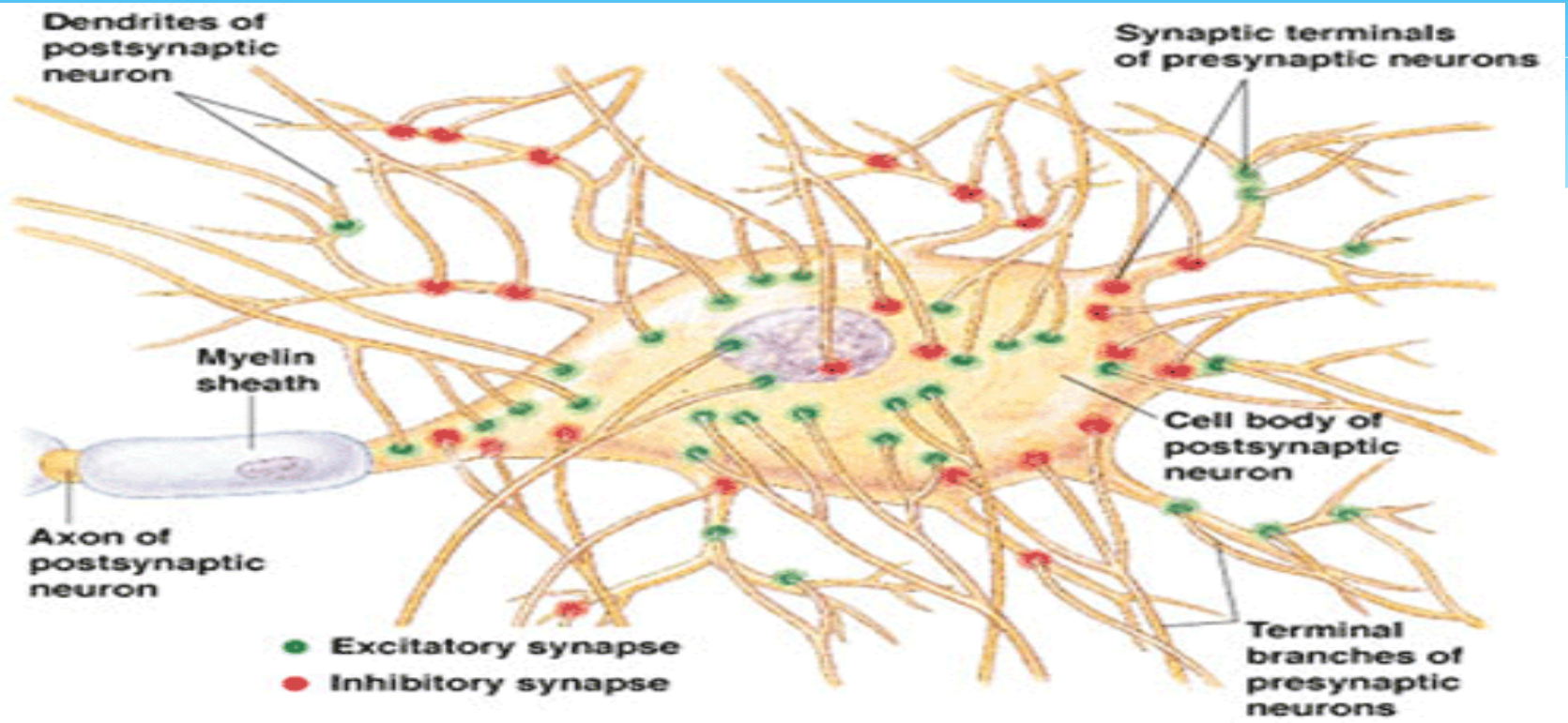
- * Define synapses
- * Functions of synapses.
- * Structure of synapses
- * Types of synapses: anatomical & functional.
- * Synaptic transmission & neurotransmitters
- * Fate of neurotransmitters.
- * Electrical events at synapses (EPSPs & IPSPs).
- * Properties of synaptic transmission
- * Factors affecting synaptic transmission

The CNS contains more than 100 billion neurons

Synapse:

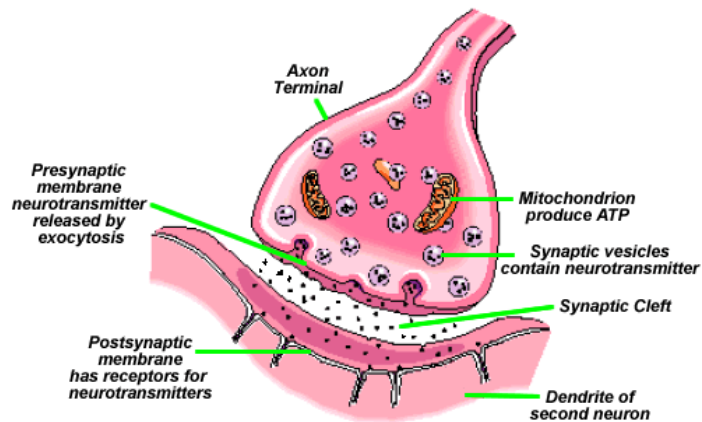
A junction where the axon or some other portion of one cell (= presynaptic cell) terminates on the dendrites, soma, or axon of another neuron (post synaptic cell).

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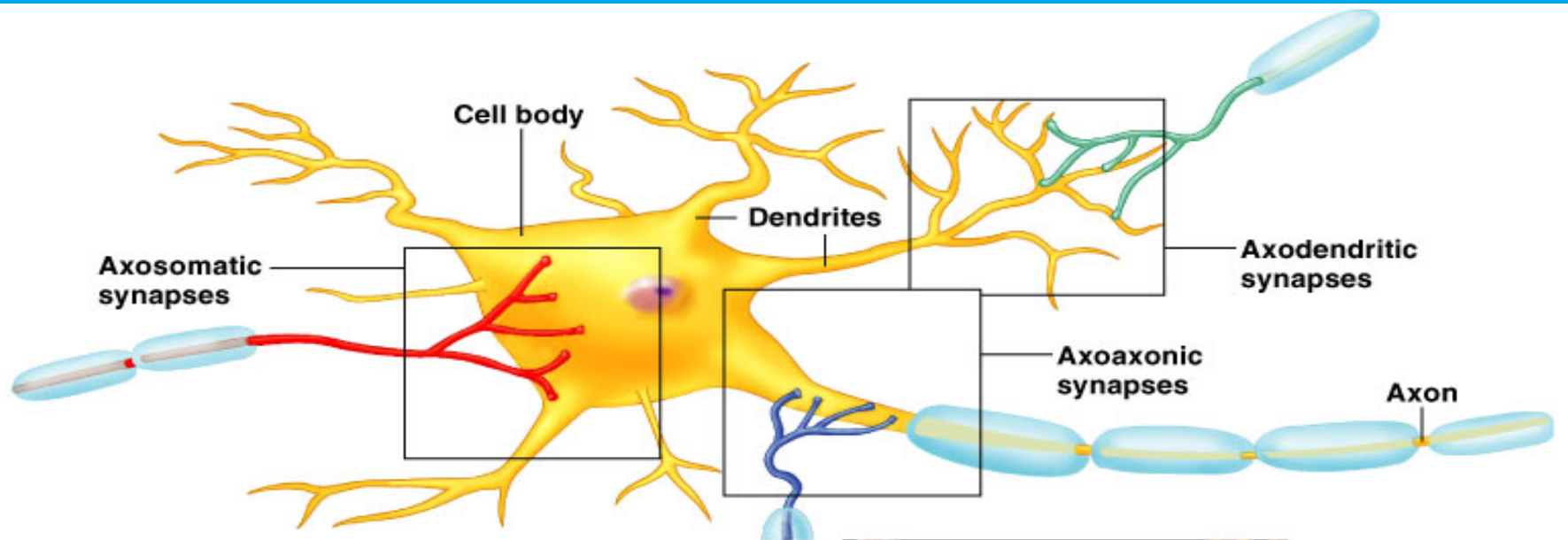
Functional Anatomy of a Synapse

A SYNAPSE

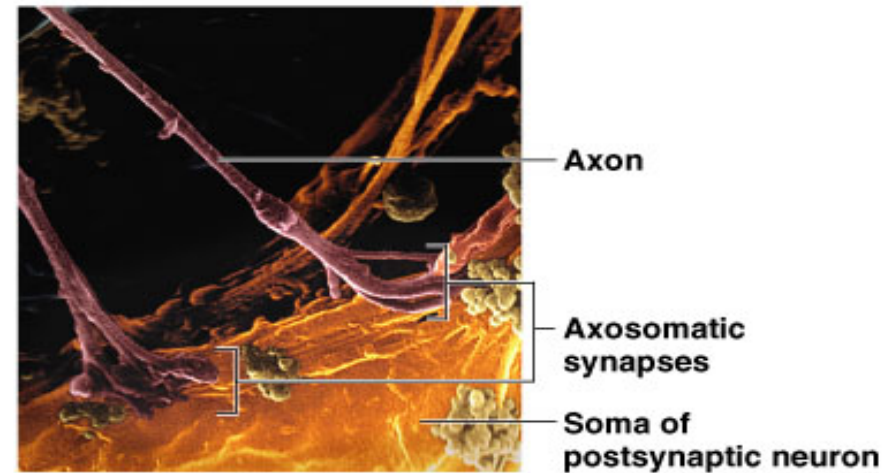


Anatomical Types of Synapses

- * Axodendritic - synapses between the axon of one neuron and the dendrite of another
- * Axosomatic - synapses between the axon of one neuron and the soma of another
- * Other types of synapses include:
 - * Axoaxonic (axon to axon)
 - * Dendrodendritic (dendrite to dendrite)
 - * Dendrosomatic (dendrites to soma)



(a)

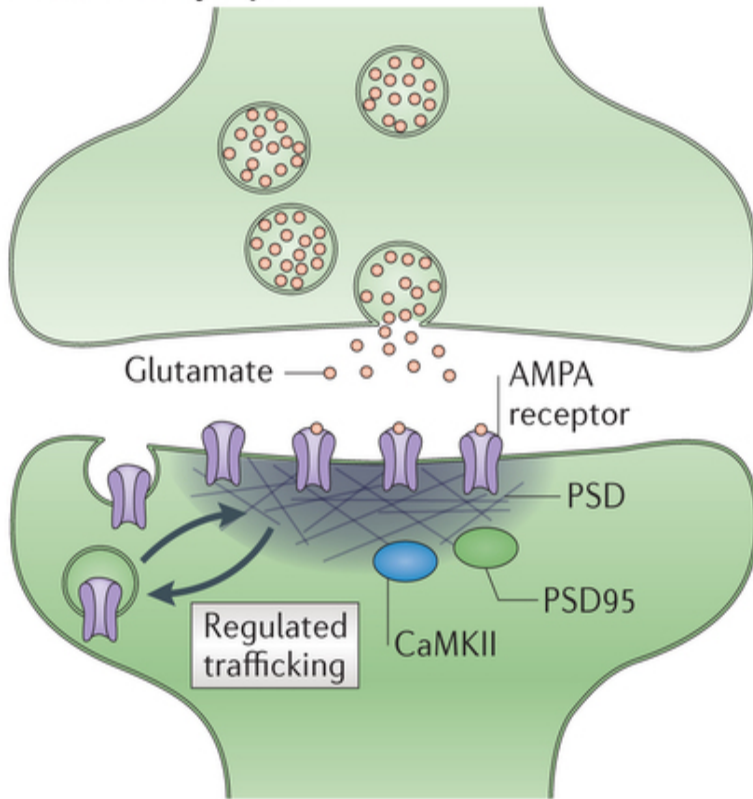


(b)

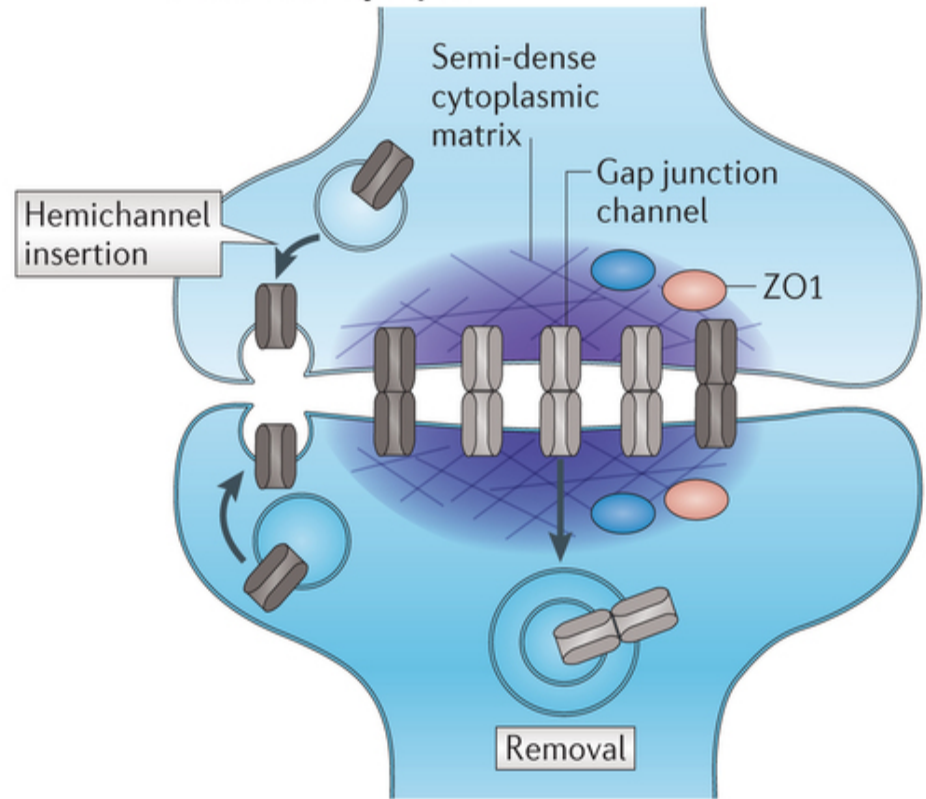
Figure 11.17

Functional types of synaps

a Chemical synapse



b Electrical synapse



Functional types of synapses

A. Chemical synapse

Almost all synapses in the CNS

((i.e. 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))

B. Electrical Synapses

Membranes of the pre- and post-synaptic neurons come close together and gap junctions forms → low membrane borders which allow passage of ions.

- * Are less common than chemical synapses
- * Correspond to gap junctions found in other cell types
- * Are important in the CNS in:
 - * Mental attention
 - * Emotions and memory
 - * Arousal from sleep

C. Conjoint synapse

Both electrical and chemical.

Examples → neurons in lateral vestibular nucleus.

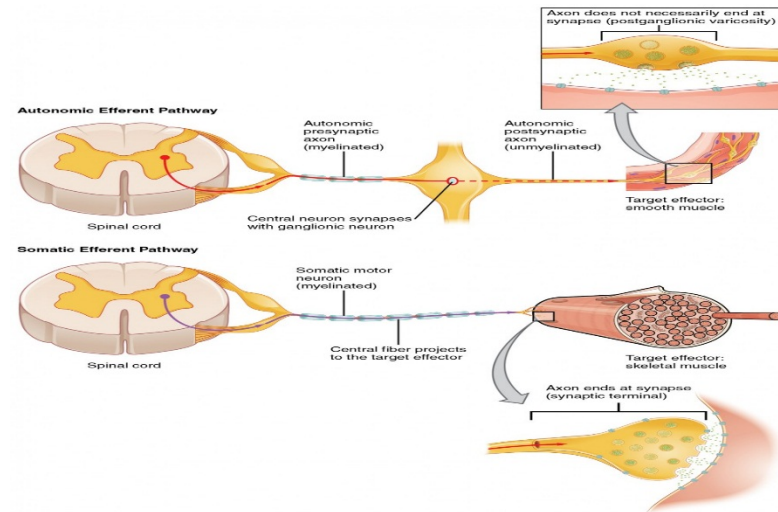
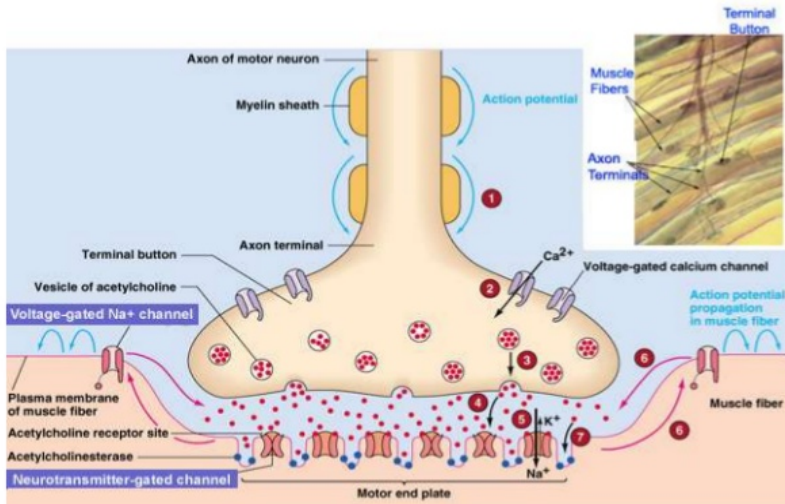
* Examples of synapses outside CNS

* - Neuromuscular junction

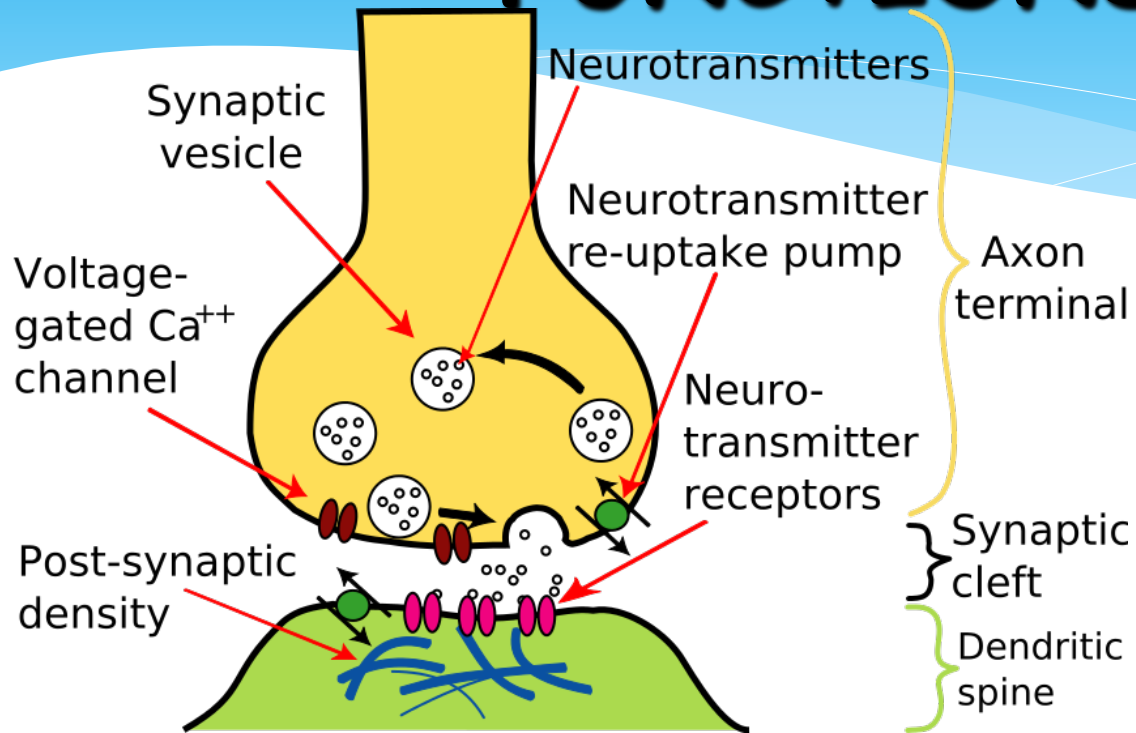
* - Contact between: autonomic neurons and smooth, cardiac muscles, and other effector

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The Neuromuscular Junction



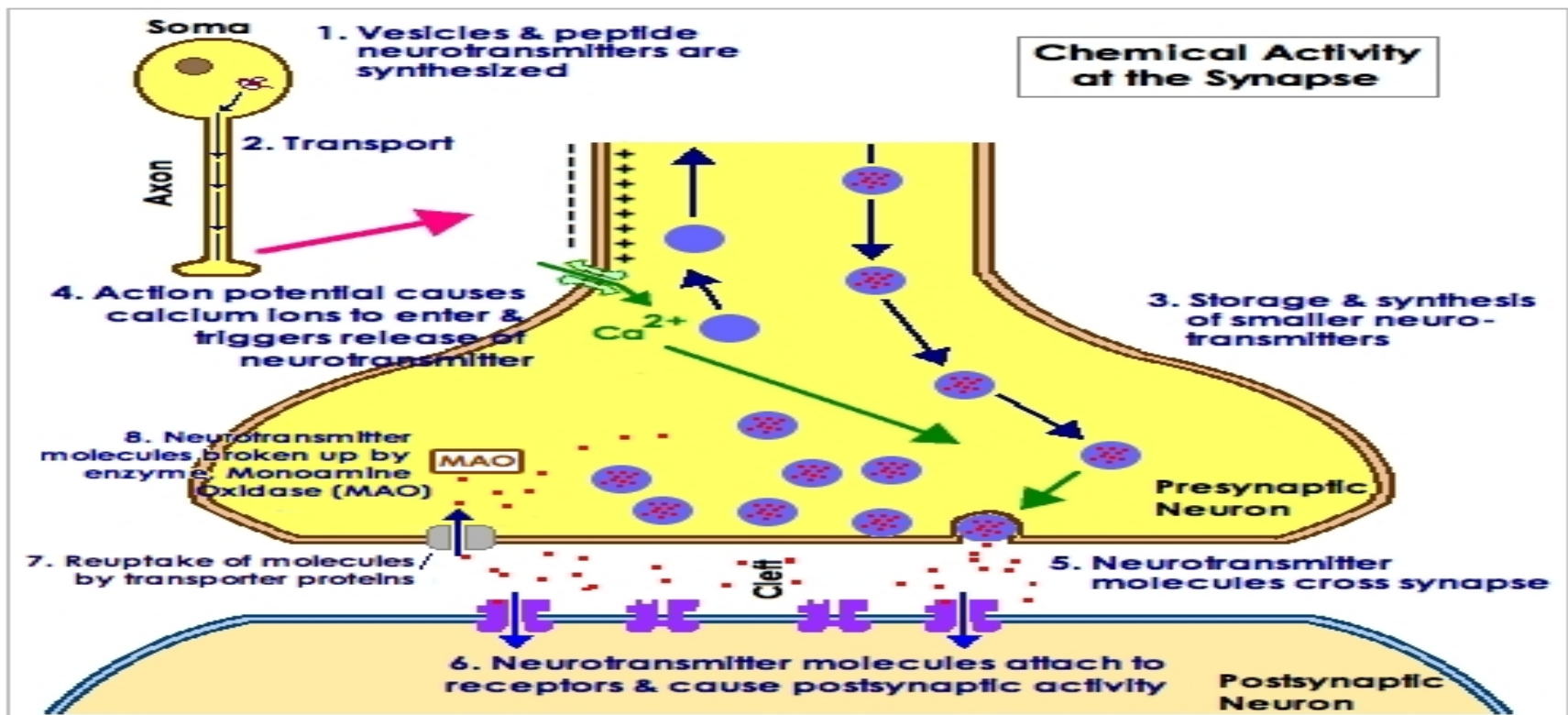
SYNAPSE: STRUCTURE & FUNCTIONS



- * **Synaptic cleft:** This is the space between the axon terminal and sarcolemma. It has a width of 200-300 angstroms.
- * **Synaptic knobs** (presynaptic terminal)



Mechanism of Synaptic Transmission:



- * AP >>>> NT release >>>> Binding to post synaptic receptors >>>> (((inhibition or excitation))) of the post-synaptic membrane
- * ((depending on the type of the neurotransmitter i.e. excitatory or inhibitory))



These receptors have two components

1. Binding site that face the cleft to bind the neurotransmitter

2. Ionophore: It passes all the way through the membrane to the interior. It is of two types

Ion channels

2nd messenger system in the post-synaptic membrane.

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...

Cation channels

Na⁺ (most common)
K⁺
Ca⁺⁺
Opening of Na⁺ channels → ↑ 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

* **Fate of a neurotransmitter**

- * After a transmitter substance is released at a synapse, it must be removed by
 - * Diffusion out of synaptic cleft into surrounding fluid
 - * Enzymatic destruction e.g. Ach esterase for Ach
 - * Active transport back into pre-synaptic terminal itself e.g. norepinephrine

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 [\uparrow 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 .

\therefore EPSPs = $+20\text{mV}$ which makes the membrane reach the firing level \rightarrow AP develops at axon hillock.

How EPSPs differs from Action potential?

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

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) \rightarrow \downarrow excitability and m. potential (more negative)

Synaptic properties

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

Is the minimum time required for transmission across the synapse. It is 0.5 ms 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 \uparrow membrane permeability
- Increased diffusion of Na^+ to \uparrow post-synaptic potential

3. Synaptic inhibition:

Types:

Direct inhibition

Indirect inhibition

Reciprocal inhibition

Inhibitory interneuron

A. Direct inhibition:

Occurs when an inhibitory neuron (releasing inhibitory substance) acts on a post synaptic neuron leading to → hyperpolarization due to opening of Cl^- [IPSPs] and/or K^+ channels.

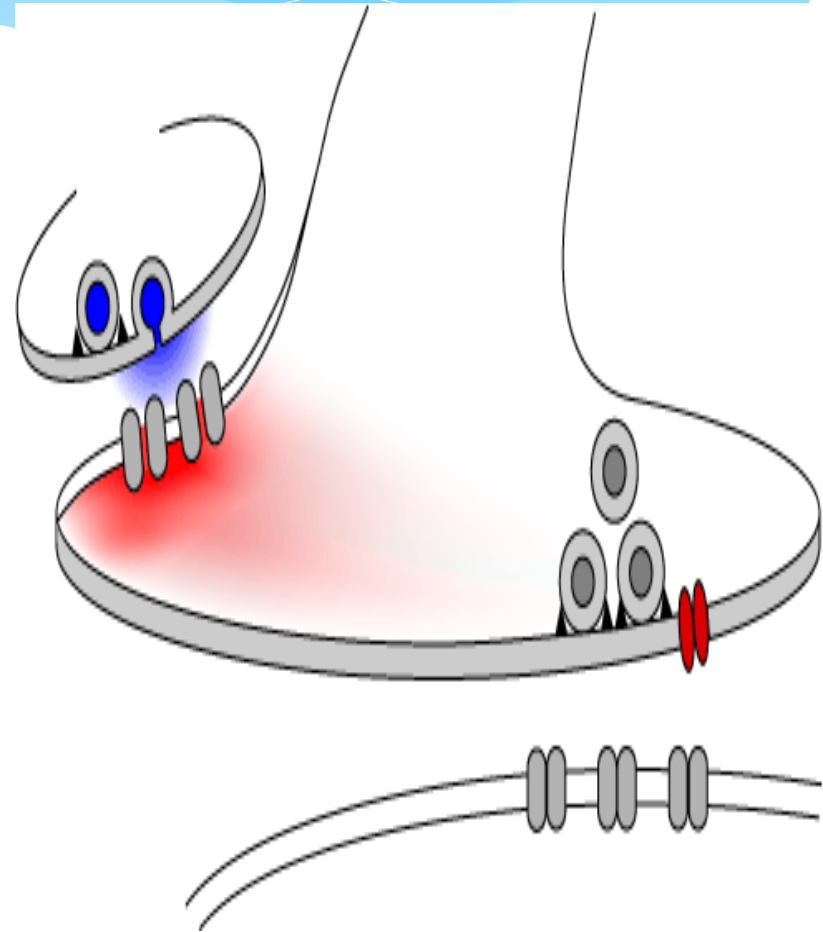
Example : Glycine at the level of the spinal cord to block pain impulses.

B. Indirect Inhibition:

(=Pre-synaptic inhibition).

This happens when an inhibitory synaptic knob lie directly on the termination of a pre-synaptic excitatory fiber.

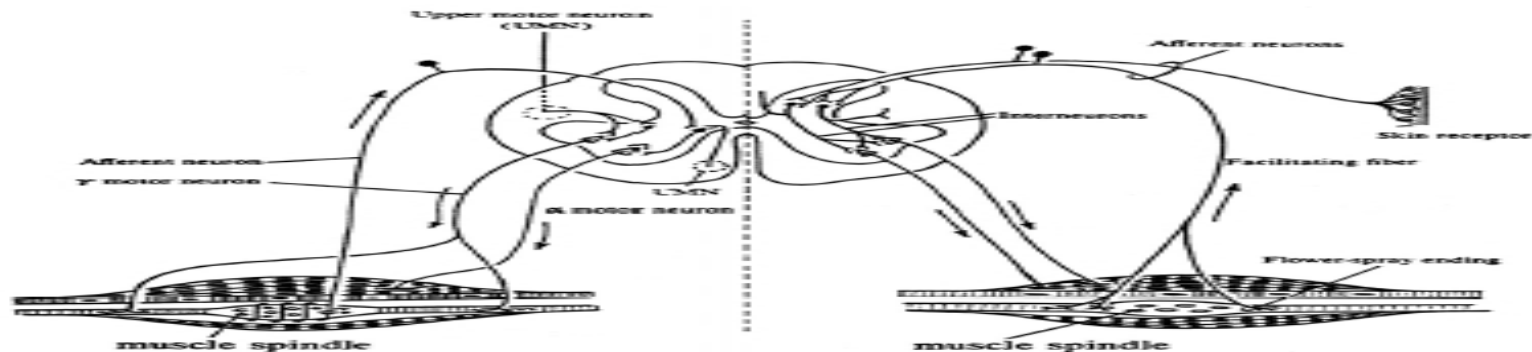
The inhibitory synaptic knob release a transmitter which inhibits the release of excitatory transmitter from the pre-synaptic fiber.
e. g. GABA (Pain modification)



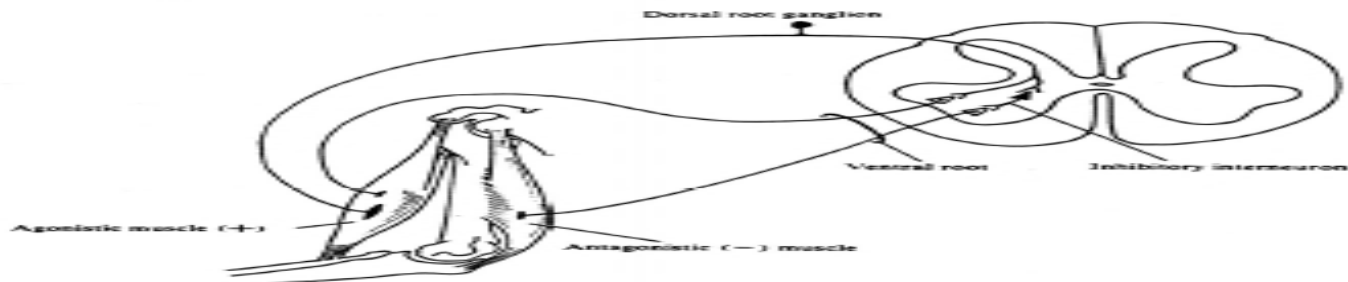
C. Reciprocal inhibition

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 neurons of antagonist muscle.



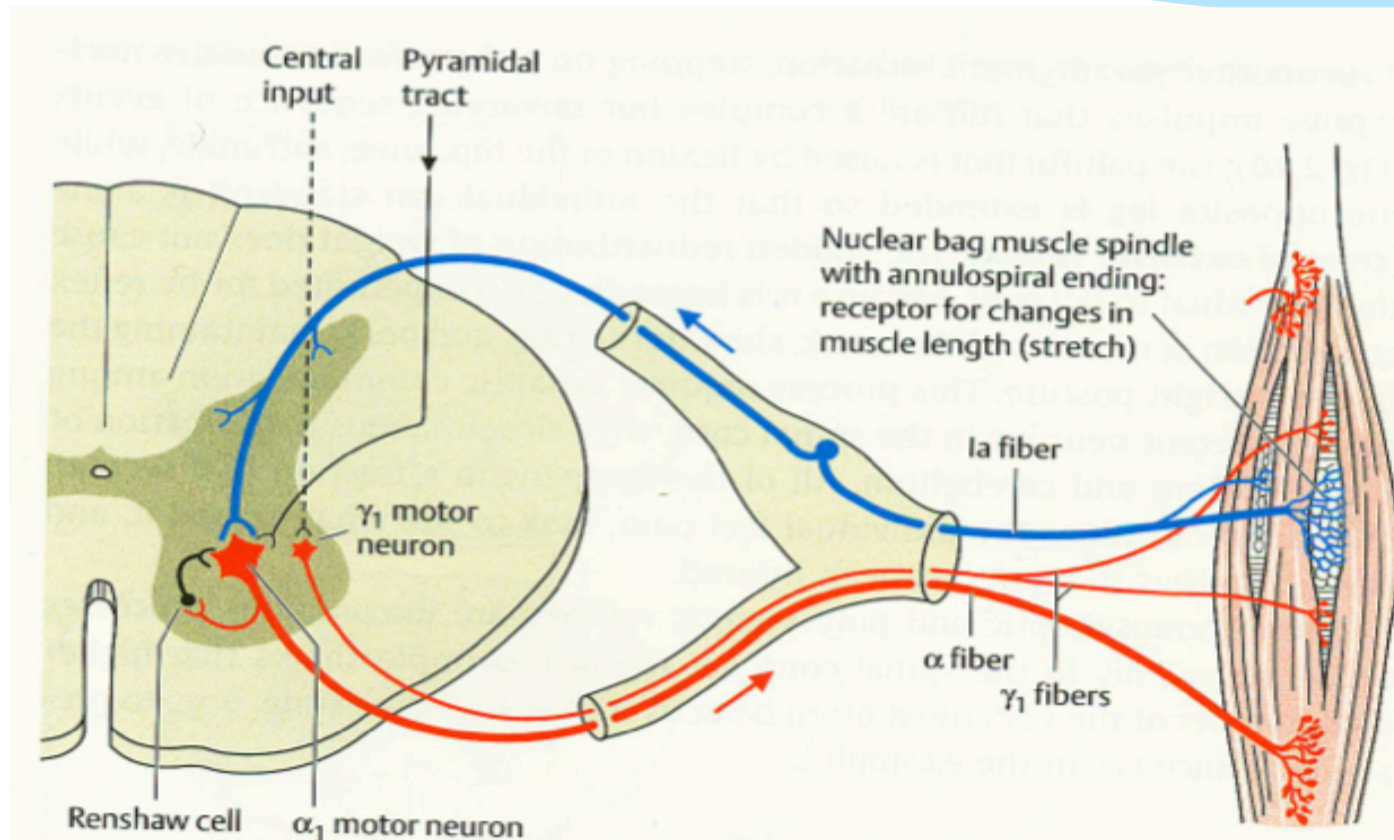
a



b

D. Inhibitory interneuron (Renshaw cells)

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



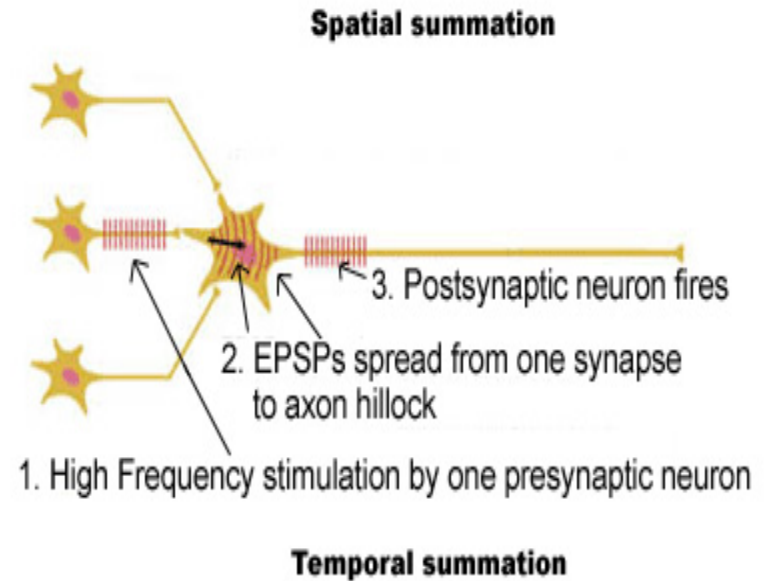
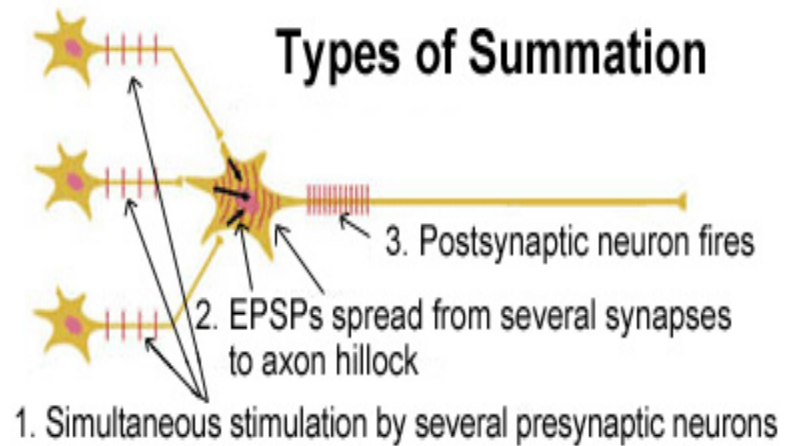
4. Summation:

a. Spatial summation.

Eliciting an action potential in a neuron with input from multiple presynaptic cells.

b. Temporal summation.

When the frequency of stimulation increased from the same presynaptic fiber



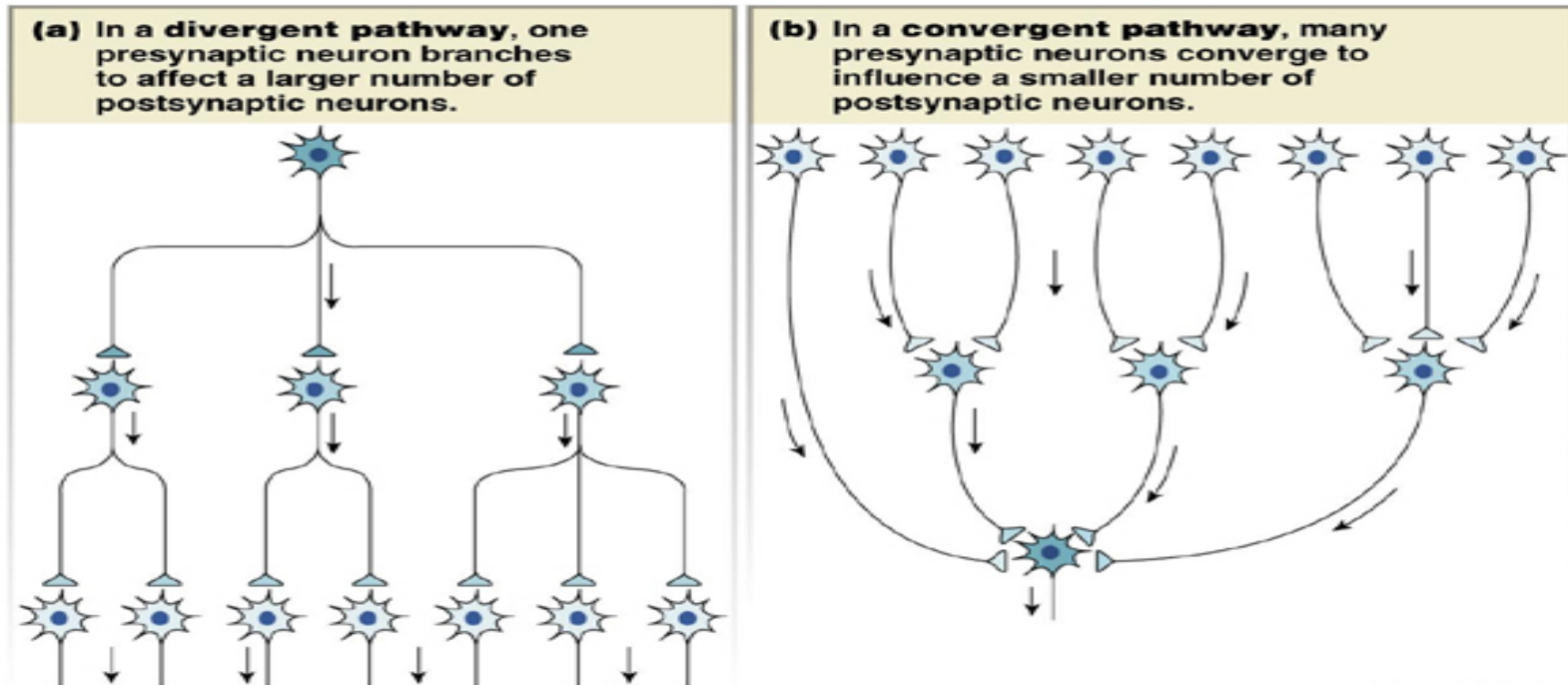
5. Convergence and divergence:

Convergence

When many pre-synaptic neurons converge on any single post-synaptic neuron.

Divergence

Axons of pre-synaptic neurons divide into many branches that diverge to end on many post-synaptic neurons.



6. Fatigue

It is due to exhaustion of neurotransmitter.

If the pre synaptic neurons are continuously stimulated there may be an exhaustion of the neurotransmitter. Resulting in stoppage of synaptic transmission.

Factors affecting synaptic transmission:

A l k a l o s i s :
Increases neuronal excitability.
Causes cerebral epileptic seizures (Increased excitability
c e r e b r a l n e u r o n s)
e. g. overbreathing in person with epilepsy
The over breathing blows off carbon dioxide and therefore
elevates the pH of the blood momentarily

- A c i d o s i s :

Depresses neuronal activity;
pH around 7.0 usually causes a coma
(e. g. severe diabetic or uremic acidosis)

- Drugs:

Caffeine found in coffee, tea, increases neuronal excitability, by reducing the threshold for excitation of neurons.

- Hypoxia

Depression of neurons

